Bioterrorism and Other Public Health Emergencies

Pediatric Terrorism and Disaster Preparedness

A Resource for Pediatricians

Prepared for:
Agency for Healthcare Research and Quality
U.S. Department of Health and Human Services
540 Gaither Road
Rockville, MD 20850
www.ahrq.gov

Purchase Order No. 05R000190

Prepared by:
American Academy of Pediatrics
Elk Grove Village, IL

Editors
George L. Foltin, MD, FAAP, FACEP
David J. Schonfeld, MD, FAAP
Michael W. Shannon, MD, MPH, FAAP

Section Editors
Jane Knapp, MD, FAAP
Marion J. Balsam, MD, FAAP
Lorraine M. Giordano, MD, FACEP
Julia McMillan, MD
Frederick Henretig, MD, FACMT, FAAP
David E. Jarrett, MD, FS, FACEP
Arthur Cooper, MD, MS, FACS, FAAP, FCCM
David Szydlo, MD, PhD
David Markenson, MD, FAAP
Irwin Redlener, MD, FAAP

AHRQ Editor
Mary L. Grady

AHRQ Publication No. 06(07)-0056
October 2006

Copyright Notice

Suggested citation

American Academy of Pediatrics. Pediatric Terrorism and Disaster Preparedness: A Resource for Pediatricians. Foltin GL, Schonfeld DJ, Shannon MW, editors. AHRQ Publication No. 06(07)-0056. Rockville, MD: Agency for Healthcare Research and Quality. October 2006.

Contents

Chapter 1. Introduction 1
Background 1
Children Are Not Small Adults 5
Anatomic Differences 5
Physiologic Differences 6
Immunologic Differences 7
Developmental Differences 7
Psychological Differences 8
Overview of Practical Considerations for Children and Families During Disasters 9
Surge Capacity 9
Pediatric Readiness 9
Summary 11
Bibliography 11

Chapter 2. Systems Issues 15
Types of Disasters 15
Natural Disasters 15
Manmade and Technological Disasters 17
Aftermath 18
Federal Response 19
Centers for Disease Control and Prevention 19
Department of Homeland Security 19
Federal Response Plan 20
Department of Defense 19
Summary 21
Bibliography 22

Chapter 3. Responding to a Disaster 27
Phases of Response 27
Preparedness 27
Actual Resonse to the Event 28
Mitigation 29
Recovery and Critique 29
Regional Response 30
State and Federal 30
Emergency Medical Services 30
Hospitals 31
Incident Command Systems 35
Regional Coordination of Hospital Response 36
Some Roles for the Pediatrician in Regional Hospital to Community Planning 37
Drills and Quality Assurance Activities 38
Integration with Children's Services 39
JCAHO and Emergency Management 40

Appropriate Triage....40
Incident Management....41
Staging....42
Triage Group....42
Treatment/Decontamination....42
Transportation....43
Bibliography....43

Chapter 4. Biological Terrorism....47
Background....47
History of Bioterrorism....47
Epidemiology of a Terrorist Attack....49
Agents Categorized by System Predominantly Affected....50
Respiratory System....50
Nervous System....51
Gastrointestinal System....52
Dermatologic Manifestations....52
Notifying Authorities....54
Health Department....55
Hospital....55
Law Enforcement....56
Laboratory Support and Submission of Specimens....56
Laboratory Response Network....56
Limiting Spread....56
Standard Precautions....56
Transmission Precautions....57
Equipment and Supplies....58
Pediatric Practices....59
Managing Patients: Treatment and Prevention....60
Smallpox Vaccine....60
Anthrax Vaccine....61
Strategic National Stockpile....61
Surge Capacity....62
Medications....62
Isolation....62
Vaccination....63
Information for Families....63
Category A Agents....63
Anthrax....63
Botulinum Toxin....66
Plague....69
Smallpox....71
Tularemia....74
Viral Hemorrhagic Fevers....76
Category B and C Agents....77
Ricin....77

Q Fever....78
Staphylococcal Enterotoxin B....79
Brucella....80
Burkholderia mallei (Glanders)....80
Encephalitis Viruses and Yellow Fever Virus....81
Clostridium perfringens....83
Bibliography....84

Chapter 5. Chemical Terrorism....107
Introduction....107
Specific Pediatric Vulnerabilities to Chemical Agents....108
Chemical Injuries and Approach to the Unknown Chemical Attack....108
Initial Approach, Decontamination, and Triage....109
Industrial Chemicals....110
Community Preparedness....111
Nerve Agents....111
Background....111
Toxicology and Clinical Manifestations....112
Diagnostic Tests....113
Treatment....114
Isolation and Control Measures....115
Cyanide....115
Toxicology....116
Clinical Presentation....116
Treatment....117
Vesicants....118
Characteristics....119
Clinical Effects....119
Treatment....120
Pediatric Considerations....122
Pulmonary Agents....123
Chlorine and Phosgene....123
Clinical Effects....124
Treatment....125
Riot Control Agents....126
Transmission and Pathogenesis....126
Clinical Manifestations....126
Diagnosis....127
Treatment and Control....128
Bibliography….....128

Chapter 6. Radiological and Nuclear Terrorism....143
Radiological Threats: Scope and Implications....143
Incident Management....143
Nuclear Weapons....144
Radiological Dispersal Devices (Dirty Bombs)....145

Medical and Industrial Sources of Radiation....146
Nuclear Power Plants....147
Historical Overview of Radiation Injury....147
Hiroshima and Nagasaki, Japan 1945....148
Mayak, Russia, former Soviet Union 1948-1990....148
Marshall Islands 1954....149
Three Mile Island, PA 1979....149
Chernobyl, Former Soviet Union 1986....149
Goiania, Brazil 1987....150
Radiological Dispersal Devices (Dirty Bombs)....150
Other Radiation Uses and Injuries....150
Physical Principles of Ionizing Radiation....151
Atomic Structure....151
Stability and Radiation....151
Radiation Interactions....153
Radiation Damage and Protection....154
Radiation Units....155
Radiation Background Exposure....156
Radiation Biology and Dosimetry....157
Radiation Biology....157
Biodosimetry....158
Biodosimetry and Radiological Terrorism....161
Pediatric Issues....161
Medical Diagnosis: Acute Radiation Syndrome....162
Pathophysiology....162
Clinical Stages....163
Syndromes....164
Emergency Care....165
Diagnostic Steps....165
Overall Dose Assessment....167
Medical Diagnosis: External Contamination....167
Background....167
Protection of Medical and Emergency Personnel....168
Emergency Care....168
Patient Evaluation....168
Medical Diagnosis: Internal Radionuclide Contamination....170
Background....170
Internal Contamination....170
Life Cycle of Internal Contamination....172
Diagnosis....173
Radiation Detection, Personal Protective Equipment, Personnel Monitoring, and Decontamination....175
Detection Using Radiation Survey Meters....175
Dosimeters....177
Personal Protective Equipment....178
Monitoring of Personnel/Decontamination....180

Basic Principles....180
Decontamination Techniques....181
Medical Treatment: General Issues Unique to Pediatrics....182
Acute Susceptibility....182
Long-Term Susceptibility....182
Psychological Vulnerability....183
Immediate Care....184
Medical Treatment: Acute Radiation Syndrome....184
Supportive Care....184
Neutropenia: Cytokine Therapy....185
Neutropenia: Antibiotic Therapy....186
Neutropenia: Viral and Fungal Infections....187
Neutropenia: Prophylactic Antimicrobials....187
Thrombocytopenia and Anemia: Blood Products....187
Bone Marrow Transplantation....187
Medical Treatment: Internal Contamination....188
Radioactive Iodine: Potassium Iodide....188
Radioactive Cesium and Thallium: Prussian Blue....190
Radioactive Plutonium, Americium, and Curium: DTPA Uranium:Bicarbonate....190
Other Radioactive Isotopes....191
Surgical Issues....191
Local Radiation Injury: Cutaneous Radiation Syndrome....191
Treatment and Management Issues....193
Trauma and Radiation (Timing of Surgery)....194
Management of the Patient with Embedded Radioactive Material and Depleted Uranium....194
Followup Care, Including Risk of Carcinogenesis....195
Environmental Issues Affecting Children After a Terrorist Incident Involving Radioactive Materials....197
Type of Incident....197
Environmental Exposure Pathways....199
Short-Term Evacuation Versus Sheltering....201
Long-Term Habitation Versus Abandonment....203
Rehabilitation/Abatement....203
Contamination of Crops, Water, Food Animals, and Milk Sources....203
Mortuary Affairs....204
Bibliography....204

Chapter 7. Blast Terrorism....237
Introduction....237
Explosives....237
Blast Fundamentals....238
Blast Trauma....239
Incendiary Weapons....247
Aviation Terrorism....248
Trauma Systems....248

Trauma Hospitals....248
Trauma Centers....248
Non-Trauma Centers....250
Treatment....250
Trauma....251
Burns....251
Multiple Casualties....251
Mass Casualties....251
Planning and Mitigation....252
Planning....252
Mitigation....253
Bibliography....255

Chapter 8. Mental Health Issues....265
Mental Health and the Role of the Pediatrician....265
Trauma-Related Disorders....265
Reaction to Disasters and Terrorism....266
Assessment and Treatment....268
Death Notification and Pediatric Bereavement....270
Considerations in Notifying Individuals About an Unexpected Death....270
Explaining Death to Children....275
Common Reactions Among Children Who Have Experienced a Personal Loss....276
Indications of the Need for Referral....277
Attendance of Children at Funerals and Memorial Services....278
Therapies for Psychic Trauma....278
Crisis Response....278
Crisis Response for Children and Families....279
Medication....280
School Crisis Response....282
Anniversary Reactions and Commemorative Activities....283
Anniversaries....283
Memorialization....284
Planning a Memorial Activity....285
Supporting School Staff....286
Impact on Health Care Providers....286
Make Psychological Contact....287
Level of Coping....287
Explore Possible Solutions....287
Take Action....287
Followup....287
Basic Objectives....287
Benefits....288
Risk Communication and Media Issues....288
Developing Goals and Key Messages....288
Delivering Accurate and Timely Information....289
Bibliography....290

Chapter 9. Integrating Terrorism and Disaster Preparedness into Your Pediatric Practice 293
Relevance for Office-Based Pediatricians 293
Internal Operations of the Practice: Office Readiness 293
External Operations: Communication and Coordination with Other Agencies 296
Communicating Directly with Children and Families 298
Relevance for Hospital-Based Pediatricians 299
Emergency Department Readiness 299
Inpatient Service Readiness 300
Hospital Infrastructure Needs 300
Bibliography 301

Chapter 10. Working with Government Agencies 303
Introduction 303
Community, Government, and Public Health Preparedness 303
Community Response 303
State Government 304
Federal Government 304
Public Health Preparedness 305
Advocating for Children and Families in Preparedness Planning 305
Resources Available from Government Agencies 306

Chapter 11. Conclusion 309
Systems Issues 309
Regional, State, and Local Efforts 309
Vulnerable Populations 310
Separation of Children from Families 310
Sheltering Families 310
Providing Urgent Medical Care to Large Populations of Displaced Children 311
Setting up a Temporary Pediatric Clinic Lessons Learned 311
Environment 312
Medical Needs 312
A Final Word 312

Acronyms 319

Appendixes

Appendix A. Learning Objectives 323
Appendix B. List of Contributors 333

Figures and Tables

Figures

Figure 2.1 Federal Response Plan....24
Figure 3.1 National Response Plan....44
Figure 4.1 Inhalational anthrax; chest radiograph taken 22 hours before death....88
Figure 4.2 Neurological signs, botulism, 6-week-old infant....89
Figure 4.3 Cutaneous anthrax lesion with eschar on neck....90
Figure 4.4 Physical distribution of smallpox lesions versus varicella lesions....91
Figure 4.5 Small pox lesions....92
Figure 4. 6 Varicella lesions....93
Figure 4.7 Expected smallpox vaccination site reaction (i.e., a "take") in a first-time vaccinate....94
Figure 4.8a Febrile rash illness algorithm for evaluating patients suspected of having smallpox....95
Figure 4.8b Classification of risk Febrile rash illness algorithm....96
Figure 6.1 Probability of radiation casualties....216
Figure 6.2 Environmental exposure pathway....217
Figure 6.3 Generic pediatric medical field card....218
Figure 6.4 Bone marrow irradiated to 3 Gy....219
Figure 6.5 Pattern of a series of blood counts graphed over time after 3 Gy of whole-body exposure....220
Figure 6.6 Classic Andrews diagram....221
Figure 6.7 Contamination versus exposure....222
Figure 6.8 Examples of radiation survey meters....223
Figure 6.9 Recommended procedures for monitoring personnel....224
Figure 6.10 Localized radiation effects....225

Tables

Table 1.1 Types of disasters....13
Table 2.1 Types of disasters, hazards, or events that may require Federal assistance....25
Table 3.1 Training and competencies of prehospital emergency personnel....45
Table 4.1 Early clinical signs and symptoms after exposure to selected bioterrorist agents....97
Table 4.2 Infection control transmission precautions for Category A agents....99
Table 4.3 Diagnostic procedures, isolation precautions, treatment, and postexposure prophylaxis for selected bioterrorist agents in children....100
Table 4.4a Diagnostic tests for anthrax....103
Table 4.4b Adjunctive diagnostic tests for anthrax....103
Table 4.5 Postexposure prophylaxis for anthrax....104
Table 4.6 Treatment recommendations for tularemia in children before test results are known....105
Table 5.1 Pediatric vulnerabilities to chemical terrorism....132
Table 5.2 Chemical weapons Summary of pediatric management considerations....133
Table 5.3 Representative classes of industrial chemicals Summary of pediatric management considerations....135

Table 5.4 Nerve agent triage and dosing 137
Table 5.5 Clinical effects from sulfur mustard exposure.....139
Table 5.6 Riot control agents.....140
Table 5.7 Medical treatment of riot control agent exposure.....141
Table 6.1 Biological dosimetry assays and operational parameters 226
Table 6.2 Biodosimetry based on acute photon-equivalent exposures.....227
Table 6.3 Initial or prodromal phase, whole body irradiation from external radiation or internal absorption (gamma radiation) 228
Table 6.4 Diagnostic steps in acute radiation syndrome.....229
Table 6.5 Timing to onset of vomiting (prodrome) 230
Table 6.6 Dose assessment summary.....231
Table 6.7 Guidelines for bioassay sampling.....232
Table 6.8 Antibiotics for treatment of post-radiation neutropenia 233
Table 6.9 Threshold radioactive exposures and recommended prophylactic single doses of KI 234
Table 6.10 Radionuclide specific therapies 235
Table 6.11 Reduction in exposure by sheltering.....236
Table 7.1 Expected injuries at relative distances from detonation to open air 258
Table 7.2 Spectrum of primary blast injury on body systems 259
Table 7.3 Principles of Advanced Trauma Life Support 260
Table 7.4 Principles of Advanced Burn Life Support 262
Table 8.1 Concepts of death and implications of incomplete understanding for adjustment to loss.....292
Table 11.1 Environmental constraints to pediatric medical care after large-scale natural disasters.....313
Table 11.2 Pediatric medical complaints after large-scale natural disasters: challenges and adaptations based on post-hurricane responses.....314

Chapter 1. Introduction

Background

A disaster is a calamitous event that affects a large population and generally results in injury, death, and destruction of property. A disaster can also be thought of as any occurrence that taxes or overwhelms local response resources (e.g., law enforcement, transportation, shelters, etc.). Local resources can be overwhelmed by natural disasters or other events that result in multiple casualties such as earthquakes, fires, large motor vehicle crashes, or terrorist incidents. Because disasters vary, preparation should vary accordingly. Disasters caused by terrorism or accidents (e.g., a multiple car crash on an interstate highway) can occur without warning. In other types of disasters, such as hurricanes, there is usually some time for warning and preparation. Some disasters end quickly, while others can affect large populations over an extended period of time (e.g., a humanitarian disaster involving famine). Disasters can have physical, mental, and emotional effects on a large number of people without regard to age or other factors. This widespread negative impact is what makes terrorist attacks so effective.

An understanding of the many types of disasters (Table 1.1) and the implications of each is essential for preparedness planning. In all cases, terrorism and disaster planning can be divided into three phases:

1. The primary, or response, phase consists of actions and care taken during and immediately after the disaster.
2. The secondary response, also called the recovery phase, is the period during which affected people work toward reestablishing normalcy. Emotional and mental health problems usually begin to emerge during this phase.
3. The tertiary response, or the mitigation phase, consists of efforts to apply lessons learned to prevent future disasters or to lessen their impact.

The lessons learned from past terrorist events and natural disasters should guide plans for future preparation and response. The first lesson is that natural disasters and terrorist events can and do occur in the United States. The second is that bombs, germs, toxic gases, and the forces of nature do not discriminate between children and adults. Despite our best efforts to shelter and protect them, children remain among the most vulnerable victims of terrorism and disasters. A third lesson is that disasters cannot always be predicted or prevented; they have the potential to affect anyone at any time. These lessons underscore the need for preparation by planning a comprehensive system of response that fully addresses and integrates the needs of everyone, including children.

Several terrorist events in recent years have had profound consequences for children and families. When the Murrah Federal Building in Oklahoma City was bombed in 1995, 19 children at the child care center inside the building were killed, and many more were injured. Hundreds of other children lost parents or relatives, and countless more suffered emotionally. Thousands of children lost parents in the terrorist attacks of 2001 at the World Trade Center and the Pentagon.

There are many gaps in knowledge, especially with regard to children, regarding disaster preparation and planning. Historically, the unique characteristics and needs of children have not been adequately addressed in the planning process for response to terrorism. Why is this so? In the past, much of the terrorism response planning in the United States has centered on military preparedness, and therefore, plans have focused on the needs of adults. As we plan for response to terrorism, it is time to reassess education and planning for all disasters and to ensure that children and their families are included. Ensuring appropriate care for children during disasters cannot be accomplished by simply modifying current practices. Basic day-to-day issues that involve families have not been considered previously (e.g., incorporating schools and child care centers into disaster preparation and planning; also, planning for the likelihood that numerous children will become separated from their families) and should now be addressed. (See also Chapter 9, Integrating Terrorism and Disaster Preparedness into Your Pediatric Practice and Chapter 11, Conclusion.) The likelihood of a disaster occurring while children are in school or at child care centers is high, and the site of the disaster could even be at the school or child care center.

Incorporating the needs of children and families into terrorism and disaster planning requires multidisciplinary pediatric expertise at all phases. This *Pediatric Terrorism and Disaster Preparedness* resource presents information on including children and families at all levels of terrorism and disaster planning. A few of the many considerations include the following:

- Writing and implementing child-specific protocols.
- Planning for children who are separated from their parents and at schools and child care centers when disaster strikes.
- Training providers to care for pediatric patients.
- Developing equipment and medication dosage forms and delivery systems appropriate for children.
- Providing education on the recognition and care of mental health needs of children in the aftermath of a disaster.
- Planning for children with special health care needs.

This resource is intended primarily to educate, inform, increase awareness among, and assist pediatricians in recognizing and fulfilling their important roles in disaster preparedness and response. Families and communities turn to pediatricians for anticipatory guidance on all issues involving children. Pediatricians can help families plan their response to disaster by referring them to available resources. The Family Readiness Kit has been developed by a coalition of the American Academy of Pediatrics (AAP), the American College of Emergency Physicians (ACEP), and 27 other State and national organizations to assist families in planning (see www.aap.org/family/frk/frkit.htm).

Questions regarding immunization for infectious agents such as smallpox, antibiotic prophylaxis after exposure to infectious agents, coping with the effects of exposure to

violence, and disaster preparedness in the home are common. Pediatricians should be ready to provide accurate answers. The AAP Web site includes information, created by the AAP Task Force on Terrorism, on disaster preparedness to meet the needs of children (www.aap.org/disaster), as well as links to many other sources of information.

Pediatricians enjoy a high degree of public trust as expert sources of information and support on matters involving the health and well-being of children and families. Therefore, their roles in disaster preparedness and management are extremely important. For example, pediatricians act as first responders and care providers when the emergency medical system and emergency departments become rapidly overwhelmed in the recovery and mitigation phases of incidents of terrorism or disasters. Pediatricians, especially in instances of bioterrorism (such as the case of anthrax in an infant in New York City), could be the first to see victims and determine a diagnosis. This means that pediatricians should acquire further knowledge of infections and the effects of exposure to toxins that most likely they have never seen. Residency training in pediatrics has been limited on subjects such as biological and chemical terrorism, as well as nuclear exposure, and it should be broadened accordingly. Children's hospitals, which serve many communities, should also generate and implement this information.

The pediatric office could also be involved in the first response phase after a disaster. A good first step for the pediatrician is preparation of an office disaster plan that is periodically updated and practiced (see also Chapter 9, Integrating Terrorism and Disaster Preparedness into Your Pediatric Practice). As the office plan is prepared, pediatricians should consider other roles they might have in the community disaster response and familiarize themselves with liability and licensure issues. Working with an agreement with local/state government agencies to provide disaster services affords the best liability coverage and often allows reimbursement. For a discussion of their liability, pediatricians should review the AAP Policy Statement (reaffirmed in 2004) *Pediatricians' Liability During Disasters* (see http://aappolicy.aappublications.org/cgi/content/full/pediatrics;106/6/1492).

Recommendations include the following:

- Familiarity with State statutes and protections afforded while providing emergency care during a disaster.
- Familiarity with individual liability insurance coverage outside of the usual practice settings when providing urgent and routine care.
- Working in concert with response agencies when providing disaster relief.

Parents turn to pediatricians for help, guidance, support, treatment, and referral regarding mental health issues in the recovery and mitigation phases of disaster. A wide range of reactions can be expected, from anxiety to adjustment reactions and posttraumatic stress disorder (PTSD). The effects can be direct or indirect, affecting children who were not actually involved in the disaster. Parents will turn to pediatricians with many questions regarding how to explain the reasons for disaster, whether to let their children watch the events on television, and what to say about loved ones or acquaintances who have been

injured or killed (see also Chapter 8, Mental Health Issues). Pediatricians should be able to recognize potential symptoms of adjustment reactions and PTSD and to give parents coping strategies and referrals. The role of pediatricians in the mental health care of children after terrorism and disasters is described in a recent article, "Psychosocial implications of disaster or terrorism on children: A guide for the pediatrician," which is available at http://www.pediatrics.org/cgi/content/full/116/3/787.

The pediatrician's perspective is well-suited to assist in the community planning process. Pediatricians have an appreciation for children as part of families that comprise communities that become regions, States, and so on. This perspective is valuable, but pediatricians may have limited knowledge and skill in planning and response. Pediatricians should educate themselves, acknowledge their limitations, and/or obtain outside expert input. The role of the pediatrician has been comprehensively described and defined in the AAP Policy Statement *The Pediatrician's Role in Disaster Preparedness* prepared by the Committee on Pediatric Emergency Medicine (http://pediatrics.aappublications.org/cgi/content/full/99/1/130).

Pediatricians are respected advocates for children. In this role, pediatricians should advocate for resources and products that currently do not exist for children, especially for children with special health care needs (including the chronically ill and technologically dependent). For example, children cannot always be decontaminated in adult decontamination units. Skin decontamination showers that are safe for adults may cause hypothermia in children unless warming equipment (e.g., heating lamps) is provided. Decontamination systems should be designed for use with children of all ages, for the child unaccompanied by a parent, for the nonambulatory child, and for the child with special health care needs. Little protective gear is available for children; when its use has been attempted, such as with gas masks, mishandling has led to fatalities from suffocation.

Vaccines for anthrax and plague are not approved for use in children. The frequency of serious complications after administration of smallpox and yellow fever vaccines is higher in children than in adults; development and approval of safer vaccines are needed (see also Chapter 4, Biological Terrorism). Antidote kits for use after nerve agent exposure such as the Mark-1 kit (for adults) have only recently been developed for children (see Chapter 5, Chemical Terrorism). Common systems for determining drug dosages in children do not include dosages for antidotes. Recently, a liquid preparation of potassium iodide (65 mg/cc) has come on the market for use in preventing radiation-induced thyroid effects after radiation exposure (see also Chapter 6, Radiological and Nuclear Terrorism and the AAP Policy Statement on *Radiation Disasters and Children* at www.aap.org/policy/s040208.html).

Planning and preparation for terrorism and disasters can be both daunting and challenging. For all, but especially for children, there are many recognized gaps in knowledge, resources, and professional education. This resource has been provided to increase pediatric expertise of those taking on the challenge of preparation and planning. This resource will be invaluable, not only for pediatricians, but also for other pediatric

health care providers, public health professionals, health administrators, and policymakers who are committed to ensuring that planning for terrorism and disasters includes the special needs of children.

Children Are Not Small Adults

Many important differences distinguish children from adults and are the origin of the oft-used truism "you can't treat children as small adults." Children have many unique anatomic, physiologic, immunologic, developmental, and psychological considerations that potentially affect their vulnerability to injury and response in a disaster. Failure to account for these differences in triage, diagnosis, and management of children is most often due to lack of knowledge or experience, or both. Experience has shown that such lack of knowledge and experience can result in grave errors, increasing the child's risk of serious harm, and even death.

Anatomic Differences

An obvious difference between children and adults is size. Children are smaller than adults and vary in size depending on stage of growth and development. Their small size makes them more vulnerable to exposure and toxicity from agents that are heavier than air such as sarin gas and chlorine. These agents accumulate close to the ground in the breathing zone of infants, toddlers, and children.

A child's smaller mass means greater force applied per unit of body area. The energy imparted from flying objects, falls, or other blunt or blast trauma is transmitted to a body with less fat, less elastic connective tissue, and closer proximity of chest and abdominal organs. The result is a higher frequency of injury to multiple organs.

A smaller body has smaller circulating blood volume (on average 80 mL/kg) and less fluid reserve. These differences have several important implications. Volumes of blood loss that would be easily handled by an adult can result in hemorrhagic shock in a child. Children are more vulnerable to the effects of agents such as staphylococcal enterotoxins or *Vibrio cholerae* that produce vomiting and diarrhea. Therefore, infections that might cause mild symptoms in adults could lead to hypovolemic dehydration and shock in infants, small children, and children with special health care needs.

The child's skeleton is more pliable than that of adults. It is incompletely calcified with active growth centers that are more susceptible to fracture. Orthopedic injuries with subtle symptoms and physical findings are easily missed in preverbal children. Internal organ damage can occur without overlying bony fracture. Serious cardiac or lung injuries without having incurred rib fractures are common.

A child's cervical spine is subject to distracting forces that are more likely to disrupt the upper cervical vertebra and ligaments. Numerous bony anatomic variations render the interpretation of radiographs potentially confusing. Additionally, children can have spinal cord injury without radiographic abnormality.

Head injury is common in children. The head is a larger, heavier portion of a child's body compared with the head of an adult. It accounts for a larger percentage of body surface area (BSA) than it does in adults, and it is a major source of heat loss. It is supported by a short neck that lacks well-developed musculature. The calvarium is thin and vulnerable to penetrating injury, thus allowing greater transmission of force to the growing brain of a child. The brain doubles in size in the first 6 months of life and achieves 80% of its adult size by age 2. During childhood, there is ongoing brain myelinization, synapse formation, dendritic arborization, and increasing neuronal plasticity and biochemical changes. Injury to the developing brain can affect or arrest these processes, resulting in permanent changes.

The mediastinum is very mobile in children. Subsequently, a tension pneumothorax can quickly become life-threatening when the mediastinum is forced to the opposite side, compromising venous return and cardiac function.

The thoracic cage of a child does not provide as much protection of upper abdominal organs as that of an adult. Hepatic or splenic injuries from blunt trauma can go unrecognized and result in significant blood loss leading to hypovolemic shock.

The airway differs between children and adults. The tongue is relatively large compared with the oropharynx, which creates the potential for obstruction of a poorly controlled airway. The larynx is higher and more anterior in the neck, and the vocal cords are at a more anterocaudal angle. The epiglottis is omega-shaped and soft. The narrowest portion of the airway is the cricoid ring, not the vocal cords as in adults. Airway differences combine to make the child's airway more difficult to maintain as well as to intubate. The short length of the trachea increases the risk of a right mainstem bronchus intubation. The lungs are smaller and subject to barotraumas, resulting in pneumothorax with inappropriate ventilation.

The BSA to mass ratio is highest at birth and gradually diminishes as the child matures. The distribution of BSA also differs between children and adults. Children have a higher percentage of BSA devoted to the head relative to the lower extremities. This should be taken into account when determining the percentage of BSA involved in burn injuries and in treating or preventing hypothermia.

The higher BSA to mass ratio also leads to more rapid absorption and systemic effects from toxins that are absorbed through thinner, less keratinized, highly permeable skin.

Physiologic Differences

Children differ physiologically in many ways from adults. They can compensate and maintain heart rate during the early phases of hypovolemic shock; this false impression of normalcy can lead to administration of too little fluid during resuscitation. This can be followed by a precipitous deterioration with little warning.

Vital signs, including heart rate, respiratory rate, and blood pressure, vary with age. Caregivers should be able to quickly interpret whether a child's vital signs are normal or

abnormal for age. Temperature is an often forgotten but important vital sign in injured children. The child's ability to control body temperature is affected not only by the BSA to mass ratio but also by thin skin and lack of substantial subcutaneous tissue. These factors increase evaporative heat loss and caloric expenditure. In fact, hypothermia is a significant risk factor for poor outcomes in many illnesses/injuries. Considerations of methods to maintain and restore normal body temperature are critical to the resuscitation of children. These can include thermal blankets, warmed resuscitation rooms, warmed intravenous fluids, and warmed inhaled gases.

Children have a higher minute ventilation per kilogram of body weight than adults. This means that over the same period of time, they are exposed to relatively larger doses of aerosolized biological and chemical agents than are adults. The result is that children suffer the effects of these agents much more rapidly. Children are also more likely to absorb more of the substance from the lungs before it is cleared or diffused through ventilation.

Fluid resuscitation, drug dosages, and equipment sizes are based on the child's weight. Estimating the weight of a child can be difficult, particularly for health care workers with limited pediatric experience. An easy, quick method for determining a child's weight is to use the Broselow–Hinkle Pediatric Resuscitation Measuring Tape®. This tool rapidly provides many common drug dosages and fluid resuscitation volumes. Health care providers should also make appropriate fluid choices for resuscitation. Children who receive large volumes of hypotonic fluid are at risk of hyponatremia and seizures.

Limited glycogen stores and a higher relative metabolism in children than in adults puts children at a higher risk of hypoglycemia. Children compensate for cardiovascular and pulmonary problems with tachycardia and tachypnea (their ability to increase stroke volume and tidal volume is limited).

Immunologic Differences

Children have an immature immunologic system, which places them at higher risk of infection. Immunologically, children have less herd immunity from infections such as smallpox and a unique susceptibility to many infectious agents. For example, Venezuelan equine encephalitis is usually a brief, self-limiting infection in adults. In children, it can be severe, and life-threatening encephalitis develops in 4% of victims. Children immunized with the current smallpox vaccine are over-represented with serious side effects such as encephalitis.

Developmental Differences

Developmental differences between children and adults are also readily apparent. Children, especially infants and toddlers, might be unable to describe symptoms or localize pain. Children rely on parents or others caregivers for food, clothing, and shelter. Infants especially are vulnerable when their food sources are eliminated or contaminated.

In situations of disaster, caregivers can be injured, killed, or simply not present. Children, especially infants and toddlers, are limited in their verbal ability to communicate their wants and needs. Children also have motor skills that are insufficient to escape from the site of an incident. Additionally, their cognitive development may limit their ability to figure out how to flee from danger or to follow directions from others, or even to recognize a threat. The developing brain has emotional instability with an inadequate ability to interact in stressful situations and an emotional state frequently dictated by that of their caregivers. A child's reaction to danger or threat is influenced by their developmental stage, which means that responders should be familiar with age-appropriate interventions.

Younger children are unable to take care of their needs for activities of daily living, so an adult caregiver must oversee them. Children with special needs often cannot perform some activities of daily living or medical interventions by themselves. Planning/response must allow for adult caregivers.

Psychological Differences

The psychological effects of disaster on children are neither uniform nor universal in nature (see Chapter 8, Mental Health Issues). Important factors in the psychological effect of a disaster on children include the nature of the disaster itself, the level of exposure to the disaster, the extent to which the children and those around them are personally affected, and individual characteristics of each child. In addition, children are unique because they are affected not only by their own reaction to the trauma of the event but also by their parent's fears and distresses. Because children depend on adults for their emotional and psychological needs, any effects of trauma on adults can magnify the psychological impact on children.

Children are still undergoing psychological development at the time of disaster. Their developmental stage characterizes their response and is responsible for the wide degree of variability in adjustment to traumatic events. This means that therapeutic interventions should be developmentally appropriate.

The response of younger children is characterized by changes in mood and behavior and by anxiety. Younger children may exhibit regressive behaviors, increased temper tantrums, and symptoms of clinginess and difficulty with separation or sleep. Even infants whose lives have been disrupted by a disaster manifest symptoms of crying and irritability, separation anxiety, and a hyperactive startle response.

School-age children may exhibit depression, anger, and despair. Their anxiety may be exacerbated by unrealistic fears for parents, families, and friends. They also may develop problems at school or somatization symptoms, typically with complaints of headache or abdominal pain.

Adolescents differ from adults in their psychological response because they are in a period of development characterized by complex physical, psychological, and social transitions. They are especially vulnerable to the development of major psychiatric

disorders such as depression. Of significant importance is the likelihood of engaging in risk-taking behaviors such as drug abuse or sexual relationships. Adolescents are also particularly vulnerable to impulsive behaviors including suicide. In addition, adolescents may try to hide their feelings or symptoms for fear of being perceived as abnormal. It is imperative that these symptoms not be minimized or overlooked because adjustment reactions left unrecognized and untreated can lead to lifelong behavioral and emotional problems.

Overview of Practical Considerations for Children and Families During Disasters

The anatomic, physiologic, immunologic, developmental, and emotional differences between children and adults give rise to many practical considerations for planning.

Emergency medical services (EMS) agencies should consider adopting triage tools such as JumpSTART® (http://www.jumpstarttriage.com/) that use physiologic decision points adapted for ranges of pediatric normals and that consider apnea as a potentially salvageable respiratory emergency. The AAP has prepared a resource and course that provides training equipment guidelines for prehospital providers in the care of children, *Pediatric Education for Prehospital Professionals* (http://www.peppsite.com/).

Surge Capacity

Surge capacity is the ability of a hospital or other health care facility to expand quickly beyond normal services to meet an increased demand for medical care in the event of bioterrorism or other large-scale public health emergency. Converting a hospital or other health care facility from its current capacity to surge capacity is a daunting task. In addition to ensuring that essential supplies, staff, and services are available, planners also must ensure that facilities can accommodate the needs of vulnerable groups, such as children, the elderly, and the disabled.

Pediatric Readiness

Ambulances, clinics, and hospital emergency departments typically carry only limited quantities of pediatric equipment. Sufficient supplies and equipment should be readily available to treat large numbers of pediatric victims. Because equipment choices and drug dosages, including IV rates, depend on the child's size, a quick, convenient system to guide appropriate choices should be in place. The system used should be comprehensive enough to include dosages for antidotes and other medications that may be relevant during a terrorist event. A surge in pediatric victims can quickly overwhelm hospital, regional, and even State pediatric capacity, so strategies should include solutions that involve hospital, regional, State, and Federal planning to manage such surges. Plans should also consider that pediatric victims will present as families when injured adults refuse to be separated from their children.

The resuscitation of children can be further complicated by the technical difficulty of procedures such as intubation and intravenous access. Alternative methods for

maintaining and securing the airway should be considered. When veins are small and/or constricted from shock or hypothermia, the equipment for alternative methods, such as intraosseous access, should be readily available.

Planning should consider all potential aspects of a child's life. Therefore, it should account for children who are at home, in school or child care, or in transit, as well as for children who cannot be reunited with their families. School disaster plans should coordinate with community plans and should also consider post-incident stress management during the recovery phase. Child care centers and community youth centers should have disaster plans that focus on ensuring safety, accessing and interacting with community emergency responders, notifying guardians, and reuniting families.

Children are predisposed to illness and injury after a disaster for a variety of reasons. There can be lack of adult caretaker supervision, and the usual resources of school or child care may be unavailable. Environmental hazards can be increased from collapsed buildings or dangerous tools or from chemicals or availability of weapons. Increased stress on adults might lead to a higher risk of domestic violence or child abuse. Contagion present in the community, especially infections such as respiratory syncytial virus or influenza, may spread rapidly in group shelters. Contaminated food or water can lead to epidemic outbreaks of infectious diseases, resulting in gastroenteritis and dehydration. Changes in the environment can lead to heat-related illness or hypothermia. Use of alternative sources for heating or generators can lead to carbon monoxide exposure. Children with asthma may have acute exacerbations due to stress or environmental contaminants. Medications may be forgotten or the supply may be exhausted, resulting in exacerbations of chronic illnesses. Stress can produce a variety of symptoms in children including headaches, abdominal pain, chest pain, vomiting, diarrhea, constipation, changes in sleep, and changes in appetite.

Many considerations in planning are prompted by the possibility of children in shelters. These include supplies and services such as diapers, infant formula, other child-appropriate food, and games and other distractions for children. Staffing is an issue with regard to supervision. Shelters should be childproofed to promote safety for children as well as the elderly. Sick children should be isolated. Children should be protected from environmental hazards such as weapons, alcohol, and cigarette smoke. Children with special health care needs, especially those that depend on technology for survival, are particularly vulnerable and should be considered in shelter planning. Also, parents/single parents with sick children cannot be caregivers simultaneously for both a hospitalized child and non-hospitalized, sheltered children.

Planning should also include pregnant women, the fetus, and the newly born. The stress of a disaster can contribute to premature labor and delivery. Infection acquired by the fetus in utero can lead to fetal death or to devastating consequences if the fetus survives. The risk of developing cancer is higher in children who have been exposed to radiation in utero. Radioactive iodine is transmitted to human breast milk and threatens infants who are breastfeeding. Cow's milk can also be quickly contaminated if radioactive material settles onto grazing areas, threatening alternative sources of nutrition.

Summary

As demonstrated by past events, there is ample opportunity to improve preparedness for children involved in disasters (both man-made and natural). This *Pediatric Terrorism and Disaster Preparedness* resource contains information needed for pediatricians to be prepared for disasters at all phases of planning, response, recovery, and mitigation. The role of the pediatrician should not be minimized, underestimated, or overlooked in disaster planning and response. Pediatricians, based on their traditional roles in prevention, anticipatory guidance, and advocacy, can make a difference in comprehensive public health plans for disaster.

Bibliography

- American Academy of Pediatrics Task Force on Terrorism. Policy statement: how pediatricians can respond to the psychosocial implications of disasters. *Pediatrics.* 1999;103(2):521–523. Available at: http://aappolicy.aappublications.org/cgi/content/full/pediatrics;103/2/521. Accessed August 17, 2006.
- American Academy of Pediatrics Committee on Environmental Health and Committee on Infectious Diseases. Chemical-biological terrorism and its impact on children. *Pediatrics* 2006 September; 118(3):1267-1278. Available at: http://pediatrics.aappublications.org/cgi/content/full/118/3/1267?submit.y=12&submit.x=83&cga=118%2F3%2F1267&. Accessed September 6, 2006.
- American Academy of Pediatrics Committee on Pediatric Emergency Medicine. Policy statement: the pediatrician's role in disaster preparedness. Pediatrics.99(1)130-133.
- American College of Surgeons Committee on Trauma. *Pediatric Trauma in Advanced Trauma Life Support Manual*, 6th ed. Chicago: American College of Surgeons; 1997. 353–375.
- Cieslak TJ, Henretig FM. Bioterrorism. *Pediatr Annals*. 2003;32(3):1–12.
- Hagan JF, Committee on Psychosocial Aspects of Child and Family Health, Task Force on Terrorism. Psychosocial implications of disaster or terrorism on children: a guide for the pediatrician. *Pediatrics.* 2005; 116:787-95. Available at: http://www.pediatrics.org/cgi/content/full/116/3/787. Accessed August 21, 2006.
- Markenson D, Redlener I, eds. Pediatric Preparedness for Disasters and Terrorism: a National Consensus Conference; New York. New York: National Center for Disaster Preparedness, Columbia University; 2003.
- Romig LE. Pediatric triage: a system to JumpSTART your triage of young patients at MCIs. JEMS. 2002;27(7):52–63.
- Romig LE. Disaster Management. In A*PLS: The Pediatric Emergency Medicine Resource* 4th ed. Gausche-Hill M, Fuchs S, Yamamoto L (eds). Sudbury MA: Jones and Bartlett; 2003. 542–567.
- Schonfeld DJ. Almost one year later: looking back and looking ahead. *J Dev Behav Pediatr*. 2002;23(4):292–294.

- Schonfeld DJ. In times of crisis, what's a pediatrician to do? *Pediatrics.* 2002;110(1 Pt 1):165.
- Schonfeld DJ. Supporting adolescents in times of national crisis: potential roles for adolescent health care providers. *J Adolesc Health.* 2002;30(5):302–307.

Table 1.1 Types of disasters

Natural Disasters	
Hurricanes or cyclones	Droughts
Tornadoes	Wildfires
Mudslides	Earthquakes
Tsunamis	Infestations or disease epidemics
Ice or hail storms	
Technological Disasters	
Hazardous materials releases or spills	Transportation crashes or derailments
Unintentional explosions or collapses	Power outages
Terrorism, National or International Violence	
Bombings or explosions	Multiple or mass shootings
Chemical agent releases	Cult-related violence
Biological agent releases	Riots
Nuclear agent releases	Arson
Humanitarian Disasters, Complex Emergencies	
War or violent political conflict	Genocidal acts
Famine	Shelter, feeding, or medical care of displaced populations
Droughts	

Source: Courtesy of the American Academy of Pediatrics. From: Romig LE. Disaster Management. In APLS: *The Pediatric Emergency Medicine Resource* 4th ed, Gausche-Hill M, Fuchs S, Yamamoto L (eds). Sudbury MA: Jones and Bartlett, 2003. pp 542-567.

Chapter 2. Systems Issues

Types of Disasters

Disasters are sudden calamitous events that can result in great damage, loss, injury, and death. They can occur naturally, such as floods, earthquakes, hurricanes, tornados, tsunamis, or wildfires, or they can be caused by human error or intervention. The widespread injury and disruption associated with disasters can pose difficult problems for health care providers including triage of mass casualties, disruption of infrastructure (e.g., loss of power and fresh water), and the need to deal with the mental anguish associated with uncertainty and the loss of loved ones.

The degree of injury, death, and damage caused by disasters is influenced by many factors, including population location and density, timing of the event, and community preparedness (e.g., emergency response infrastructure, local building codes, etc). Similarly, recovery after a disaster is influenced by resources (e.g., savings, insurance, and relief aid), preexisting conditions (e.g., season, local infrastructure, etc), experience, and access to information. In almost all cases, disasters are associated with mental and physical stress (both during and after the event) that can increase morbidity and mortality over and above that caused directly by the event itself.

Natural Disasters

Natural disasters usually occur suddenly and are often uncontrollable. However, they frequently cluster temporally or geographically, and therefore are somewhat predictable. In the United States and other developed countries, most natural disasters tend to cause extensive damage and social disruption with comparatively little loss of life. The most frequent types of natural disasters experienced in the United States are floods, earthquakes, hurricanes, tornados, and fires.

Floods. The most common natural disaster is flood, which accounts for roughly 30% of disasters worldwide. Approximately 25–50 million Americans live or work in floodplains, and another 110 million live in coastal areas. The frequency of flooding is increasing, due in part to increasing habitation in flood-prone areas and in part to deforestation and changing land-use patterns, which can increase the degree of flooding.

Flash floods are especially hazardous and occur during sudden heavy rains, tidal surges, or when dams or levees give way. Most of the deaths during flash floods are caused by drowning, usually from people wading or driving through moving water. The hazards posed by rapidly moving water are often unrecognized. A gallon of water weighs 8 pounds; hundreds of gallons of rushing water represent thousands of pounds of force. As little as 2 feet of rushing water can carry a vehicle away, trapping the passengers.

Except for flash flooding, floods generally are not directly associated with significant loss of life. However, flooding results in considerable destruction and disruption, and has the potential for widespread disease. Floodwaters frequently contain human or animal waste from sewage or agricultural systems that can lead to epidemics of infectious disease. Drinking water must be disinfected through boiling and/or chlorination, or an alternative clean water supply (e.g., bottled

water) must be identified and made accessible. Water supplies and household surfaces can also become contaminated with petroleum products (e.g., fuel oil or kerosene), household chemicals, and molds.

Contamination of floodwaters also poses a hazard to those participating in the clean up. Rubber boots and gloves should be worn, and open wounds and sores protected. Hands should be washed frequently, especially when handling food or food containers. Foods that may have been contaminated should be discarded. Eating utensils should be thoroughly washed with soap and hot water and disinfected with a solution of 1 cup bleach to 1 gallon water. All inside surfaces, especially those used for food preparation, should be similarly cleaned. Likewise, all child play areas need to be cleaned and disinfected, along with all toys, clothing, etc. Materials that cannot be readily disinfected should be discarded.

Earthquakes. Earthquakes are a potential hazard throughout the continental United States, especially within the tectonically unstable areas of California, Idaho, Utah, and the Pacific Northwest. Only part of the destruction caused by earthquakes and their aftershocks occurs during the event. Subsequent events triggered by the quake, such as fires, tidal waves, and so on, can cause significant destruction.

The force of an earthquake is measured on the Richter scale, which estimates the energy imparted by the quake or aftershock. Every increasing Richter unit represents an increase in energy by an order of magnitude. Richter units can be used as an estimate of earthquake probability/frequency, with an order of magnitude decrease in likelihood with every unit increase. For example, on average approximately 2 earthquakes of magnitude 8 are expected worldwide per year, 20 quakes of magnitude 7, and 200 quakes of magnitude 6.

Although earthquakes cannot be prevented, much of the injury and damage they produce can. Improvements in emergency response and health infrastructure can speed up response time and lessen death and disability. Perhaps most importantly, structures built under improved building codes and with stronger construction materials can survive earthquakes with less damage. Also, as with all natural disasters, damage can be mitigated considerably through simple preventive measures, such as turning off utilities, securing appliances, and taping windows.

Hurricanes and tornados. Hurricanes and tornados are similar weather events that differ in magnitude and location. Both involve rotating masses of air associated with severe weather. Tornados usually measure only a few hundred meters across and travel over only a few kilometers of land, while hurricanes can stretch over hundreds of kilometers. Both can have winds of up to 200 mph, but hurricanes are associated with much more energy and have much more potential for destruction. Tornados develop primarily over landmasses, especially those within the Midwestern and Southwestern United States, while hurricanes are associated with the coastal United States, primarily the East and Gulf coasts.

Although hurricanes are associated with high winds, much of the destruction they cause is from the so-called “storm surge” and subsequent flooding. High winds and low pressure can cause water to pile up in coastal areas up to 14 meters above normal sea level. This can result in all the

problems noted above for flooding, including the risk of drowning, electrocution, and disease associated with contaminated drinking water.

Much of the risk associated with these severe weather events can be mitigated through advanced warning and preparation. This is especially true for tornados, because of their sudden onset. Redundant warning systems should be developed, and everyone should be encouraged to practice tornado drills. Special outreach efforts should be made to those with special needs or disabilities, including designation of a "buddy" who knows the individual's needs and can ensure that they are prepared for an emergency. Each person/family should have a tornado shelter (e.g., cellar, basement, etc) that is equipped with an appropriate emergency kit.

Most of the injury and death associated with hurricanes is through failure to heed warnings. Individuals may refuse to evacuate or seek shelter, may not properly secure their property, and may ignore guidelines on food and water safety and injury prevention. Therefore, effective risk communication is important both in preparation for the event and during cleanup and mitigation efforts.

Tsunamis. Underwater earthquakes can result in the formation of gigantic waves that can cross thousands of miles of ocean at speeds up to 500 mph. These waves are often no taller than wind-generated waves, but they are much more dangerous. Tsunamis have long wavelengths up to several hundred miles, making them more like prolonged flood waves than normal surf. The waves slow as they reach shallow water, causing them to crest at heights up to 100 feet. When the waves break, they can destroy piers, buildings, and human life far inland. There is little warning as a tsunami wave front approaches the coast, allowing few life-saving preventive actions. Therefore, the best hope for protecting human life is prediction and advance warning through seismology, wave gauges, etc.

Wildfires. Brush or forest fires can disrupt communities and cause substantial property damage, displacement, serious burns, and death. In addition, smoke from wildfires can result in irritation and respiratory difficulties, especially among those with preexisting medical conditions or impairment. As with other natural disasters, serious injury and death often result from failure to evacuate or otherwise heed warnings. Official agencies need to make provisions for those with disabilities to ensure that they are evacuated and that their special needs are addressed.

Manmade and Technological Disasters

Anthropogenic disasters are explosions, chemical releases, etc, directly associated with human action. They can be caused by accidents or deliberate malicious activities or when industrial facilities are disrupted by natural disasters. Examples of accidental anthropogenic disasters include the Bhopal chemical release and the Three-Mile Island nuclear accident. Intentional disasters include arson and terrorist attacks, such as the events of September 11, 2001. Anthropogenic disasters share many of the characteristics of natural ones but are typically less predictable.

Accidental anthropogenic disasters. Accidental anthropogenic disasters include a broad range of incidents that vary with the nature of the industry involved. These include hazardous material releases of various types (e.g., caustic agents, asphyxiants, radioactive materials), fires,

explosions, structural collapses, transportation failures, and many more. The medical and public health responses to such events depend on the incident and type of hazardous material involved.

Although less predictable than natural disasters, accidental disasters are more preventable. Basic safety procedures and equipment, adequate training, and proper maintenance can go a long way toward preventing accidents. Emergency response training, safety drills and simulations, and medical training in appropriate responses to hazardous agents can greatly limit subsequent injury and death when accidents occur.

Intentional anthropogenic disasters. Intentional manmade disasters are the least predictable, with no restrictions other than the limits of the imagination of a deviant mind. The nature of these disasters can vary from simple arson or sabotage, to release of chemical or biological agents, or even to detonation of a primitive nuclear device. These disasters are associated with most of the hazards described for accidental, and sometimes even natural, disasters. However, the malicious nature of the event and the fear associated with biological, chemical, and nuclear agents result in even greater stress and social disruption.

Terrorists do not consider the age of victims, only the impact of their act on furthering a cause. Children have been and will be victims of terrorist acts. Schools, gyms, sporting events, concerts, amusement parks, shopping malls, or any other place where there are mass gatherings are all potential terrorist targets. Release of a product into the ventilation system of a local school or any of the other sites could result in rapid spread of an agent throughout a community.

Aftermath

Many deaths are possible in the aftermath of a disaster. Because considerable injury and destruction can be associated with any disaster, management after a disaster is critically important. The disruption caused by disasters can result in widespread disease from unhygienic conditions. Fuel leaks, live wires, and other hazards can cause injury or start fires. The physical and emotional stress associated with the event and cleanup can result in heart attacks, musculoskeletal injuries, mental illness, and other stress-related disorders. Displaced wildlife can hamper relief efforts and endanger workers. Injuries can also result from improper use of chain saws or other mechanical equipment involved in clean-up efforts. Children are especially prone to injury or poisoning through access to debris, chemicals, equipment, and other agents discovered while exploring in the aftermath of the disaster.

When returning to a building or structure after a disaster, occupants need to check for structural damage, gas leaks, downed power lines, or other potentially hazardous situations. Sites should be inspected during daylight so that hazards are visible, and only battery-powered flashlights or lanterns should be used for auxiliary light, rather than candles, gas lanterns, or other open-flame devices.

Immediately after a disaster, governments and community organizations will be called upon to provide safe (e.g., bottled) drinking water, as well as shelter, food, clothing, and medical care for displaced people. Victims will also look to these organizations for other services, including counseling and assistance with insurance claims and other sources of emergency funds.

Federal Response

The United States has established a robust emergency medical support infrastructure to respond to disasters at local, State, regional, and Federal government levels. Many response resources are deployable and can be relocated to accommodate varying disaster scenarios. Other response resources are integral to established health and medical systems in our country and provide health care to Americans and others on a daily basis. Populations with specific emergency medical needs in disasters—such as neonatal, pediatric, or adolescent populations—have limited support that is quickly available and specifically designed for to meet their urgent life-sustaining needs.

Response resources dedicated to pediatric populations continue to be inadequate for most emergency medical response activities related to disasters, even though victims often include children. Many children were injured or killed during the attacks on the World Trade Center in New York City and the Federal Building in Oklahoma City, and countless more witnessed these events. These experiences highlight the need to include pediatric and other special populations in local, State, regional, and Federal disaster medical planning. Table 2.1 depicts the types of disasters that my require Federal assistance.

Centers for Disease Control and Prevention

The Department of Health and Human Services (DHHS) is the lead Federal agency for public health issues. The Centers for Disease Control and Prevention (CDC), one of 12 DHHS agencies, provides support to the Department in carrying out its mission. CDC priorities include disease prevention and control, environmental health, and health promotion and education activities.

Within CDC, the Director's Emergency Operations Center (DEOC) operates 24 hours a day, 7 days a week to provide emergency consultation and assistance to clinicians, State and local health agencies, and citizens. The DEOC can be reached at 770-488-7100. The Clinician Information Line (877-554-4625) is available to clinicians 24 hours a day to provide guidance on the management of patients suspected of having bioterrorism-related illnesses and, when necessary, can refer pediatricians to agent-specific subject matter experts.

Pediatricians can register to receive real-time CDC updates about preparing for (and possibly responding to) terrorism and other emergency events at http://www.bt.cdc.gov/clinregistry/index.asp.
For more information about CDC's organization and overall mission, see http://www.cdc.gov/aboutcdc.htm.

Department of Homeland Security

Within the last decade, because of the severity of manmade disasters and their devastating effects on life and safety, as well as the increased threat of terrorism, the Federal Government has positioned itself to respond promptly to any future terrorist events that may occur in the United States. A key step was creation of the Department of Homeland Security (DHS) in 2002. One of its missions is to minimize damage and assist in recovery from terrorist attacks that occur within the United Sates.

State and local governments share the primary responsibility for protecting their citizens from disasters, and for helping them recover when a disaster strikes. In some cases, a mass disaster is beyond the capabilities of State and local governments to respond, and the Federal sector is called upon to provide assistance.

The DHS now comprises 22 agencies, including the Federal Emergency Management Agency (FEMA). DHS is responsible for the comprehensive National Strategy for Homeland Security, which is focused on six key areas:

- Intelligence and warning.
- Border and transportation security.
- Domestic counterterrorism; protecting critical infrastructure.
- Defending against catastrophic threats.
- Emergency preparedness and response.

To learn more about DHS and its role in disaster preparedness and response, go to http://www.dhs.gov/dhspublic/theme_home2.jsp.

Federal Response Plan

The Federal Response Plan (FRP) was initially developed as the central document for the delivery of assistance to State and local governments in disasters or emergencies. It is a signed agreement among Federal departments and agencies that identifies actions that will be taken in the overall Federal response to disasters or emergencies. It is to be implemented in anticipation of an event that is likely to require Federal assistance and in response to an actual event requiring Federal disaster or emergency assistance. The FRP also may be implemented in response to a request made by a governor to the president for Federal assistance.

Federal response operations are coordinated with State, local, and regional officials and include positioning of a Federal coordinating officer, and deployment of emergency response teams, regional support teams, and emergency support teams to operations centers near the incident site and at the regional and national operations centers. See Figure 2.1 for a graphic depicting the FRP or go to http://www.dhs.gov/dhspublic/interweb/assetlibrary/NRP_Brochure.pdf for more detailed information.

Preserving lives and safety of victims are main priorities of disaster response. One component of the FRP, known as Emergency Support Function #8 (ESF #8), Health and Medical Services, is led by the Department of Health and Human Services (DHHS). This function is supported by 15 Federal and non-Federal agencies and provides coordinated Federal assistance to augment State and local resources after a major disaster or during the development of an anticipated public health and/or medical emergency. Assistance is also provided when State, local, or tribal public health or medical assets are overwhelmed and Federal support has been requested through proper authorities or when pending disasters are expected to overwhelm State, local, or tribal resources. Federal support also can be provided when these public health resources are not able to address all public health needs.

The scope of ESF #8 is broad and involves supplemental health and medical assistance to meet the needs of victims of a major disaster, emergency, or terrorist attack. In the FRP, this support is categorized in the following functional areas:

- Assessment of health/medical needs.
- Health surveillance.
- Medical care personnel.
- Health/medical equipment and supplies.
- Patient movement/evacuation.
- Patient care.
- Safety and security of human drugs, biologics, medical devices, veterinary drugs, etc.
- Blood and blood products.
- Food safety and security.
- Agriculture safety and security.
- Worker health/safety.
- All-hazard public health and medical consultation, technical assistance, and support
- Behavioral health care.
- Public health and medical information.
- Vector control.
- Potable water/wastewater and solid waste disposal.
- Victim identification/mortuary services.
- Protection of animal health.

Federal resources that supplement regional, State, and local response are primarily from within DHHS and ESF #8 support agencies. However, other non-Federal sources such as major pharmaceutical suppliers, hospital supply vendors, the National Foundation for Mortuary Care, and certain international disaster response organizations and international health organizations also provide support.

Department of Defense

The Department of Defense (DOD) has many capabilities through which it can provide assistance to lead Federal agencies in response to a disaster. DOD medical and support capabilities include the following:

- Triage and medical treatment.
- Displaced populations support.
- Hospital personnel augmentation.
- Epidemiologic support.
- Stress management.
- Preventive medicine support.
- Veterinary support.
- Prophylaxis and immunization augmentation.
- Mortuary affairs.
- Medical logistics.
- Transportation.

- Communication.
- Technical augmentation (e.g., modeling).
- Surveying and monitoring the incident site.
- Facility decontamination.

For more information about DOD and its capabilities, go to http://www.defenselink.mil/.

Summary

Federal assistance has been used successfully for many years to respond to the emergent medical requirements of victims of natural disasters throughout the United States. Recent terrorist attacks within the United States have increased the Nation's investment in homeland security through increased State, local, and regional health and medical resource development and acquisition in response to events involving weapons of mass destruction. Federal assistance is provided through the lead and supporting agencies designated within the NRP, including DoD.

While Federal response assets are robust, timely management focused on the needs of at-risk groups is paramount to victim survival and positive medical outcome. Pediatric patients are among the populations gaining national attention regarding the need for appropriate medical support, including appropriate supplies and equipment, during a disaster. Historical data on disaster response, including the personal experiences of health care providers and victims, reinforce the medical requirements of pediatric populations. All sectors—Federal, State, regional, and local—must continue to be prepared to provide medical resources to a disaster, but these entities also must give attention to the needs of specific populations, including children. This is the best way to improve morbidity and mortality in response to mass casualty events.

Bibliography

- Centers for Disease Control and Prevention. About CDC. Available at: http://www.cdc.gov/aboutcdc.htm. Accessed August 17, 2006.
- Centers for Disease Control and Prevention. Biological and chemical terrorism: strategic plan for preparedness and response. Recommendations of the CDC Strategic Work Group. *MMWR.* 2000;49(No. RR-4):1–14.
- Centers for Disease Control and Prevention. PHIN's Early Event Detection Component. Available at: http://www.cdc.gov/phincomponents/PHIN%20Brochure-%20BioSense%20.ppt. Accessed August 17, 2006.
- Centers for Disease Control and Prevention. CDC's Disease Surveillance System Efforts: Testimony of Joseph M. Henderson, Director, Centers for Disease Control and Prevention, Before the Select Committee on Homeland Security, Subcommittee on Emergency Preparedness and Response, U.S. House of Representatives, September 24, 2003. Available at: http://www.cdc.gov/washington/testimony/Bi924200355.htm. Accessed August 17, 2006.
- Centers for Disease Control and Prevention. CDC Clinician Registry. Available at: http://www.bt.cdc.gov/clinregistry/index.asp. Accessed August 7, 2006.
- Centers for Disease Control and Prevention. Epidemic Intelligence Service. Available at http://www.cdc.gov/eis/index.htm. Accessed August 17, 2006.

- Centers for Disease Control and Prevention. Epidemic Intelligence Service. Available at: http://www.cdc.gov/eis/index.htm
- Centers for Disease Control and Prevention. Laboratory Response Network. Available at: http://www.bt.cdc.gov/lrn/. Accessed August 17, 2006.
- Centers for Disease Control and Prevention. CDC Clinician Registry for Terrorism and Emergency Response Updates and Training Opportunities.Available at: http://www.bt.cdc.gov/clinregistry/index.asp. Accessed August 17, 2006.
- Centers for Disease Control and Prevention. Strategic National Stockpile. Available at: http://www.bt.cdc.gov/stockpile/index.asp. Accessed August 17, 2006.
- Department of Homeland Security Home Page. Available at: http://www.dhs.gov/dhspublic/index.jsp. Accessed September 13, 2006.
- Department of Homeland Security. National Response Plan. Available at: http://www.dhs.gov/interweb/assetlibrary/NRP_Brochure.pdf. Accessed July 7, 2006.
- Homeland Security Act, Public Law 107-296, 6 U.S.C. 101 et seq. November 25, 2002.
- Homeland Security Presidential Directive-1. Organization and Operation of Homeland Security Council. October 29, 2001. Available at: http://www.whitehouse.gov/news/releases/2001/10/20011030-1.html. Accessed August 17, 2006.
- Homeland Security Presidential Directive-2, Combating Terrorism Through Immigration Policies, October 29, 2001. Available at: http://www.whitehouse.gov/news/releases/2001/10/20011030-2.html. Accessed August 17, 2006.
- Hughes JM, Gerberding JL. Anthrax bioterrorism: lessons learned and future directions. *Emerg Infect Dis*. 2002;8(10):1013–14.
- Hutwagner L, Thompson W, Seeman GM, et al. The bioterrorism preparedness and response Early Aberration Reporting System (EARS). *J Urban Health.* 2003;S1;80(2):89–96.
- Landesman LY. Public Health Management of Disasters: The Practice Guide. Washington, DC: American Public Health Association; 2001.
- National Advisory Committee on Children and Terrorism. Recommendations to the Secretary. June 2003. Available at: http://www.bt.cdc.gov/children/recommend.asp. Accessed August 17, 2006.
- National Archives and Records Administration. Public Health Security and Bioterrorism Preparedness and Response Act of 2002. Available at: http://frwebgate.access.gpo.gov/cgi-bin/getdoc.cgi?dbname=107_cong_public_laws&docid=f:publ188.107.pdf. Accessed August 17, 2006.
- Perkins BA, Popovic T, Yeskey K. Public health in the time of bioterrorism. *Emerg Infect Dis.* 2002;8(10):1015–1018.
- Public Health Security and Bioterrorism Preparedness and Response Act of 2002, Public Law 107-188, U.S.C. 247d and 300hh, June 12, 2002, Draft National Response Plan. February 25, 2004. Department of Homeland Security. Available at: http://www.fda.gov/oc/bioterrorism/PL107-188.html . Accessed August 17, 2006.
- Robert T. Stafford Disaster Relief and Emergency Assistance Act, Public Law 93-288, Sections 5121-5206, et seq of Title 42 United States Code.

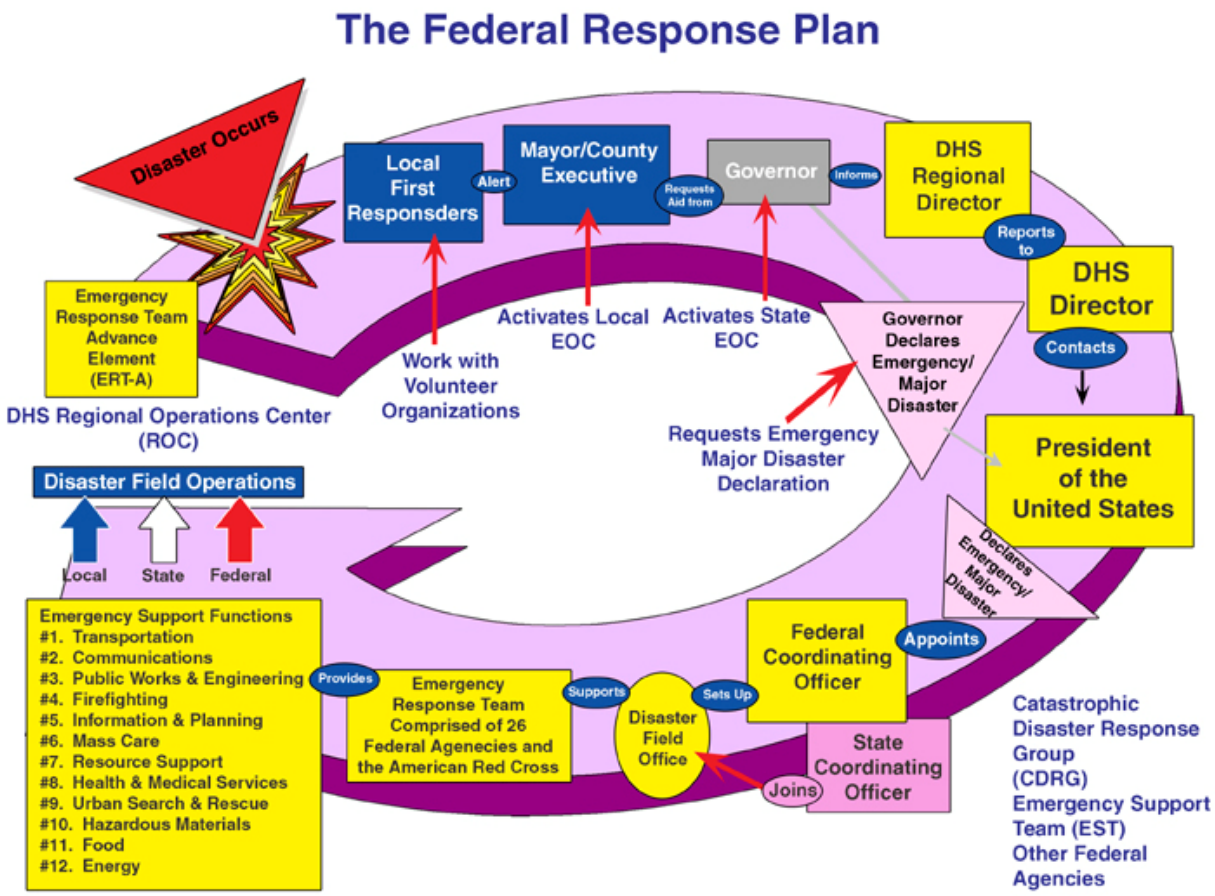


Figure 2.1 Federal Response Plan

EOC = Emergency Operations Center
DHS = Department of Homeland Security
ROC = Regional Operations Center

Table 2.1 Types of disasters, hazards, or events that may require Federal assistance

Type of event	Examples
Natural	Flood, earthquake, hurricane, tornado, typhoon, landslide, tsunami, ice storm, drought, wildfire, epidemic, disease
Accidental	Chemical spill, transportation accident, industrial accident, radiological incident, nuclear incident, explosion, utility outage
Civil/Political	Public demonstration, protest, civil disturbance, strike, mass immigration
Terrorist/Criminal	Chemical attack, biological attack, radiological attack, nuclear attack, high-explosive attack, war
Other	Inauguration, State of the Union, Olympics, major sporting event, summit conference, cyber attack

Chapter 3. Responding to a Disaster

Phases of Response

There are four basic phases of response to a disaster. They are:

1. Preparedness (including prevention and planning).
2. Actual response to the event.
3. Mitigation.
4. Recovery (short- and long-term) and critique.

Preparedness

Although we usually cannot predict disasters, we can control them through prevention and planning efforts. Prevention through preparedness is probably the most important phase of response in emergency management. During the preparedness phase, governments, organizations, and individuals develop plans to save lives, minimize disaster damage, and enhance disaster response. Preparedness efforts include preparedness and evacuation planning; emergency exercises and training; warning systems; emergency communication systems; public information and education; and development of resource inventories, personnel contact lists, and mutual aid agreements.

Physicians participate in preparedness and prevention in many different ways, including: immunization programs, dietary advice, health education, and safety precautions and planning. As participants in an emergency action plan, physicians need to help formulate ways of preventing incidents from occurring or limiting the consequences from an incident that has already occurred. Physicians need to know what will be expected of their hospital in the case of a potential infectious disease outbreak. They should also be prepared with the knowledge and resources needed to help identify the etiology of a problem and to provide timely treatment.

In the case of acts of terrorism, law enforcement plays the lead role in prevention, although physicians are often called upon to lend their expertise in an effort to identify the impact that various scenarios would or could have on the health of the community. Part of prevention consists of participating in a pre-established medical surveillance network of communities that will alert public health and safety officials of suspicious trends. A communications network of pediatricians, school nurses, freestanding pediatric walk-in clinics, and pediatric emergency departments should be formed.

Plans to share information within the Health Insurance Portability and Accountability Act (HIPAA) guidelines need to be developed and implemented so that patterns of illness can be tracked and investigated. An information-sharing network like this can provide invaluable data and assistance to public health and safety officials that will help them to make informed decisions about evacuations, quarantines, and any other planned responses to a biological or chemical incident. Law enforcement officials may also ask physicians to advise them on how best to educate and create awareness in school personnel and parent organizations without causing panic or mass hysteria.

Medical staffs need to know what will be expected of them and their facilities in the event of a large-scale infectious disease outbreak. Because of their unique knowledge base, physicians, especially pediatricians, can be very valuable sources of information to law enforcement and public health policymakers in helping to identify and isolate the source of an outbreak and providing guidance on the need for isolation, quarantine, and treatment.

Planning and prevention are closely related and work hand-in-hand. The people doing the planning cannot be strangers. It is important that they routinely and regularly meet and speak with each other to facilitate communication during a crisis. Communication is a key element for success. If managers cannot communicate successfully during routine circumstances, they cannot be expected to effectively communicate during times of crisis.

Plans should be developed and then tested and refined, over and over again. For a plan to work efficiently and effectively during a crisis, it must be well rehearsed. Plans that have been tested on a regular basis enable the responders to know and understand their roles. During a crisis is not the time to find out that a vital component is missing or nonfunctional.

Response plans must be shared with the people who will be doing the actual responding. Periodic in-service training should be conducted, including tabletop exercises with key players and full-scale field exercises. Pediatricians should be proactive in providing input regarding the unique needs of children during disasters and ensure that children's issues are included in all preparedness activities. Lessons learned from either actual responses or from the exercises and discussions should be incorporated into existing plans and then tested and evaluated again.

Planners need to ask themselves over and over again: "Are we ready?" Plans must be constantly changing and evolving to meet changing circumstances, and no matter how well prepared we think we may be, we can always be more prepared. Careful review and personal communication with all involved in both incident management and potential response can always identify more opportunities for improvement. Because disasters are dynamic events, plans must be flexible so that they can be adapted to an incident as it develops. People involved in the planning process should stay current regarding new trends, technologies, and intelligence information that become available. Planning done in a vacuum cannot be successful.

Actual Response to the Event

The next phase is the response to the actual event. Response activities provide emergency assistance for casualties, reduce the probability of secondary damage, and speed recovery. Response activities include activating public warning systems, notifying public authorities, mobilizing emergency personnel and equipment, providing emergency medical assistance, manning emergency operation centers, declaring disasters, evacuating the public, mobilizing security forces, providing search and rescue operations, and suspending laws on an emergency basis.

Response to a mass casualty incident (MCI) begins at the scene by first responders. An integral role of the first responder is coordination with agencies able to recognize characteristics of MCI secondary to bombs or to biological, chemical, or radiological agents, such that ongoing risk is minimized. First responders collect casualties, triage survivors, institute treatment (including

decontamination), and transport victims to emergency departments. In blast trauma, first responders should convey information to hospital personnel so that management of casualties can be facilitated. This information should include the sorts of injuries that are expected, initial estimates of the number of casualties, and any additional risks to personnel from toxic substances. Involvement of hazardous substances such as chemical or biological agents, fires, collapsed structures, or the possibility of a radiation dispersal device (dirty-bomb) should initiate specific response protocols.

Incidents can be very dynamic, so personnel should be able to adapt plans to deal with the incident as needed. There should be an incident commander—a qualified, visible leader—who can take charge of the response and direct the responders. The incident commander must be able to think quickly, make rapid assessments, and switch direction as needed without holding a lengthy caucus. The incident commander should be surrounded by competent, knowledgeable, and trusted people. They will be called upon to provide complete and accurate information to the incident commander so that he or she has the tools needed to make rapid, informed decisions. The National Incident Management System (NIMS) provides the framework needed to successfully manage an incident. This is a standardized plan that allows for flexibility. The NIMS can be used by local, state, and federal authorities to use resources depending on the nature and the scope of the incident. The NIMS is available online at http://www.dhs.gov/interweb/assetlibrary/NIMS-90-web.pdf.

Mitigation

The next phase of response is mitigation, in which actions are taken to stop the incident from doing any further damage and to stabilize the situation. Although disasters cannot always be predicted, their consequences often can be controlled by preparation and planning. Mitigation activities are also important in the preparedness phase, where they can eliminate or reduce the probability of a disaster or reduce the impact of unavoidable disasters. The damage done can be limited or confined using the dynamics of the incident management plan.

Mitigation preparedness measures include building codes, vulnerability analyses, tax incentives and disincentives, zoning and land use management, building-use regulations, safety codes, sharing of resources among States, preventive health care, and public education. Information resources, data, and services important in mitigation activities include: geographic information systems (GIS)-based risk assessment, claims history data, facility/resource identification, land use/zoning, building code information, and modeling/prediction tools for trend and risk analysis.

Recovery and Critique

The recovery phase evolves as steps are taken to mitigate the event. The objective of recovery is to return things to normal as quickly as possible, and recovery activities continue until all systems have been returned to normal or better. Depending on the scope of the incident, the recovery period can range from hours to years. Damage assessments are made, financial needs are identified, and timelines and plans are developed and implemented.

Short- and long-term recovery measures include returning vital life-support systems to minimum operating standards; reconstruction; temporary housing; ongoing medical care; and public

information, health and safety education, and counseling. One aspect of long-term recovery involves assessing the infrastructure, how it held up during the incident, what the cost of the response was, and how that cost can be recovered. Recovery efforts in economic support include paying out insurance/loans and grants to cover damage, providing disaster unemployment insurance, and performing economic impact studies. Information resources and services related to recovery include data collection related to rebuilding, claims processing, and documentation of lessons learned.

During long-term recovery, participants also review and critique the response, evaluating how the overall plan worked in a real event, determining what needs to be done to update the plan and educate responders, and making changes necessary to improve the original response plan and prevent a recurrence.

Regional Response

State and Federal

Communication and information sharing are key parts of successful incident management—both before and during an actual event. Although each area of the country handles emergency responses in somewhat different ways, all emergency response agencies use some form of an incident management system. Almost all use the NIMS with unified command.

Regional physicians should review community emergency response plans, as well as the collaborative efforts between responders and planners designated by pertinent emergency response agencies. Local physicians should become familiar with the following:

- The response agencies in their area.
- Regional medical, operational, and administration protocols.
- The various levels of training and the roles of all the different responders in a mass casualty incident, and how these change with the changing situation. For example, some responders may move victims only after they have been triaged by other more highly trained responders. In a different scenario, those same responders may actually move victims before triage because of unstable conditions (e.g., structural collapse, hazardous materials release). The dynamics of the incident dictate whether triage or transport is done first.
- Whether the firefighters or police officers in their community are certified emergency medical technicians (EMTs) or paramedics.
- The person who is in charge of an incident when there is a multi-agency response.
- Where to go for information and to offer assistance during an actual emergency.
- The regional and State emergency planning interface. Each state has its own unique emergency planning office and incident management system that ties in with the overall National Response Plan (see Figure 3.1).

Emergency Medical Services

The availability and capabilities of emergency medical services (EMS) in the United States have undergone explosive growth over the last 40 years. The Comprehensive Emergency Medical Services Systems Act of 1973 established the regional basis for coordination of emergency

medical care throughout the United States. In addition, the National Highway Traffic Safety Administration (NHTSA) is charged with coordinating the development of national standard curricula for and education of EMTs.

During the past 25 years, the scope and complexity of care rendered by prehospital EMS providers have expanded greatly. Four levels of prehospital emergency medical personnel are currently recognized, in order of increasing skill level with respect to the care of the trauma patient (Table 3.1).

In recent years, the addition and expansion of initial and continuing education in prehospital pediatric trauma care (such as that provided by the *Pediatric Emergencies for Prehospital Professionals* [PEPP] course of the American Academy of Pediatrics), as well as the provision of expert pediatric medical direction, have greatly enhanced the capabilities of most regional EMS systems. In most regions, injured children can now receive emergency medical assistance comparable to that of injured adults.

Hospitals

In mass casualty incidents, including those involving release of biological or chemical agents, both children and adults are likely to be significantly affected. Children would probably be disproportionately affected by such an incident, so pediatricians should assist in planning coordinated responses for local hospitals that may have limited pediatric resources (see Chapter 1). Health care facilities could also be a primary or secondary target. At the very least, facilities will be overwhelmed by a massive number of anxious and worried individuals.

The problems associated with terrorist incidents differ from those usually faced by hospital disaster alert systems. In the typical scenario, most victims are triaged in the field and then carefully distributed among available resources, to avoid a single facility from being overwhelmed. In a terrorist attack, facilities will be particularly vulnerable to inundation with many victims who have not been appropriately triaged or transported by EMS. Arrivals without full notification could interfere with attempts to isolate contaminated victims and ensure protection of health care personnel. In addition, terrorist events will be further complicated by the issues of security and forensics.

Hospital emergency department personnel become involved both before and after the arrival of victims. Activities prior to arrival include processing current patients in the emergency department to prepare for new arrivals, checking all equipment, activating additional personnel, assigning team leaders, and possibly assigning liaisons to government agencies. On arrival of patients, emergency department staff should ascertain (whenever possible) a victim's location with respect to detonation, whether a victim was within an enclosed space or near a body of water, or whether the victim was crushed by debris. These data provide valuable information as to the degree of injury to expect in other victims.

Triage is crucial, given the large number of minimally injured and ambulatory victims presenting to emergency departments after a terrorist incident. The importance of triage is highlighted by the Oklahoma City experience in April 1995. This explosion caused 759 casualties, of whom 167 died, 83 were hospitalized, and 509 were treated as outpatients either in an emergency room or

by private physicians. Approximately 85% of the 592 survivors sustained non-life-threatening soft-tissue injuries (including lacerations, abrasions, contusions, and puncture wounds), and 35% sustained musculoskeletal injuries (including fracture/dislocations and sprains). The 66 children who were injured in the Oklahoma City blast showed a similar pattern of soft-tissue and musculoskeletal injury.

The first wave of patients from the Oklahoma City blast arrived either by ambulance or some other means of transportation within 15 to 30 minutes of the event. Medical systems were overloaded with minimally injured patients. As would be expected, hospitals closest to the attack were overwhelmed first. More seriously ill, non-ambulatory patients tended to arrive later because of the delay associated with field triage and transport via EMS. The experience after the World Trade Center attacks in 2001 was similar in that the vast majority of patients seen in emergency departments were ambulatory and were treated for minor soft-tissue injuries and released. However, hospital overload was mitigated somewhat due to the large number of fatalities, which decreased the number of survivors presenting for treatment. The main lesson to be learned from these experiences is that casualty profiles are event specific, but an effective triage system can better direct attention toward the critically ill.

Regional coordination. The objective of risk assessment is to estimate the likelihood that an incident will have an impact on the hospital, as well as the size of that impact. Considerations in risk assessment include the following:

- Attack has the potential to generate large number of causalities.
- Effects may be immediate or delayed.
- Response will require specialized equipment, procedures (decontamination), and medications, all adapted to pediatric needs.
- Hospitals may be targets of secondary attacks to amplify effect.

Situations with both high probability and the potential for high impact (e.g., an earthquake in California, or a tornado in the Midwest) should receive more attention in preparedness planning than either situations of low probability with the potential for high impact (e.g., industrial plant chemical leak) or situations of high probability and the potential for low impact (e.g., community outbreak of infectious gastroenteritis).

Hazard vulnerability analysis (HVA) is an aspect of risk analysis that considers the hospital's capabilities regarding the traditional elements of risk. This analysis allows a comparison between the potential risk factor (hazard) and the hospital's ability to cope. The action plan resulting from this type of risk analysis should be directed toward those hazards against which the hospital is less able to cope (i.e., vulnerabilities). Areas of vulnerability may include attack on hospital information systems, inadequate ventilation systems (negative pressure, contained exhaust) for decontamination procedures in toxic exposures, hospital staff untrained in the proper use of personal protective equipment (PPE), and so on.

The key benefit of HVA analysis is the ability to prioritize planning for the hospital in any given situation. The key to effective HVA is a good, frequently updated inventory of the resources and capabilities (within both the hospital and the community) that are available for dealing with a particular hazard-related emergency.

Surge capacity. Most of our medical systems operate at near capacity in normal times. Pre-event planning and preparedness are essential to develop local capacity and expand health care resources to respond to increased needs. Surge capacity should be created on all levels, including the following:

- Emergency department space.
- Decontamination equipment.
- Antitoxins and medications.
- Hospital bed capacity.
- Extra provider capacity.
- Increased integration back into a community that can provide mental health services.

In general, hospitals should plan to be self-sufficient for the first day or two after an incident. Most victims in the first 24 hours will be anxious or worried individuals who may or may not need decontamination before medical treatment. Assessment of hospital capacity for these victims is essential. Several teams in small areas can perform triage and rapid treatment. A system should be established to initially treat victims and then assign them to other facilities (away from the main site) for definitive treatment. There should also be followup to ensure that appropriate care is available at the other facilities. A system should also be established to rotate and supplement staff for the first 24–48 hours (or longer) until additional medical help can arrive.

The following points should be considered in measurement and management of surge capacity:

- Surge capacity expressed in terms of beds is not specific enough. Specific pediatric surge capacity that is somewhat intervention-specific is preferable. For example, there may be 1,000 hospital beds available in a large community but only 10 pediatric intensive care unit beds. If these types of pediatric-specific resources are needed, the actual surge capacity is only 10 beds.
- Non-disaster-related patients must be cared for in addition to disaster victims. Surge capacity and overall planning should accommodate both sets of patients.
- Surge capacity and capabilities are determined by many factors (e.g., facilities, human resources, patients' needs, legal and regulatory issues, policies, process design, supplies, equipment, etc.). Each factor should be systematically considered and optimized. A "bottleneck" in any factor can become the limiting condition. Poor management of these issues can affect outcomes more than the skill of the health professionals caring for individual patients.
- Assumptions that pediatric patients will be cared for by adult health providers and facilities are not universally true or necessary in at least some situations.
- Local contexts differ regarding inpatient capacity for high-acuity pediatric patients. In large urban areas, there are likely multiple pediatric hospitals within a short distance of each other. They can collaborate and probably handle patients from all but the largest of disasters. However, many communities have only one facility that may be a significant distance that is capable of handling high-acuity pediatric cases. These facilities often operate near or even above capacity many days each year. So, surge capacity and capability for pediatric but not adult disaster victims may be critically limited. Transporting pediatric patients to facilities outside of the region may be beneficial or

even required (particularly if a pediatric facility is damaged or incapacitated). Pediatricians should educate and advocate regarding this type of planning. This is similar to the situation for high-end pediatric cardiac surgery, organ transplantation, and burn unit care for which pediatricians already refer to resources outside their region.
- The frequent practice of making superheroic efforts at an overwhelmed hospital needs to be considered against the risk/benefit and outcomes of transferring patients to hospitals that are not overwhelmed. Generally, pediatric capability and capacity are available, but they may be at distant facilities.
- Agencies other than hospitals may be needed to care for unaccompanied but otherwise medically stable children or for children with social but no serious physical medical issues. This will not occur unless pediatricians help the responsible agencies prepare in advance.

Protection of personnel and levels of precaution. Hospital staff members are at high risk for secondary exposure from contaminated victims (e.g., skin, clothing, etc). The Occupational Safety and Health Administration (OSHA) provides protective standards for hospital response, including:

- A written plan describing how contaminated patients will be managed.
- An Incident Command System described for each type of hazard.
- On-the-spot training and briefing for support personnel, such as physicians.
- A plan for providing exposed employees with medical care and surveillance.
- Training at a first-responder level for employees involved in decontamination operations, including training in hazard containment and prevention of spread.

Biological agents are generally associated with a delay of hours to days in onset of illness. Therefore, illness may go unrecognized in the initial stages, which can result in widespread secondary exposure to others, including health care personnel and other patients. In this situation, containment of the exposing agents in negative-pressure environments is mandatory. In contrast, toxins derived from biological agents produce illness within hours of exposure. The patient exposed to a toxin does not usually pose a significant threat of secondary exposure to medical personnel, although decontamination may still be warranted (as in chemical exposure).

PPE includes specifically designed barrier clothing (e.g., gown, boots, and gloves) to protect the skin and a mask to protect the respiratory tract. Clothing is designed to provide protection against liquids, vapors, dust, and particles. Respiratory masks fall into two categories: those that filter the ambient air to rid it of hazardous particles, and direct-line masks that provide pure air under pressure.

Chemical weapons are intended to produce immediate discomfort, incapacitation, or death. Incapacitating chemical agents may be particularly toxic to small children. The mainstay of decontamination is rinsing with water, shedding exposed clothing, and in some instances, administering pharmacologic antidotes.

The risks of contamination are usually recognized at the scene, so that personnel at the receiving hospital can be alerted. However, hospital personnel are at particular risk of contamination from exposure, due to the high number of anxious or worried victims who arrive at the hospital on

their own without previous triage or information on risk factors from the incident scene. Health care personnel and any adjunct personnel in contact with victims or the hospital decontamination site should wear full PPE and self-contained breathing apparatus until the risk of exposure by secondary contamination is completely eliminated. Equipment used for universal precautions, such as surgical masks and latex gloves, are inadequate. Recognition of all agents involved in the exposure and determination of their toxic potential often take time and close coordination with the regional poison center, the fire department, and the CDC. Hospital personnel responsible for decontamination and protection should remember the possibility of more than one agent being used in an assault and also the possibility of terrorists using a "decoy" agent to mask and delay recognition of release of a more toxic or lethal agent.

Radiological or nuclear agents are generally associated with a delay in onset of illness. As with biological agents, illness may go unrecognized in the early stages, so that the risk of contamination of hospital personnel by secondary exposure to radiation carried from the scene is significant. Contamination varies with the emission levels.

PPE for radiological agents includes clothing barriers that prevent radioactive particles from reaching the skin. Any mask that will prevent dust from reaching the respiratory tract is protective. Gamma and neutron emitters penetrate clothing easily and require lead-type barriers. Lead aprons, such as those used for routine radiology, are not feasible for protection. Some exposure of hospital personnel may be unavoidable, and in these instances, the radiation exposure should be monitored and limited to safe doses.

Potential problems with use of PPE include the following:

- Bulky and cumbersome.
  - Impedes bending, kneeling to reach small children, infants.
  - Impedes nimble use of hands and fingers (needed for starting an IV line, intubating, drawing up medications, etc.).
  - May not be adapted to stethoscope use.
- Poor ventilation and temperature control.
  - Profuse sweating, discomfort.
  - Potential fluid losses and dehydration.
  - Hyperthermia (for personnel working in warm environments [outside tents, hospital air conditioning system down], or working over-extended hours).
- Unfamiliar "alien" appearance.
  - Frightens children.
  - Contributes to stress of the crisis.

For additional information on PPE, see Chapters 5 and 6.

Incident Command Systems

Incident command systems (ICS) use a consistent organizational structure that includes individual positions for overall management of emergency situations. ICS systems are designed to facilitate interagency coordination (because each agency has organized their response on the same model). This is one of the system's most important advantages. ICS can also expand and contract to meet the needs of the particular emergency situation at hand.

ICS structure is hierarchical. For example, there will be one incident commander, three key assistants (safety officer, liaison officer, and public information officer), and four subordinate managers who report directly to the incident commander (operations, logistics, planning, and finance). Go to http://www.fema.gov/pdf/nims/nims_training_development.pdf for more information on developing an ICS.

San Mateo County Emergency Services developed their Hospital Emergency Incident Command System (HEICS) in the 1990s to facilitate earthquake preparedness among California hospitals. This HEICS provides a useful example of a system that employs the concept of "unified command," with establishment of an emergency operations center within the hospital, pre-designed job action sheets, response activities, lines of communication, and reporting relationships. The HEICS structure is modeled on the ICS hierarchy. Key participants in the hospital ICS include the following:

- Hospital chief executive officer.
- Vice president of operations.
- Medical director.
- Emergency manager.
- Community affairs director.
- Critical care manager.
- Emergency department manager
- Hospital communications.
- Facilities and engineering.

For more information on the HEICS, see http://www.emsa.cahwnet.gov/dms2/heics3.htm.

Regional Coordination of Hospital Response

Emergency incidents also require hospitals to coordinate with community and medical stakeholders within the regional area. Coordination with community stakeholders includes liaison and planning with various local, State, and national agencies/organizations within the region:

- Primary/prehospital/infrastructure Response:
  - EMS.
  - Fire.
  - Police, local environmental protection agency, sheriffs.
  - Military (local or regional).
  - Regional poison centers.
  - Local health department.

- Community/citizen response:
  - Schools, public and private.
  - Day care units, public and private.
  - Service groups (Kiwanis, Rotary, Salvation Army, parent/teacher associations [PTAs], etc.).
  - Nonsecular groups (churches, synagogues).

- Public recreation administrations (zoos, amusement parks, sports stadiums, museums, and the like).

Regional coordination with medical stakeholders includes liaison and planning with various medical entities within the region:

- Children's hospital-based:
  - Pediatricians and pediatric subspecialists.
  - Pediatric nurse practitioners, physician assistants.
  - Administration.
  - Ancillary services (nursing, technicians, etc.).
  - Air/ground transport services.
  - Laboratory services.
  - Children's services.
  - Support services (dietary, environmental).
- Community-based private practitioners:
  - Pediatricians.
  - Family practice physicians.
  - Emergency medicine physicians.
  - Nurse practitioners.
  - Physician assistants.
  - Other types of physicians and health care providers.
- Community/ regional hospitals:
  - Emergency department staff.
  - Hospitalists.
  - Surgeons.
  - Anesthesiologists.
  - Administration.

Some Roles for the Pediatrician in Regional Hospital to Community Planning

- Meet with hospital planners to ensure children's needs are met, equipment is adequate, and contingency plans are in place.
- Be present when hospitals work with prehospital groups on triage, stabilization, equipment, distribution, etc.
- Help organize the response of community pediatricians to assist at the hospital and local secondary areas to redistribute care back to the community level.
- Help coordinate the identification and movement of community pediatricians. Assure that local pediatricians have the appropriate identification needed to cross barriers, along with designated means of transportation to areas where care is needed. Local pediatricians should also be familiar with the regional disaster management plan, reporting requirements, and contingency plans for alternate forms of communication, as needed.
- Plan for primary offices to initially use alternative areas, e.g., Disaster Medical Assistance Team (DMAT) field units, school gyms, etc.
- Help develop education and assistance packets for family preparedness.

- Provide community education so families are prepared with the basics, such as flashlights, alternative heating, lighting, water, food, and clothing.
- Be familiar with protocols for the following:
    - Isolating and decontaminating victims.
    - Mobilizing additional staff.
    - Potentially using secondary-care sites (e.g., school auditoriums).
    - In-hospital care protecting existing patients, as well as medical and ancillary staff (e.g., cafeteria workers).
    - Use of reverse-ventilation isolation areas.
    - Use of decontamination showers with separate water collection systems.
- Coordinate with the local educational system because children spend most of their time in school. Know plans for rapid evacuation and holding areas where triage and initial treatment of severely injured victims can begin.
- Work with hospitals and schools to develop decision trees for the initial steps of decontamination, further triage, transport, and so forth.
- Know designated sites for stockpiled antidotes, antibiotics, vaccines, and other drugs and routes for rapidly obtaining them from outside the hospital if necessary (e.g., CDC).
- Help develop protocols for proper doses of vaccines and antidotes for use in children.
- Coordinate with teams of health professionals dealing with post-event programs, including rehabilitation, posttraumatic stress syndrome (PTSS), and critical incident stress management for health care professionals.
- Perform triage of pediatric victims, including those who arrive at the scene with EMS and those who arrive at the hospital without previous triage.
- Help hospitals develop color-coded triage systems for adult and pediatric patients that arrive without previous triage. Systems should ensure that children are not separated from their caregiver(s) during the chaos (unless for valid medical reasons).
- Help address long-term needs, such as counseling (e.g., for PTSS), rehabilitation, social support (e.g., orphaned children), triage systems, etc.
- Work with local pediatricians to coordinate protocols for a variety of emergency processes and procedures, including disaster system management, procurement of PPE, setting up decontamination areas, hospital reporting, identification of sentinel cases, and post-incidence stress reduction. These protocols can be communicated by a variety of methods, including seminars for disaster management on the local level. These efforts should be coordinated with the hospitals and government agencies involved.

Drills and Quality Assurance Activities

It is essential to organize disaster drills in the hospital that are coordinated with community resources. These drills should include the following scenarios:

- The incident is small and contained, with most patients triaged and transported by the EMS systems to designated facilities.
- The facility is inundated with large numbers of anxious and worried individuals, both immediately and for a short time after the incident.

Drills should include Disaster Life Support Teams performing their respective functions.

The goal of disaster life support training is to standardize incident response across the Nation as a way of strengthening national public health. Basic disaster life support training provides a didactic review of all-hazard topics, including critical information on the role of the health care professional. Advanced disaster life support extends this training into incident-specific scenarios (e.g., decontamination of mass casualties) through didactic training and interactive sessions and drills. Drills for advanced disaster life support teams should include the following:

- Human pediatric simulator scenarios including decontamination for biological, chemical, and nuclear hazards.
- Essential clinical skills, performed in central and mini clinical areas.
- Use and wear of PPE.
- Implementation of the incident command center.

Integration with Children's Services

The pediatrician's strongest role can be in helping hospital disaster planning teams anticipate and manage pediatric victims who have been separated from their primary caregivers during a disaster. These children need immediate support until a definitive caregiver can be located.

In the event that in-hospital support services are overwhelmed, other efforts will also be needed, including the following:

- Alternative social support from the community. Community-based organizations can provide clothing, toys, and bedside sitter support and communicate with family members who may be out of town.
- Psychological support services from the surrounding community that can be brought to the hospital.
- Outreach support teams, using community members, pediatricians, and mental health providers. These teams can go into the communities, schools, daycare centers, churches, etc., to provide stress debriefing, triage for further mental health care, and long-term monitoring.

In the event that community support and child-protective services are also overwhelmed, additional efforts will be needed, including the following:

- Alternative plans need to be in place to cope with a large group of children needing immediate caretaker support (i.e., those who are orphaned or temporarily separated from caretakers because of decontamination or medical treatment needs).
- Pediatricians can help families find alternative systems within their churches and neighborhood communities.
- Pediatricians can create information cards of resources and supervise rehearsal scenarios.

Pediatricians also can help communities plan to provide other support services for families and children:

- Establish a plan with existing communication systems (e.g., television, radio) to provide ongoing information support.
- Plan for non-medical family support centers to provide water, food, clothing, etc.
- Plan for a system to notify next of kin (anticipation of this information system can be done by pediatricians).

- Provide for crisis counseling.
- Provide for legal services.
- Provide for translation services.
- Facilitate the implementation of State and Federal disaster relief programs.
- Plan for temporary housing alternatives, immediate, short, and long term.
- Conduct community memorial and grieving services.

JCAHO and Emergency Management

In January 2001, the Joint Commission on Accreditation of Healthcare Organizations (JCAHO) introduced new disaster preparedness standards, building on its prior position that health care organizations should be prepared for all types of emergencies. These standards represent an important evolution in the concept of managing emergencies. Health care organizations are expected to address the four specific phases of disaster management—preparedness, response, mitigation, and recovery—and conduct hazard vulnerability analysis. This "all-hazards" approach should result in a thorough review and risk analysis of credible hazards and serious threats to the facility and its surrounding community, as well as the subsequent development of plans to address the ramifications of all possible hazards. After plans are developed, the organization should implement and execute them by conducting training and drills.

The new JCAHO Guidelines require hospitals to use formal emergency management processes as the foundation of their planning. These include the following:

- Develop a hazard vulnerability analysis (risk assessment).
- Assure the operational needs of the hospital facility and ample resources of critical services such as water, electricity, sewage services, and ventilation.
- Integrate with local emergency response systems.
- Identify alternative roles and responsibilities of personnel.
- Establish a command structure, also known as an "incident command system."
- Perform ongoing monitoring of plan performance, including annual reevaluation.

JCAHO has published an emergency planning guide for use by officials in small, rural, and suburban communities. The guide is available online at http://www.jointcommission.org/NR/rdonlyres/FE29E7D3-22AA-4DEB-94B2-5E8D507F92D1/0/planning_guide.pdf..

Appropriate Triage

Triage to the appropriate facility can be critical for a child. Therefore, the development and implementation of a mass casualty plan that considers the unique needs of children is imperative to avoid overloading one local facility (whenever possible). The community should have designated facilities for referral of critically injured pediatric patients. At the same time, each facility should be able to care for and at least initially stabilize both children and adults. Pediatric skills and equipment should be maintained at all facilities.

Emergency departments and hospitals should have some mechanism to provide for the physical, as well as the medical needs of pediatric populations. These needs include shelter, clothing, food,

supervision, and entertainment for pediatric victims, as well as protection from the media in the critical period immediately after a disaster.

Incident Management

The incident command system (ICS) is widely recognized and used in the field of emergency management to effectively manage resources and casualties during a disaster. The ICS is a unified management system that allows for expandability and accountability based on the magnitude and needs created by the specific incident. It is an organizational system of "best practices" for on-scene, all-hazard incident management. There are five major management functions:

- Incident command.
- Operations.
- Planning.
- Logistics.
- Finance/administration.

All have standardized position titles. The following nomenclature is used for organizational components and their supervisory personnel:

- Incident commander.
- Command staff officer.
- General staff/section chief.
- Division supervisor.
- Group supervisor.
- Branch director.
- Task force leader.
- Strike team leader.
- Unit leader.

In a unified command structure, there is one recognizable leader—the incident commander—who has overall responsibility. Because the system is expandable, it allows for the use of many components that may be needed to manage the incident. Various component managers are granted the authority to manage their specific component, and they are held accountable for the performance of their area to the incident commander.

This type of system is widely used in everyday business. For example, in the hospital setting, the incident commander could be compared with the chairperson of the medical board, and the group managers could be compared with the various chiefs of service.

The National Incident Management System (NIMS) has been developed for use by all emergency response agencies in the country. NIMS is an updated more inclusive version of ICS. Its standardized framework, common terminology, and flexibility allow it to be used by Federal, State, and local agencies/authorities.

ICS is modular and can be expanded to meet needs that arise during an incident. In a disaster, the triage, treatment, and transportation of casualties fall to the EMS Operations Branch. The

incident command officer is most often a field officer of the local EMS agency. That officer is responsible for managing all medical resources needed to effectively handle the incident and for minimizing the impact on normal operations of the EMS system. Based on the scope of the incident, the divisions of smaller functional units are formed to manage the following:

- Staging.
- Triage.
- Treatment/decontamination.
- Transport.

Staging

The staging officer is responsible for setting up an area where incoming resources, including personnel, can gather and await an actual assignment. The staging group can be broken down further into smaller units of operation for specific incident needs. For example, incoming ambulances would be directed to a vehicle staging area; personnel not assigned to a specific unit or task would be sent to a personnel staging location; and needed equipment would be directed to a logistical staging location. These areas may be off site from the incident but easily accessible to it. This allows greater accountability of who is on the incident ground and monitoring of their respective functions and performance.

Triage Group

The triage group is tasked with prioritizing casualties based on pre-established medical protocol. Simple Triage and Rapid Transport (START) allows rescuers to assess and prioritize a victim with a 30-second, hands-on assessment. Immediate life-threatening conditions are rapidly identified and corrected with minimal intervention, and casualties are identified for immediate transport (e.g., airway problems are corrected with a tilt of the head, and the patient is marked "red" for immediate transport; intubation in the field would not be done during this initial assessment). For children, a variant known as JumpSTART pediatric mass casualty triage is used (see http://www.jumpstarttriage.com/).

In incidents involving victims exposed to hazardous chemicals, a similar system of triage is used. Normal START procedures would be extremely difficult for rescuers in chemical protective clothing to use. In these cases, a form of triage based on observation of symptoms is used, and response to tactile stimulus determines the triage priority.

Treatment/Decontamination

The treatment group is tasked with providing a more definitive treatment regimen for incident victims. Casualties are removed from the incident ground to a safe, protected area so that treatment can be started, particularly if transport is to be delayed. Personnel in this group constantly monitor victims for changes in their condition and change their triage priority as needed. In large-scale incidents in which the patient load would cripple normal hospital resources, victims with minor injuries may be held in an off-site treatment area for an extended time. Physicians, nurses, and other ancillary staff may be assigned to staff this area.

Victims that have been exposed to a chemical or biological agent should be decontaminated. Personnel assigned to this task should be trained hazardous materials technicians because they need to operate in chemical protective equipment. This may be as simple as a splash suit and gloves or as complex as a fully encapsulated suit and positive-pressure breathing apparatus, depending on what type of chemical or agent the victims were exposed to.

Transportation

The transport group is tasked with the responsibility for transporting victims from the incident ground to field treatment centers, hospitals, or specialty referral centers (trauma, burn, replant, etc.). The group officer usually maintains listings of available hospital beds and medical evacuation and public transportation resources that may be needed to move victims. The transport group also performs the critical function of maintaining the log of destinations to which victims have been moved so that they may be tracked for public information and quality assurance reasons.

Bibliography

- Comprehensive Emergency Medical Services Systems Act of 1973. Washington, DC: United States Congress, Senate Labor and Public Welfare Committee; 1973.
- Department of Homeland Security. National Incident Management System. March 2004. Available at: http://www.dhs.gov/interweb/assetlibrary/NIMS-90-web.pdf. Accessed July 10, 2006.
- JumpSTART. Combined JumpSTART algorithm. Available at: http://www.jumpstarttriage.com/. Accessed August 17, 2006.
- National Health Professionals Preparedness Consortium: Healthcare leadership and administrative decision-making in response to WMD incidents. *Nobel Exercise Scenario Information*, v.2.0. December 31, 2002.
- President's Disaster Management Egov Initiative. Available at: http://www.whitehouse.gov/omb/egov/c-2-2-disaster.html. Accessed August 17, 2006.
- San Mateo County Emergency Services. Hospital Emergency Incident Command System (HEICS) Update Project. January 1998. Available at: http://www.emsa.cahwnet.gov/dms2/heics3.htm. Accessed July 10, 2006.
- U. S. Department of Justice, Office for Domestic Preparedness. Hospital Emergency Management—Concepts and Implications of WMD Terrorist Incidents. Washington, DC: U.S. Department of Justice; April 2002.

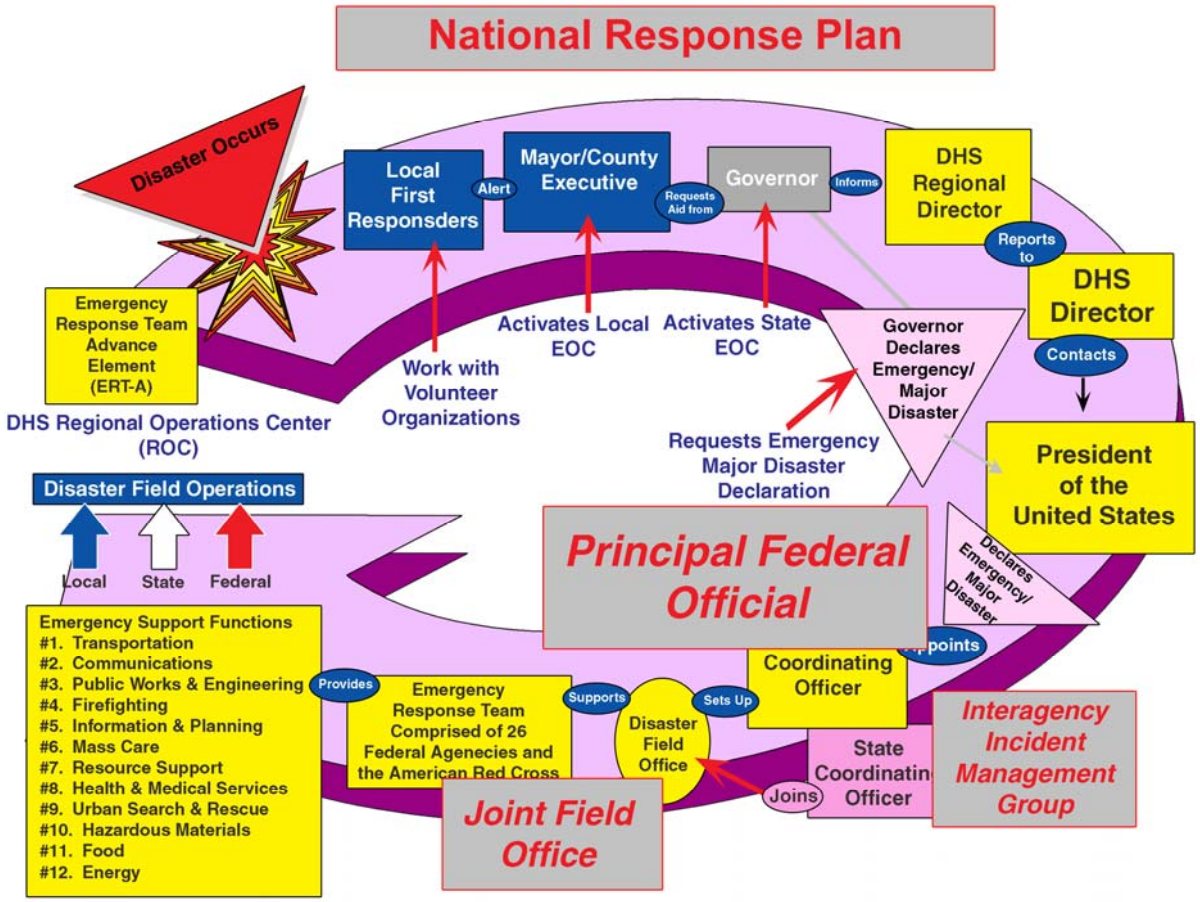


Figure 3.1 National Response Plan

DHS = Department of Homeland Security
EOC = Emergency operations center
ERT = Emergency response team
ROC = Regional operations center

Source: Courtesy U.S. Department of Defense

Table 3.1 Training and competencies of prehospital emergency personnel

Prehospital emergency personnel	Hours of training (approximate)	Medical procedures able to perform
First responders	40	Basic airway support, including use of supplemental oxygen, airway adjuncts, and bag-mask ventilation Direct pressure bleeding control Cervical spine stabilization Cardiopulmonary resuscitation and automated external defibrillation
Basic EMTs	120	All of the above, *plus*: Full spinal immobilization Splinting of extremities Management of open chest wounds and impaled sharp objects Emergency ambulance transport
Advanced EMTs	300–400	All of the above, *plus*: Endotracheal intubation Intravenous fluid administration
Paramedics	1000–1200	All of the above, *plus*: Needle cricothyroidotomy Needle decompression of tension pneumothorax

Chapter 4. Biological Terrorism

Background

History of Bioterrorism

Although recent world events have heightened awareness of bioterrorism and biowarfare, there are many historical accounts of both. During the middle ages, the Tartars are reported to have catapulted plague-infested cadavers into the walled city of Caffa. In the 15th century, Pizarro supplied the native people of South America with smallpox-contaminated clothing in an effort to gain control of land. During the Spanish-American war, the Spaniards are alleged to have supplied the American Indians with blankets infected with smallpox virus. Japanese researchers have admitted to feeding cultures of *Clostridium botulinum* to Chinese prisoners of war during the 1930s. During WWII, the Japanese again used bioterrorism against the Chinese when they dropped plague-infested fleas over China, causing outbreaks of plague.

Two more recent bioterrorist events in U.S. history involved contamination of the food supply. In 1984, in The Dalles, OR, an outbreak of 751 cases of *Salmonella typhimurium* was linked to the intentional contamination of restaurant salad bars by members of the Rajneesh religious cult. In 1996, an outbreak of *Shigella dysenteriae* type 2 among laboratory workers at a large Texas medical center was traced to muffins and donuts anonymously left in the break room. *Shigella* isolates from infected victims matched those found in an uneaten muffin and in the laboratory's stock strain.

There are reports that the Japanese cult Aum Shinrikyo has attempted bioterrorist attacks across Tokyo using anthrax, sarin gas, and botulinum toxin. In 1994, this cult succeeded in sickening 500 people and killing 7 with sarin gas. In 1995, again using sarin gas, Aum Shinrikyo injured 3,800 people and killed 12 by releasing the gas in five subway stations around Tokyo.

The anthrax attacks of October 2001, propagated through the U.S. Postal Service, led to infections in 22 people (11 cases of cutaneous anthrax and 11 cases of inhalational anthrax) and 5 deaths. These attacks affected thousands of people around the world, including those who were presumed exposed and required antibiotic prophylaxis and/or vaccination; the numerous anxious and worried individuals who flooded hospital emergency rooms, physicians' offices, and public health information hotlines; and the thousands of public health, medical, and law enforcement workers who investigated potential attacks.

For a video about the history of bioterrorism, see
http://www.bt.cdc.gov/training/historyofbt/index.asp.

Bioterrorism research. Beginning in the 1920s, the Soviet biowarfare program reportedly conducted research on gas gangrene, tetanus, botulism, plague, and typhus. In the 1970s, this program was greatly expanded as the secret organization Biopreparat. At its height, this program involved 60,000 people working in more than 50 facilities across the former USSR. Plague, anthrax, smallpox, tularemia, brucellosis, glanders, Marburg virus, and Venezuelan equine encephalitis (VEE) virus were produced. *Yersinia pestis*, anthrax, and variola reportedly were

prepared for use in intercontinental missiles. By WWII, the United States, the United Kingdom, Canada, Germany, Japan, and the USSR all had active biological weapons programs.

The Iraqi bioterrorist program, initiated in 1974, has been of recent interest. Although much is still unknown about this program, the United Nations Special Commission has information from Iraq that this program studied the use of botulinum toxin, *B. anthracis*, influenza virus, aflatoxin, trichothecene mycotoxins, and ricin. During the Gulf War, Iraq reportedly prepared missiles and bombs that contained aflatoxin, botulinum toxin, and *B. anthracis*, although they were never used.

Disarmament and legislation. In 1969, the United Kingdom and the USSR began to call for bioweapons disarmament. That same year, the U.S. offensive bioterrorist program was dismantled, although the biodefense program continued. In 1971, the U.S. Army Medical Research Institute of Infectious Diseases was opened to research biological protective measures, diagnostic procedures, and therapeutics. By 1973, the United States had destroyed its entire arsenal of bioterrorist agents.

The Convention on the Prohibition of the Development, Production, and Stockpiling of Bacteriological (Biological) and Toxin Weapons and on Their Destruction, also called the Biological Weapons Convention (BWC), was opened for signature in 1972 and became effective in 1975. It was the first multilateral disarmament treaty banning an entire category of weapons. Although the BWC is an international agreement, there is no monitoring mechanism to ensure each party's adherence.

In 1979, a few years after the signing of BWC, there was a massive accidental release of aerosolized *B. anthracis* spores in Sverdlovsk, Russia; 79 people became ill and 69 died. The Soviets maintained that this outbreak was due to the ingestion of contaminated meat sold on the black market. However, President Yeltsin acknowledged in 1992 that in 1979 there had been an accidental release of an unspecified biological agent from a military facility. This is an important event in world history because it was the first major evidence that a nation was in direct violation of the BWC.

In the United States in 1995, a member of a white supremacist group attempted to buy *Y. pestis* from an Ohio laboratory supply company and later attempted to purchase anthrax from a Nevada company. This resulted in the passage of the Antiterrorism and Effective Death Penalty Act of 1996, commonly referred to as the "Select Agent Rule" (42 CFR Part 72.6, Fed Reg Oct. 24, 1996).

In June 2002, the Public Health Security and Bioterrorism Preparedness and Response Act of 2002 was signed into law (PL 107-188). This Act updated the existing Select Agent Rule by requiring facilities to register if they possessed select agents. Previously, only facilities that wanted to transfer select agents needed to register with the Centers for Disease Control and Prevention (CDC). See also http://www.fda.gov/oc/bioterrorism/PL107-188.html.

Epidemiology of a Terrorist Attack

Biological terrorism is the deliberate use of any biological agent against people, animals, or agriculture to cause disease, death, destruction, or panic, for political or social gains. A bioterrorist agent may be a common organism, such as influenza or *Salmonella*, or a more exotic organism such as Ebola virus or variola virus.

In June 1999, a panel of public health, infectious disease, military and civilian intelligence, and law enforcement experts was convened to determine which biological agents (microorganisms and toxins) posed the greatest potential for use in a bioterrorist attack, to be designated as "Category A" agents. These are the following:

- Variola major (smallpox).
- *B. anthracis* (anthrax).
- *Y. pestis* (plague).
- *Francisella tularensis* (tularemia).
- Botulinum toxin (botulism).
- Filoviruses and arena viruses (viral hemorrhagic fevers [VHF]).

Category A agents would have the greatest adverse public health, medical, and social impact if used as a bioterrorist agent for the following reasons:

- They are infectious and stable in aerosol form.
- The world population is highly susceptible to the infections they cause.
- They cause high morbidity and mortality.
- Some can be transmitted from person to person (smallpox, plague, VHF).
- The illnesses they cause can be difficult to diagnose and treat.
- They have been previously developed for biowarfare.

Although bioterrorist attacks ultimately could affect large numbers of people, disease in a single patient may be enough reason to investigate the possibility of biological terrorism. Although some bioterrorist events are subtle, a number of clues should heighten suspicion that a bioterrorist attack has occurred:

- Disease caused by an uncommon organism (e.g., smallpox, anthrax, or VHF).
- A less common presentation of infection with one of these organisms. For example, while a small number of cases of cutaneous anthrax occur naturally each year in the United States, cases of inhalational anthrax are highly unusual.
- A disease identified in a geographic location where it is not usually found (e.g., anthrax in a non-rural area, or plague in the northeastern United States).
- Unexpected seasonal distribution of disease (such as influenza in the summer).
- Antiquated, genetically engineered, or unusual strains of infectious agents.
- Multiple unusual or unexplained diseases in the same patient.
- Disease in an atypical age group or population, such as anthrax in children or varicella-like rashes in adults.
- Large numbers of cases of unexplained disease or death.

- An unexplained increase in the incidence of an endemic disease that previously had a stable incidence rate.
- An unusual condition striking a disparate population, such as respiratory illness in a large population.
- A large number of people seeking medical care at a particular time (signaling they may have been present at a common site, timed with the release of an agent).
- A large number of people presenting with similar illnesses, in noncontiguous regions (may be a sign that there have been simultaneous releases of an agent).
- Animal illness or death that precedes, follows, or occurs simultaneously with human illness or death (may indicate release of an agent that affects both animals and people).

However, because no list of clues can be all inclusive, all health care providers should be alert for the possibility that a patient's condition may not be due to natural causes. When there is no other explanation for an outbreak of illness, it may be reasonable to investigate bioterrorism as a possible source. Common sources of exposure to an agent may include the following:

- Food and water that has been deliberately contaminated.
- Respiratory illness due to proximity to a ventilation source.
- Absence of illness among those in geographic proximity but not directly exposed to the contaminated food, water, or air.

Agents Categorized by System Predominantly Affected

See also Table 4.1.

Respiratory System

Anthrax, plague, and tularemia are all caused by infections with Category A agents and may present as respiratory illnesses.

Anthrax. The incubation period of inhalational anthrax is usually 1–6 days, although it can be longer. The initial symptoms are nonspecific and may resemble those of the common cold (low-grade fever, nonproductive cough, fatigue, malaise, fussiness, poor feeding, sweats, and chest tightness or discomfort), although rhinorrhea is absent. During this phase, chest auscultation usually reveals no abnormalities, although vague rhonchi may be heard. The chest radiograph may reveal pathognomonic mediastinal widening, pleural effusion, and rarely, infiltrates (Figure 4.1). The patient may seem to begin to recover and then become severely ill 1–5 days later. During this phase, sometimes called the "subsequent phase," there is an abrupt onset of high fever and severe respiratory distress, including dyspnea, stridor, diaphoresis, and cyanosis. Despite ventilatory support and antibiotic therapy, shock and death (75% case fatality rate) often occur within 24 to 36 hours. Patients with inhalational anthrax are not contagious, so the only infection control measure necessary is standard precautions.

Plague. Although natural plague can present in a number of forms (septicemic, bubonic, and pneumonic), aerosolization of *Yersinia pestis* causing pneumonic plague would be the most effective mode for a bioterrorist attack. The incubation period is short, about 2 to 4 days and is followed by fever, headache, malaise, cough, dyspnea, and cyanosis. The cough is productive

and may be watery, purulent, or bloody. Chest radiographs often reveal bilateral infiltrates and lobar consolidation. Sometimes, gastrointestinal (GI) symptoms accompany pneumonic plague and include nausea, vomiting, diarrhea, and abdominal pain. The disease is rapidly progressive, often leading to disseminated intravascular coagulation, and 100% of patients die if untreated. Differential diagnoses include community-acquired pneumonias and hantavirus respiratory distress syndrome. The time from exposure to death may be as short as 2 days and is often between 2 and 6 days. Pneumonic plague is spread by respiratory droplet, so droplet precautions should be strictly enforced.

Tularemia. The presentations of tularemia include glandular, oculoglandular, oropharyngeal, septicemic, typhoidal, and pneumonic forms. Similar to plague, the most effective bioterrorist release would be aerosolization, causing the pneumonic form, although the typhoidal form is possible. The incubation period is 1 to 14 days, and the initial illness is often influenza-like, beginning 3 to 5 days later. Clinical findings include sudden onset of fever (38–40°C), headache, malaise, coryza, sore throat, and chills and rigors. A dry, nonproductive cough may progress to bronchiolitis, pneumonitis, pleuritis, pleural effusions, and hilar lymphadenitis and may not be accompanied by objective signs of pneumonia (dyspnea, tachypnea, pleuritic pain, purulent sputum, or hemoptysis). The earliest findings on chest radiograph are peribronchial infiltrates that progress to bronchopneumonia. Only 25 to 50% of patients have radiological evidence of pneumonia in the disease's early stages, and some patients show only minimal, discrete infiltrates. Other cases progress rapidly to respiratory failure and death. Mortality from tularemia pneumonia is 30% if untreated but drops to less than 10% with prompt antibiotic treatment.

Nervous System

Botulism is the category A disease most likely to present with CNS findings. The toxin from the *C. botulinum* bacteria is the most lethal toxin known for people, with an LD_{50} of 1 ng/kg, 100,000 times more toxic than sarin gas. There are four forms of botulism:

- Foodborne.
- Wound.
- Infant (the most common form, accounting for 72% of cases).
- Inhalational (no natural occurrence).

Botulinum toxin can be disseminated through contamination of food or water or via aerosolization and inhalation. When botulinum toxin is ingested, it may cause GI symptoms including abdominal cramping, nausea, vomiting, and diarrhea. Inhalational botulism does not cause a pneumonic process.

Both ingestion and inhalation of the toxin lead to nervous system findings, i.e., an acute, afebrile, symmetrical, descending flaccid paralysis (Figure 4.2). The first signs may appear as quickly as 2 to 72 hours; however, the rate of progression is dose dependent. In natural exposure, the symptoms may be insidious and unapparent for months. In a bioterrorist event, doses may be high, with prompt onset of symptoms. The first manifestation is a cranial nerve palsy, which may present as double or blurred vision, dysphagia, dysarthria, dysphonia, dry mouth, ptosis, gaze paralysis, enlarged or sluggishly reacting pupils, and nystagmus. Sensory changes do not occur.

The paralysis eventually progresses to loss of head control, hypotonia, limb weakness, and respiratory muscle paralysis. Constipation often develops. Patients may appear comatose because of extreme weakness, but sensorium is intact. Deep tendon reflexes may be intact initially but eventually diminish. Without treatment, antitoxin, and ventilatory assistance, patients die of airway obstruction and inadequate ventilation due to respiratory muscle paralysis. Secondary respiratory infections due to aspiration pneumonia may also develop.

Differential diagnoses for botulism include Guillain-Barré syndrome, myasthenia gravis, stroke, other ingestions/intoxications, tick paralysis, viral syndromes, and hypothyroidism. Bioterrorism should be considered when a botulism outbreak occurs within a common geographic area, yet no common source of ingestion can be identified. Botulism is not transmissible from person to person, so standard precautions are sufficient infection control measures.

Gastrointestinal System

A number of infections caused by Category A agents present primarily as syndromes other than GI, although they may be accompanied by some GI complaints. Those that present as respiratory syndromes after aerosol exposure (anthrax, plague, and tularemia) may also present with GI symptoms caused by respiratory distress, especially in children. It is not unusual for children with pneumonia and some degree of respiratory compromise and accessory respiratory muscle use to feed poorly and have nausea, vomiting, mild to moderate abdominal pain, and diarrhea. Botulism, in any of its forms, is primarily a nervous system illness manifested by paralysis. Paralysis may cause some GI manifestations such as poor feeding and constipation.

GI anthrax can occur when food is purposefully contaminated with anthrax spores. The incubation period via this route ranges from a few hours to a week. Depending on where the spores are deposited and germinate, disease may affect the upper or lower GI system, causing acute inflammation and eschar formation, much like in cutaneous anthrax. Upper GI illness may result in an oral or esophageal ulcer, which may present with fever, drooling, dysphagia, regional lymphadenopathy, edema, and sepsis. Lower GI illness often affects the terminal ileum or cecum, and presents with fever, loss of appetite, vomiting, and malaise and progresses to vomiting, hematemesis, severe bloody diarrhea, an acute abdomen, or sepsis. Sometimes, massive ascites develops. This form of anthrax is not transmissible from person-to-person, and standard precautions suffice.

Dermatologic Manifestations

Almost all of the diseases caused by Category A agents (anthrax, smallpox, plague, tularemia, VHF) can cause skin lesions, although dermatologic findings may not be the primary finding in a bioterrorist attack using aerosol dispersion.

Anthrax. Anthrax spores mixed with a fine powder substrate can be used as a weapon to cause respiratory and cutaneous disease. Cutaneous anthrax is the most common form of naturally occurring disease and accounts for 95% of cases. The incubation period is a few hours to 12 days. A small pruritic papule, often mistaken for an insect bite, forms at the inoculation site and rapidly progresses to an ulcer (1–3 cm in diameter) over the course of 1–2 days and may be surrounded by small vesicles (1–3 mm). The organism may be isolated from the serosanguineous

fluid in these vesicles. A painless, depressed eschar of dark necrotic tissue forms at the site, and toxin production causes surrounding edema (Figure 4.3). Adjacent lymph glands may become enlarged and painful. The eschar separates from the skin in 1 to 2 weeks, often leaving no scar. The mortality rate is 20% without treatment, although death is rare with prompt treatment.

Smallpox. Rash is the key feature of smallpox, whether the disease is contracted via mechanical aerosolization or from person-to-person transmission. In ordinary-type smallpox, the most common form, exposure to the virus is followed by an asymptomatic incubation period of 7 to 17 days (mean 12 to 14 days). The prodromal phase, lasting 2 to 4 days, begins with acute onset of high fever, malaise, head and body aches, and sometimes vomiting. The fever usually ranges from 101°F to 104°F, and patients are usually too ill to carry on their normal activities. The patient is not contagious during this period.

The first sign of rash is an enanthema in the mouth that lasts less than 24 hours. These macules break down and shed large amounts of virus into the mouth and throat, making the patient highly contagious. A macular rash then develops on the face and forearms and spreads to the trunk and legs. When the rash begins, patients may defervesce and begin to feel better. Over 1 to 3 days, the lesions progress to papules, which within 1 to 2 days, progress to vesicles and then pustules. The pustules are painful and deep-seated, sometimes described as feeling like lentils or "BB" pellets under the skin. After about 8 to 9 days from onset of the rash, the lesions scab over and eventually separate from the skin. Once the scabs have separated (about 21 days from onset), the patient is no longer contagious, although extensive pitting scars may remain. The rash of smallpox may be confused with rashes of other conditions such as the vesicular pustular rashes (such as varicella), herpes zoster, monkeypox, herpes simplex, drug eruptions, and impetigo. The rash of smallpox may be distinguished from that of chickenpox by physical distribution of the lesions; smallpox lesions tend to concentrate on the face and extremities including the palms and soles, while varicella lesions concentrate on the face and trunk, usually sparing the palms and soles (Figure 4.4). Other distinguishing features of the smallpox rash are a monotonous appearance, with deep-seated lesions in the same stage of development. The pustules may be umbilicated (Figure 4.5). Varicella lesions are superficial, sometimes described as "dew drops on rose petals," and appear in crops, resulting in lesions in different stages of development (Figure 4.6).

There are two clinical forms of smallpox. Variola major is the most severe and most common form of smallpox, with a more extensive rash and higher fever. There are four types of variola major smallpox: ordinary (accounting for 90% or more of cases), modified (mild and occurring in previously vaccinated individuals), flat (malignant), and hemorrhagic. Historically, 30% of patients with variola major smallpox die, usually during the second week of illness. Variola minor smallpox is a less common and less severe form of smallpox, with death rates historically of 1% or less.

Two types of variola major smallpox are both rare and very severe. Malignant and hemorrhagic smallpox progress rapidly and are usually fatal, with death occurring about 5 to 6 days after the rash begins. In malignant smallpox, the rash appears as soft, velvety, confluent vesicles that do not progress to pustules or scabs. In hemorrhagic smallpox, the rash is petechial, with

hemorrhages into the skin and mucous membranes. Patients with all types of smallpox require immediate isolation with precautions for airborne infection.

The CDC has developed an "Acute, Generalized Vesicular or Pustular Rash Illness Protocol" to assist in the evaluation of patients for whom a diagnosis of smallpox is being considered. This algorithm classifies patients as at low, moderate, or high risk of smallpox, which in turn directs clinical laboratory testing. Before submitting laboratory specimens from patients suspected of having smallpox, consult your local and State health departments.

For the algorithm and an accompanying worksheet, see
http://www.bt.cdc.gov/agent/smallpox/diagnosis/evalposter.asp.
For an interactive version of the algorithm, *see*
http://www.bt.cdc.gov/agent/smallpox/diagnosis/riskalgorithm/index.asp.
For laboratory testing algorithms that complement the patient evaluation protocols, *see*
http://www.bt.cdc.gov/agent/smallpox/diagnosis/pdf/rashtestingprotocol.pdf.

Tularemia. Although airborne *Francisella tularensis* would most likely cause pneumonic or typhoidal disease, ulceroglandular and oculoglandular forms may occur that have cutaneous manifestations. There is also a glandular form of the disease, which does not result in skin lesions. The rash of ulceroglandular tularemia begins with a papule at the inoculation site, accompanied by systemic symptoms (fever, chills, rigors, sore throat). The lesion forms a pustule that becomes a tender ulcer and may form an eschar. Regional lymph nodes become inflamed and fluctuant. The oculoglandular form of tularemia leads to conjunctival ulceration, blepharitis, chemosis, vasculitis, and regional lymphadenopathy.

Viral hemorrhagic fevers. Viral hemorrhagic fevers **(**VHFs) are caused by a variety of organisms, with a variety of presentations, making clinical diagnosis difficult. After an incubation period of 2 to 21 days, a rash develops that may range from a subtle cutaneous flushing to a nonpruritic maculopapular rash, similar to that seen in measles. The condition progresses to a bleeding diathesis of petechiae, mucosal and conjunctival hemorrhages, hematuria, hematemesis, and melena.

Notifying Authorities

All public health and medical responses to events of bioterrorism begin at the local level. Pediatricians are front-line health care providers in every community and may become front-line responders in a bioterrorist attack. It is impossible to predict where a child or parent may first seek care for an illness caused by a bioterrorist agent, so primary-care pediatricians, as well as those working at secondary- and tertiary-care facilities, must be prepared to promptly diagnose and isolate a patient who has an illness potentially related to bioterrorism and to notify the proper authorities.

Good infection control practices require that anyone, child or adult, who presents with a fever and rash be immediately placed in a private room with the door closed. This is standard practice because a number of highly contagious childhood infectious diseases present this same way (varicella, measles, rubeola, meningococcus), regardless of whether the illness is ultimately

determined to be due to an agent of bioterrorism. Infection control precautions may also include the use of masks, gowns, gloves, and equipment for eye protection, depending on each situation.

Once the initial history and physical examination have been completed, if a disease related to bioterrorism is suspected, the pediatrician must notify the proper authorities, including the infection-control practitioner (if one is available at the facility) and local public health authorities. Each local public health system is organized slightly differently, so pediatricians should become familiar with their own local public health agency and phone number for local reporting.

Rapid reporting to authorities is essential. Each agency level has developed and continues to refine response plans to handle a bioterrorist event. Rapid activation of these plans provides the best opportunity to limit disease spread during an outbreak. Local authorities may initiate an immediate investigation or seek assistance from the State health department.

For a list of State health department websites, see http://www.cdc.gov/other.htm#states.
For a list of State epidemiologists, see
http://www.cste.org/members/state_and_territorial_epi.asp.

States report their investigations to and request epidemiologic assistance from the CDC. The CDC can provide public health consultation, epidemiologic support, and other technical assistance to State health departments. The CDC usually becomes involved in a State's investigation at the request of the lead State epidemiologist or health officer.

The CDC can be reached during all hours through the CDC Director's Emergency Operation Center at 770-488-7100.

Health Department

Each State has a public health system, although the structure and reporting network varies from State to State. In some States, the State health department has authority over county and local health departments. In other States, county and local health departments function autonomously. State health departments facilitate consultations with specially designated laboratories that are capable of responding to public health emergencies (see the following section, Laboratory Response Network). State bioterrorism response plans will reflect these differences. All pediatricians and health care facilities should learn the structure of their local public health system and the point of contact for reporting illnesses suspected of being related to bioterrorism.

Hospital

Most hospitals have started to develop bioterrorist response plans that may be part of a larger hospital disaster plan. Hospitals play a very large role in the care of bioterrorist victims and anxious or worried parents and others. Optimally, hospitals should have been included in the response planning of local public health agencies. Office and hospital-based pediatricians can become better prepared to respond to a bioterrorist attack by becoming familiar with local hospital bioterrorist and disaster plans. In addition, pediatricians are uniquely qualified to ensure that the special needs of children (e.g., medical supplies and therapeutics specific for children)

are addressed in local medical response plans. (See also Chapter 9. Integrating Terrorism and Disaster Preparedness into Your Pediatric Practice).

Law Enforcement

All suspected cases of bioterrorism are subject to criminal investigation. Public health authorities are responsible for notifying local and Federal law enforcement. In many jurisdictions, these relationships are already established and detailed in State and local public health bioterrorist response plans.

Laboratory Support and Submission of Specimens

Collecting the appropriate clinical laboratory specimens in a case of actual or suspected bioterrorist-related illness is critical for the medical care of the patient, as well as for public health and legal investigations. Specimen collection varies by the agent suspected and should be done in consultation with public health authorities. Local and State public health authorities can advise on specific specimen collection and shipping in each case and consult with the CDC as needed. For detailed information regarding specimen collection, packaging, and shipping, see http://www.bt.cdc.gov/labissues/index.asp.

Laboratory Response Network

The Laboratory Response Network (LRN) is a national network of local, State, and Federal public health, hospital-based, veterinary, agriculture, food, and environmental testing laboratories that provide laboratory diagnostic capability to respond to biological and chemical terrorism and to other public health emergencies. The CDC, along with the Association of Public Health Laboratories and the Federal Bureau of Investigation (FBI), created the LRN, which has been operational since 1999. There are more than 100 LRN laboratories across the United States, and the network continues to expand. Consultation with LRN facilities is facilitated through State health departments. For more information, see http://www.bt.cdc.gov/lrn/.

Limiting Spread

Rapidly detecting and isolating patients with an infectious illness related to bioterrorism is essential to prevent transmission in health care settings. If an infection related to bioterrorism is suspected, the patient should be placed on contact precautions and airborne infection isolation, in addition to standard precautions, until preliminary test results are available and the transmissibility of disease can be reevaluated.

Agents of bioterrorism are generally not transmitted from person to person. The release of an agent is most likely from a point source. However, smallpox, VHFs, and pneumonic plague may be highly transmissible from person to person via respiratory droplet and, in some cases, by aerosol spread.

Standard Precautions

All patients in a health care facility and all patients suspected of infection with a Category A bioterrorist agent (anthrax, botulinum toxin, plague, smallpox, tularemia, and VHFs) should be

cared for using standard precautions. Formerly known as universal precautions, standard precautions are the minimal accepted level of precaution. Standard precautions prevent direct contact with blood, other body fluids, secretions, excretions, nonintact skin/rashes, and mucous membranes and should be observed during all aspects of patient care.

Standard precautions include the following:

- Gloves. Clean, nonsterile gloves should be worn when touching any of the above mentioned substances. Gloves should be removed immediately after contact with these fluids and hands should be washed between care of each patient.
- Handwashing. Hands should be washed after contact with blood or body fluids, whether or not gloves have been worn. Plain or antimicrobial soap with warm water should be used according to facility policy. Alcohol-based hand rubs (≥60%) may also be used when soap and water are not readily available.
- Masks/eye protection or face shields. Whenever procedures are performed that may cause splashes of blood or other body fluids, a mask and eye protection should be worn. These should be removed and discarded or cleaned between care of each patient.
- Gowns. Whenever procedures are performed that may cause splashes of blood or other body fluids, a gown should be worn to protect the skin and clothing. The type of gown selected should be based on the amount of exposure anticipated for each patient care procedure.

Transmission Precautions

Effective infection control sometimes requires additional precautions beyond standard precautions. These are called transmission precautions and consist of the following:

- Contact precautions.
- Droplet precautions.
- Airborne infection isolation.

Transmission precautions are instituted based on the type of organism suspected (Table 4.2).

Contact precautions. Contact precautions are used in addition to standard precautions when patients are suspected or known to be infected with agents transmitted by direct contact with the patient's skin or by indirect contact with surfaces or patient-care items in the patient's environment.

- The patient should be placed in a private room.
- Gloves should be worn when entering the room and at all times while in the room. Hands should be washed immediately after gloves are removed. After handwashing, care should be taken not to touch potentially contaminated surfaces or items.
- Gowns (clean, nonsterile) should be worn when entering the patient's room and removed immediately after leaving the patient's room. After the gown is removed, care should be taken not to touch potentially contaminated surfaces or items.

- The patient should be moved from the room only when necessary. When patients are moved, precautions must be maintained while they are in transport.
- Equipment used for patient care should be dedicated for a single patient to avoid transmitting infection to other patients. If equipment must be shared, it must be cleaned and disinfected between patients.

Droplet precautions. Droplet precautions are used in addition to standard precautions when patients are suspected or known to be infected with microorganisms transmitted by droplets >5µm in size that are generated when a patient coughs, sneezes, or talks. These particles, when expelled, may travel 3 feet before landing on a surface.

- The patient should be placed in a private room or with patients who have only the same known illness.
- Masks should be worn when entering the room or when working within 3 feet of the patient, according to facility policy.
- The patient should be moved from the room only when necessary. When patients are moved, they should wear a mask to prevent dispersal of droplet particles.

Airborne infection isolation. Formerly known as airborne precautions, airborne infection isolation is the highest level of precaution. It is used in addition to standard precautions when caring for patients suspected or known to be infected with microorganisms transmitted by nuclei of airborne droplets <5µm in size. When dispersed, these nuclei remain suspended in the air and can be dispersed over long distances and potentially throughout a health care facility.

- The patient should be placed in a private, negative-air pressure room, with 6 to 12 air exchanges per hour. The room should be monitored for negative pressure and should have appropriate discharge of air to the outdoors or have high-efficiency filtration before the air is recirculated to other parts of the facility. The door should be closed at all times.
- Only essential personnel should enter the room and should wear a PAPR or a fitted N95 (or higher) respirator at all times.
- Patients should be moved from the room only when necessary. When patients are moved, they should wear a mask to limit potential spread of particles.

For additional information about infection control measures, see "Guideline for Isolation Precautions in Hospitals" at http://www.cdc.gov/ncidod/dhqp/gl_isolation.html and "Guideline for Environmental Infection Control in Health-Care Facilities, 2003: Recommendations of CDC and the Healthcare Infection Control Practices Advisory Committee" at http://www.cdc.gov/ncidod/dhqp/gl_environinfection.html.

Equipment and Supplies

The equipment and supplies necessary to diagnose and treat a patient suspected of being infected with a bioterrorist agent vary by the level of care that will be provided at a particular facility. An office-based primary care pediatrician may need to be concerned only with short-term isolation and preliminary stabilization of a patient, which will require a relatively short list of supplies that usually are available in the well-stocked pediatric medical office. Hospital-based pediatricians

may be providing longer term and more complex care to patients and should consult their hospital administration regarding the hospital's bioterrorist response plan and the response plans of State and local health authorities.

Response planning requires a detailed and integrated approach between public health and medical facility administrators. For guidance and helpful checklists, see "The Public Health Response to Biological and Chemical Terrorism: Interim Planning Guidance for State Public Health Officials, July 2001," at http://www.bt.cdc.gov/Documents/Planning/PlanningGuidance.PDF, and "Bioterrorism Readiness Plan: A Template for Healthcare Facilities, April 1999," at http://www.cdc.gov/ncidod/dhqp/pdf/bt/13apr99APIC-CDCBioterrorism.PDF.

Pediatric Practices

During a bioterrorist event, local pediatricians and their staffs should maximize their ability to keep the office running smoothly and to provide care. The first step is for every staff member to have a personal family emergency plan. Once staff members are assured that they and their family members are safe, they are better able to focus on their professional duties.

Second, every office needs an emergency plan. This plan should include details for handling an emergency both in-office and in the community. Items that should be included in an in-office emergency plan include the following:

- Isolation of the patient and family.
- PPE for staff.
- Contact information for local public health authorities.
- Phone numbers for emergency patient transport.

Items that should be included in a plan for an emergency in the community include the following:

- Information sheets and telephone hotline numbers.
- Telephone triage protocols.
- Back-up staffing schedules.

Depending on the situation, dedicated staff may be needed just to handle anxious or worried parents. (See also Chapter 9, Integrating Terrorism and Disaster Preparedness into Your Pediatric Practice, for further information on office emergency plans.)

The community-based pediatrician should have the following items readily available to evaluate children suspected of having an illness related to bioterrorism:

- An examining room with a door that closes, in which to isolate a patient and accompanying family members.
- Surgical masks.
- Clean, nonsterile gowns.

- Clean, nonsterile disposable gloves.
- Eye protection equipment (i.e., goggles, face shields).
- The round-the-clock phone numbers of local and State public health authorities.

Other laboratory supplies that might be required in the early evaluation of a patient should be available through public health authorities. Public health authorities will advise on the collection of specimens and on patient transport to locally designated emergency response facilities.

Health care professionals can register to receive e-mail updates about bioterrorism and response planning at http://www.bt.cdc.gov/clinregistry/index.asp.

Managing Patients: Treatment and Prevention

Treatment consists of supportive care (e.g., fever management, fluid management, nutritional supplementation, ventilatory support, and emotional care) and medical treatment (antibiotics and antitoxins) specific to the bioterrorist organism implicated.

The Department of Health and Human Services has stockpiled vaccine for potential use in an outbreak of smallpox and anthrax. Although these vaccines are not available to the public before a bioterrorist event, they could be made rapidly available to high-risk populations in the event of an attack.

Smallpox Vaccine

Smallpox vaccine contains live vaccinia virus, an orthopox virus that is related to smallpox virus but that does not cause smallpox. Currently, the United States has stockpiled two types of vaccine: 1) vaccine manufactured from calf lymph and produced in the 1960s (Wyeth and Aventis), and 2) a newly manufactured cell culture-derived vaccine (Acambis),which has completed phase III trials and is awaiting FDA decisions for next steps.

The smallpox vaccine is administered using a bifurcated needle that results in multiple punctures to the superficial layers of the skin. For a video about smallpox vaccination administration, see http://www.bt.cdc.gov/agent/smallpox/vaccination/administration-video/index.asp.

The vaccination site heals over the course of 3 weeks and leaves a small scar. In the 1960s through 1972 when smallpox vaccination was part of the routine childhood immunization schedule, children—especially girls—were often vaccinated in locations on the body where the scar was not visible. Because smallpox vaccine will be released only in response to a bioterrorist attack, it is recommended that the vaccine be administered only in the deltoid region of the arm, so that the vaccination site and scar can be seen easily and evaluated for both medical and public health purposes (Figure 4.7).

If given before exposure, the smallpox vaccine is highly effective against smallpox (95–97%), and if given up to 4 days after exposure, it also can prevent or minimize the severity of disease. The lower age limit for vaccine administration for a child who has not been exposed is 1 year. There is no lower age limit for a child who has been exposed to smallpox.

The vaccine has a number of common side effects, as well as the risk of more serious adverse reactions. Although many children experience side effects commonly seen after other childhood vaccinations (e.g., fever, lymphadenopathy, fatigue, malaise, and fussiness), far fewer children experience vaccinia-specific adverse reactions such as generalized vaccinia, eczema vaccinatum, vaccinia necrosum, postvaccinal encephalitis, and fetal vaccinia. Fortunately, by screening for contraindications, these complications may be largely avoidable.

For extensive information about smallpox vaccination, administration, screening for contraindications, adverse events, and treatment of adverse events, see http://www.bt.cdc.gov/agent/smallpox/vaccination/index.asp.

Anthrax Vaccine

Anthrax vaccine protects against invasive disease and is currently recommended only for high-risk populations:

- Laboratory personnel working with anthrax.
- People working with imported animal hides and furs in areas where standards are insufficient to prevent exposure to anthrax spores.
- People who handle potentially infected animal products in high-incidence areas.
- Military personnel deployed to areas with high risk for exposure to anthrax.

The licensed AVA anthrax vaccine, manufactured by BioPort Corporation, Lansing, MI, is prepared from cell-free filtrate of *Bacillus anthracis* culture that contains no live or dead bacteria. The vaccine is administered in a series of six subcutaneous injections, given at 0, 2 weeks, 4 weeks, 6 months, 12 months, and 18 months, followed by annual boosters. Mild local reactions are common in adults, occurring in approximately 30% of vaccinates. Systemic reactions are uncommon, occurring in <0.2% of vaccinates.

The anthrax vaccine has not been tested in children and currently is not recommended for children younger than age18. However, the CDC is evaluating the safety and effectiveness of using anthrax vaccine after exposure in children. Prophylaxis for anthrax in children is currently based on antimicrobial treatment (e.g., ciprofloxacin, doxycycline, and amoxicillin).

Strategic National Stockpile

The Strategic National Stockpile (SNS) was established in 2003 and is jointly managed by the Department of Homeland Security (DHS) and the Department of Health and Human Services (DHHS). The SNS is a national repository of antibiotics, chemical antidotes, antitoxins, vaccines, life-support medications, and other medical and surgical items. SNS maintains a stock of supplies that are specific for the medical needs of children and has received guidance from the AAP, as well as from academic and public health experts in general pediatrics, pediatric infectious diseases, pediatric pharmacology, and pediatric critical care medicine.

The SNS is designed to supplement and re-supply State and local public health agencies in the event of a national emergency anywhere and at any time in the United States or its territories. The SNS is prepared for immediate response by having push packs strategically positioned

across the United States. Push packs provide medical supplies for an initial response to a broad range of emergencies and arrive on site within 12 hours of deployment. If additional supplies are required, they can be shipped within 24 to 36 hours through vendor-managed inventory. (See also Chapter 2, Systems Issues, Centers for Disease Control and Prevention and Federal Response Plan.)

To receive SNS assets, the governor of the affected State should directly request deployment from the CDC or DHHS. Most likely there will already have been public health consultation between the State department of health and the CDC. Requests for consultation with the CDC or SNS deployment can be made through the CDC Director's Emergency Operation Center at 770-488-7100. For further details on the SNS, see http://www.bt.cdc.gov/stockpile/.

Surge Capacity

Medications

Each State's emergency response plan, which is coordinated with public health authorities, should involve some local stockpiling of critical antibiotics and emergency medical supplies. If needed, additional supplies are available through the SNS. Each State plan details the logistics for transporting shipments within the State to where they are needed. In addition, many local and State health departments have partnered with local health facilities to hold practice runs of large-scale drug dispensing and vaccination clinics.

Isolation

Isolation needs will vary greatly depending on the type of attack. For those diseases that are not transmitted person to person (anthrax, tularemia, and botulism), isolation is not needed. The people exposed will be those at the geographic location where the organism or toxin was released.

For diseases that are transmissible, such as smallpox, plague, and VHF, infection control measures include isolation. Depending on the number of cases, victims may be isolated within a hospital. If demand exceeds the capabilities of a traditional health care facility, supplemental isolation and medical care facilities may be needed (e.g., schools, college campuses, motels, churches, or unused hospitals). If patients do not require advanced medical care, home isolation may be sufficient. Home isolation was used successfully during the SARS and monkeypox outbreaks of 2003.

As part of their bioterrorism response planning activities, public health agencies are identifying facilities that can be used to isolate patients during an outbreak. Related planning includes having an inventory of the isolation and negative-pressure rooms in a particular area, establishing back-up isolation facilities, establishing and training public health and medical response teams, and reviewing and updating State quarantine laws. For public health bioterrorism planning guidance documents, see http://www.bt.cdc.gov/.

Vaccination

Large-scale vaccination may be recommended in some outbreaks related to bioterrorism, namely smallpox. Vaccination may be offered to an affected community, county, State, or the entire Nation. Large-scale smallpox vaccination clinics are intended to supplement the concurrent "surveillance and containment" strategy, (also called "search and containment" and "ring vaccination"). Surveillance and containment requires that individuals who are ill with smallpox are quickly identified and isolated, followed by rapid identification and vaccination of their contacts within 4 days of exposure.

For additional information about vaccination strategies to be used during a smallpox outbreak, see http://www.bt.cdc.gov/agent/smallpox/response-plan/index.asp.

Large-scale vaccination clinics may offer vaccination to anyone who does not have medical contraindications to receiving the vaccine under emergency circumstances. The Advisory Committee on Immunization Practices (ACIP) is reviewing and refining the contraindications to smallpox vaccination after an event for people who have not been exposed.

Information for Families

During a bioterrorist attack, one of the most important and challenging roles for the local pediatrician is providing information to families with children. During the anthrax attacks of 2001, public health and medical facilities were inundated with requests for information and medical evaluation. As a result, these same agencies have prepared communication messages and information sheets that can be shared with families before and during a crisis. Parents will want information that is age-appropriate for their children, as well as suggestions for ways to answer their children's questions. Pediatricians may want to consider accessing some of these materials and having them available before an emergency occurs.

A number of organizations have developed materials to help educate children and their families about emergencies and bioterrorism. More information can be obtained from the following organizations and their Web sites:

- American Academy of Pediatrics
  http://www.aap.org/terrorism/resources/federal_resources.html
- The Department of Health and Human Services
  http://www.os.dhhs.gov/emergency/index.shtml
- The National Child Traumatic Stress Network
  http://www.nctsnet.org/nccts/nav.do?pid=ctr_aud_prnt

Category A Agents

See also Table 4.3.

Anthrax

Bacillus anthracis, the etiologic agent of anthrax, is a gram-positive, anaerobic, spore-forming, bacterial rod. The three virulence factors of *B. anthracis* are edema toxin, lethal toxin and a capsular antigen. Human anthrax has three major clinical forms:

- Cutaneous.
- Inhalational.
- Gastrointestinal.

If untreated, anthrax in all forms can lead to septicemia and death. Anthrax generally is not contagious, but person-to-person transmission from cutaneous lesions has been reported rarely. For more information, see http://www.bt.cdc.gov/agent/anthrax/index.asp.

Signs and symptoms. Symptoms usually occur within 2 weeks of exposure; however, the incubation period for inhalational anthrax may be as long as several months because of spore dormancy and delayed clearance from the lungs.

Cutaneous anthrax. Cutaneous anthrax is the most common type of infection (>95%). It usually develops after skin contact with contaminated meat, wool, hides, or leather from infected animals. The incubation period ranges from 1 to 12 days. The skin infection begins as a small papule and progresses to a vesicle in 1 to 2 days, followed by a painless, necrotic ulcer with a black eschar, usually 1–3 cm in diameter (Figure 4.3). Patients may have fever, malaise, headache, and regional lymphadenopathy.

Inhalational anthrax. Inhalational disease is the most lethal form of anthrax. The incubation time of inhalational anthrax in people is unclear, but it is reported to range from 1 to 7 days, possibly up to 60 days. Initial symptoms resemble common respiratory infections and include mild fever, muscle aches, and malaise. Some patients also complain of sore throat. These symptoms progress to nonproductive cough, pleuritic chest pain, shortness of breath, respiratory failure, and frequently, meningitis. Upper respiratory symptoms such as rhinorrhea are generally not seen with inhalational anthrax.

Gastrointestinal anthrax. Gastrointestinal disease is the least common form of anthrax. It usually follows the consumption of raw or undercooked contaminated meat and has an incubation period of 1 to 7 days. Severe abdominal distress is followed by fever and signs of septicemia. The disease can take an oropharyngeal or abdominal form. Lesions at the base of the tongue, sore throat, dysphagia, fever, and regional lymphadenopathy usually characterize involvement of the oropharynx. Lower bowel inflammation usually causes nausea, loss of appetite, vomiting, and fever, followed by abdominal pain, hematemesis, and bloody diarrhea.

Diagnosis. The clinical evaluation of patients suspected of having inhalational anthrax should include a chest radiograph and/or CT scan to evaluate for widened mediastinum and pleural effusion. (Figure 4.1). For chest radiographs, see http://phil.cdc.gov/PHIL_Images/02122002/00041/PHIL_1795_thumb.jpg and http://phil.cdc.gov/PHIL_Images/02122002/00042/PHIL_1796_thumb.jpg.

Anthrax is not spread by person-to-person contact except in rare cases of transmission from cutaneous lesions. If the history does not reveal possible environmental exposure, anthrax is not a likely diagnosis. Depending on the clinical presentation, Gram stain and culture should be performed on specimens of blood, pleural fluid, CSF, and tissue biopsy or discharge from

cutaneous lesions; however, previous treatment with antimicrobial agents can result in false negatives. Isolates can be definitely identified through the LRN in each State. Additional diagnostic tests, including immunohistochemistry, real-time PCR, time-resolved fluorescence, and an enzyme immunoassay that measures IgG antibodies against *B. anthracis* protective antigen, are performed at the CDC and can be accessed through State health departments.

Nasal swabs for detection of *B. anthracis* may assist in epidemiologic investigations but should not be relied on as a guide for prophylaxis or treatment of individual patients. Epidemiologic investigation in response to threats of exposure to *B. anthracis* may use nasal swabs of potentially exposed individuals as an adjunct to environmental sampling to determine the extent of exposure. See Table 4.4a and Table 4.4b.

Treatment. A high index of clinical suspicion and rapid administration of effective antimicrobial therapy are essential for prompt diagnosis and effective treatment. No controlled trials have been performed in people to validate current treatment recommendations, and clinical experience is limited. For bioterrorism-associated cutaneous disease in adults or children, ciprofloxacin (500 mg, PO, BID, or 10–15 mg/kg/day for children, PO, divided BID) or doxycycline (100 mg, PO, BID, or 5 mg/kg/day, PO, divided BID for children younger than 8 years of age) are recommended for initial treatment until antimicrobial susceptibility data are available. Because of the risk of concomitant inhalational exposure, consideration should be given to continuing an appropriate antimicrobial regimen for postexposure prophylaxis.

Ciprofloxacin (400 mg, IV, every 8–12 hours) or doxycycline (200 mg, IV, every 8–12 hours) should be used initially as part of a multidrug regimen for treating inhalational anthrax, anthrax meningitis, cutaneous anthrax with systemic signs, and GI anthrax until results of antimicrobial susceptibility testing are known. Other agents with *in vitro* activity suggested for use in conjunction with ciprofloxacin or doxycycline include rifampin, vancomycin hydrochloride, imipenem, chloramphenicol, penicillin, ampicillin, clindamycin, and clarithromycin. Cephalosporins and trimethoprim-sulfamethoxazole should not be used. Treatment should continue for at least 60 days. Neither ciprofloxacin nor tetracycline is routinely used in children or pregnant women because of safety concerns. However, ciprofloxacin or tetracycline should be used for treatment of anthrax in children who have life-threatening infections until antimicrobial susceptibility patterns are known.

About 20% of untreated cases of cutaneous anthrax result in death, but deaths are rare if patients receive appropriate antimicrobial therapy. The case fatality rate of inhalational anthrax is estimated to be 50% to 75%, even with early treatment. The case fatality rate of GI anthrax is estimated to be between 25% and 60%. The impact of antibiotic treatment on the case fatality rate of GI anthrax is unknown.

Control measures. Standard precautions are recommended for hospitalized patients. Contaminated dressings and bed linens should be incinerated or steam sterilized to destroy spores. Autopsies performed on patients with systemic anthrax require special precautions.

BioThrax (formerly known as Anthrax Vaccine Adsorbed [manufactured by BioPort Corp, Lansing, MI]) is the only vaccine licensed in the United States for prevention of anthrax in

people. This vaccine is prepared from a cell-free culture filtrate. Immunization consists of six SC injections at 0, 2, and 4 weeks and at 6, 12, and 18 months, followed by annual boosters. The vaccine is currently recommended for people at risk of repeated exposures to *B. anthracis* spores, including select laboratory workers and military personnel. The vaccine is effective for preventing cutaneous anthrax in adults. Protection against inhalational anthrax has not been evaluated in people, but the vaccine has been effective in studies in nonhuman primates. Adverse events are mainly local injection site reactions; systemic symptoms, including fever, chills, muscle aches, and hypersensitivity are rare. No data on vaccine effectiveness or safety in children are available, and the vaccine is not licensed for use in children or pregnant women. Anthrax vaccine is not licensed for postexposure use in preventing anthrax.

Based on the limited available data, the best means of preventing inhalational anthrax after exposure to *B. anthracis* spores is prolonged antimicrobial therapy in conjunction with a three-dose regimen (at 0, 2, and 4 weeks) of anthrax immunization. However, because BioThrax is not licensed for postexposure prophylaxis or for use as a three-dose regimen or for use in children, it can be used only under an investigational new drug application as part of an emergency public health intervention. When no information is available about the antimicrobial susceptibility of the implicated strain of *B. anthracis*, initial postexposure prophylaxis for adults or children with ciprofloxacin or doxycycline is recommended. Although fluoroquinolones and tetracyclines are not recommended as first-choice drugs in children because of adverse effects, these concerns may be outweighed by the need for early treatment of pregnant women and children exposed to *B. anthracis* after a terrorist attack. As soon as susceptibility of the organism to penicillin has been confirmed, prophylactic therapy for children should be changed to oral amoxicillin, 80 mg/kg/day, divided TID (not to exceed 500 mg, TID). *Bacillus anthracis* is not susceptible to cephalosporins and trimethoprim-sulfamethoxazole; therefore, these agents should not be used for prophylaxis (see Table 4.5).
For more information, see http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5045a5.htm.

Reporting. If a case of anthrax is suspected, immediately contact the local and State health departments and hospital infection control practitioner. If they are unavailable, contact the CDC at 770-488-7100.

Botulinum Toxin

For a quick fact sheet, see http://www.bioterrorism.slu.edu/botulism/quick/botulism01.pdf.
For additional information, see http://jama.ama-assn.org/cgi/content/full/285/8/1059?.

Disease. Botulism is a rare disease caused by ingestion of the anaerobic, spore-forming bacillus *Clostridium botulinum*. Botulism neurotoxins are the most potent toxins known. There are three forms of naturally occurring botulism:

- Foodborne.
- Wound.
- Infant (intestinal).

In addition, inhalational disease could occur if aerosolized botulinum toxin were used, such as in a bioterrorist incident. A thorough history may help determine the mode of infection. If no

common food source is identified during an outbreak or cluster of cases, bioterrorism should be suspected.

Signs and symptoms. The incubation period varies according to the type of botulism and the extent of exposure to the toxin:

- Foodborne: 12-36 hours (range 6 hours to 10 days).
- Wound: 7-8 days (range 4–18 days) after injury.
- Infant: 18-36 hours after ingestion.
- Inhalational: The true incubation period for aerosolized botulism is unknown. In the three known inhalational cases, onset was approximately 72 hours. In laboratory studies, monkeys developed disease 12–18 hours after exposure.

Regardless of the means of exposure, botulinum toxin causes permanent nerve damage by irreversibly binding to nerve synapses and interfering with the release of acetylcholine. Botulinum toxin cannot cross the blood-brain barrier and does not affect the CNS. Sensory systems remain intact while the peripheral cholinergic synapses are damaged, resulting in flaccid paralysis in a patient who remains mentally alert and afebrile.

The toxin first affects the muscles connected to the cranial nerves. Early symptoms of all forms of the disease include double or blurred vision, difficulty with speaking and swallowing, dry mouth, and fatigue. As the disease progresses, symmetrical muscle weakness develops, starting at the trunk and descending to the extremities; deep tendon reflexes generally remain intact.

Without ventilatory support, death results when the toxin attacks the respiratory system, resulting in airway obstruction and respiratory paralysis. Recovery may occur if paralyzed muscles are reinnervated, but this process requires weeks to months of intensive supportive therapy.

Foodborne. Infants may develop disease after ingestion of *C. botulinum* organisms and subsequent GI absorption of toxin. Among babies older than 6–12 months, disease results only from ingestion or inhalation of preformed toxin. Initial symptoms include vomiting, constipation, GI upset, and rarely diarrhea, followed by symptoms listed above. These GI symptoms are thought to be caused by other bacterial metabolites also present in the food and may not occur if purified botulinum toxin is intentionally placed in foods or aerosols. Respiratory support is required in 57% to 81% of patients.

Wound. This form of the disease most closely resembles tetanus. Neurotoxins produced by the contaminating organisms in the affected wound disseminate throughout the body and destroy the nerve endings. Symptoms are similar to those of foodborne illness, except that there are no GI symptoms.

Infants. The initial symptom is generally constipation, although lethargy, lack of appetite, drooling, and weakness also occur. Descending symmetrical paralysis follows, evidenced by bulbar palsies: poor head and muscle control; flat affect; ptosis; impaired gag, suck, and swallow reflexes; dilated or sluggish pupillary reaction; and a weak cry. Respiratory failure is common. Intubation is required in >80% of cases, and ventilatory support is necessary (see Figure 4.2).

Diagnosis. A presumptive diagnosis can be made based on signs and symptoms. Laboratory confirmation is needed for definitive diagnosis. Obtaining a history that focuses on food intake and potential exposure to the organism is imperative.

Signs and symptoms of botulism that help distinguish it from other causes of weakness include the following:

- Disproportionate involvement of cranial nerves.
- Involvement of facial muscles to a greater extent than more distal weakness.
- The lack of sensory changes that usually accompany other disorders that result in flaccid paralysis.

Confirmatory tests include detection of toxin through mouse bioassay using the following specimen(s): blood and feces (foodborne), blood and wound (wound), and feces (intestinal). Toxin can also be detected in gastric secretions, which might be the most useful specimen in a case of inhalational disease. For results of the bioassay to be accurate, all specimens should be refrigerated during storage, serum samples should be obtained before antitoxin treatment, and the laboratory should be notified if the patient has taken anticholinesterase medications. Definitive diagnosis may be made through monovalent and polyvalent diagnostic antitoxins available from the CDC and a limited number of public health departments.

Treatment. Rapid diagnosis and initiation of treatment and supportive care provide the best opportunity for survival. Treatment should begin as soon as the diagnosis is suspected without waiting for laboratory confirmation. Antitoxin, available from the CDC by calling 770-488-7100, should be administered to all patients with known or suspected disease. Antitoxin cannot reverse the effects of toxin bound to nerve receptors, but it does prevent further progression of nerve damage. Because the antitoxin is derived from horse serum, serious complications (including anaphylaxis and serum sickness) can develop. Supportive care generally includes intensive care, tube feedings or total parenteral nutrition (TPN), and ventilator support (in 29% of foodborne cases and 80% of infant cases).

Recommendations for safe and effective administration of antitoxin have changed over time; package insert materials should be reviewed before initiation.

Foodborne and inhalational botulism. Trivalent equine botulinum antitoxin (types A, B, and E) and bivalent antitoxin (types A and B) are available from the CDC at 770-488-7100 or through State health departments for treatment of foodborne or wound botulism. Patients should be tested for hypersensitivity to equine sera before administration. Approximately 9% of treated people experience some degree of hypersensitivity to equine serum, but severe reactions are rare.

Infant botulism. A 5-year, randomized, double-blind, placebo-controlled treatment trial of human-derived botulinum antitoxin (formally known as botulism immune globulin intravenous [BIGIV]) in infants with botulism showed a significant decrease in hospital days, mechanical ventilation, tube feedings, and cost associated with BIGIV administration ($70,000 less per case). The California Department of Health Services (24-hour telephone number, 510-540-2646) should be contacted to procure BIGIV. Treatment with BIGIV should begin as early in the illness as possible. BIGIV is available only for treatment of infant botulism. Approximately 9% of

treated people experience some degree of hypersensitivity reaction to equine serum, but severe reactions are rare.

Antimicrobial agents should be avoided in infant botulism because lysis of intraluminal *C. botulinum* could increase the amount of toxin available for absorption. Aminoglycosides can potentiate the paralytic effects of the toxin and should be avoided.

Control. Standard precautions should be used in the care of hospitalized patients with botulism. Person-to-person transmission does not occur.

After exposure. Individuals known to be exposed or suspected of having been exposed to aerosolized botulinum toxin should be closely monitored and treated with antitoxin at the first sign of disease. Prophylactic equine antitoxin for asymptomatic people who have ingested a food known to contain botulinum toxin is not recommended. Because of the danger of hypersensitivity reactions, the decision to administer antitoxin requires careful consideration. Consultation about antitoxin use may be obtained from the State health department or the CDC.

Elimination of recently ingested toxin may be facilitated by induction of vomiting, gastric lavage, rapid purgation, and high enemas. These measures should not be used in infant botulism. Enemas should not be administered to people with illness except to obtain a fecal specimen for diagnostic purposes. Exposed people should be observed closely.

Decontamination. *Clostridium botulinum* is a hardy spore that is highly heat resistant, but botulism toxin in food is easily destroyed through the normal cooking process (heating >85º C for 5 minutes). Weather conditions and size of the aerosolized particles determine how long the toxin can remain airborne, but it is estimated that most toxin would be inactive within 2 days of aerosol release. If a warning is issued before a release, some protection can be achieved by covering the mouth with cloth or a mask; toxin may be absorbed through mucous membranes but cannot penetrate intact skin. After a known exposure, patients and their clothing should be washed with soap and water. Surfaces exposed to the initial release should be cleaned with a 1:10 hypochlorite (bleach) solution.

Reporting. If you suspect a case of botulism, immediately contact your hospital epidemiologist or infection control practitioner and local and State health departments. If local and State health departments are unavailable, contact the CDC at 770-488-7100.

For further information and fact sheets, see http://www.bt.cdc.gov/agent/botulism/index.asp.

Plague

Plague is caused by *Yersinia pestis,* a pleomorphic, bipolar-staining, gram-negative coccobacillus. In nature, plague is a zoonotic infection of rodents, carnivores, and their fleas that are found in many areas of the world. Plague has been reported throughout the Western United States, but most human cases occur in New Mexico, Arizona, California, and Colorado as isolated cases or in small clusters.

- Bubonic plague usually is transmitted by bites of infected rodent fleas and uncommonly by direct contact with tissues and fluids of infected rodents or other mammals, including domestic cats.
- Septicemic plague occurs most often as a complication of bubonic plague but may result from direct contact with infectious materials or the bite of an infected flea.
- Primary pneumonic plague is acquired by inhalation of respiratory droplets from a human or animal with respiratory plague or from exposure to laboratory aerosols.
- Secondary pneumonic plague arises from hematogenous seeding of the lungs with *Y. pestis* in patients with bubonic or septicemic plague**.**

The incubation period is 2–6 days for bubonic plague and 2–4 days for primary pneumonic plague.

Signs and symptoms. A bioterrorist incident involving plague would most likely occur through aerosolization and result in pneumonic involvement. The incubation period after flea-borne transmission is 2–8 days. Incubation after aerosolization would be expected to be shorter (1–3 days). Clinical features of pneumonic plague include fever, cough with mucopurulent sputum (gram-negative rods may be seen on Gram stain), hemoptysis, and chest pain. A chest radiograph will show evidence of bronchopneumonia.

Diagnosis. Plague is characterized by massive growth of *Y. pestis* in affected tissues, especially lymph nodes, spleen, and liver. The organism has a bipolar (safety-pin) appearance when viewed with Wayson or Gram stains. If plague organisms are suspected, the laboratory examining the specimens should be informed to minimize risks of transmission to laboratory personnel. Handling of specimens should be coordinated with local or State health departments and undertaken in Biosafety Level 2 or Level 3 laboratories.

A positive fluorescent antibody test result for the presence of *Y. pestis* in direct smears or cultures of a bubo aspirate, sputum, CSF, or blood specimen provides presumptive evidence of *Y. pestis* infection. A single seropositive result by passive hemagglutination assay or enzyme immunoassay in an unimmunized patient who has not had plague previously also provides presumptive evidence of infection. Seroconversion and/or a four-fold difference in antibody titer between two serum specimens obtained 1 to 3 months apart provides serologic confirmation. The diagnosis of plague usually is confirmed by culture of *Y. pestis* from blood, bubo aspirate, or another clinical specimen. PCR assay or immunohistochemical staining for rapid diagnosis of *Y pestis* is available in some reference or public health laboratories. Isolates suspected as *Y. pestis* should be reported immediately to the State health department and submitted to the Division of Vector-Borne Infectious Diseases of the CDC. For additional information, see http://www.cdc.gov/ncidod/dvbid/plague/diagnosis.htm
http://www.bt.cdc.gov/Agent/Plague/ype_la_cp_121301.pdf.

Treatment. Streptomycin sulfate (30 mg/kg/day, IM, divided BID-TID) is the treatment of choice for most children. Gentamicin sulfate in standard dosages for age given IM or IV is an equally effective alternative to streptomycin. Tetracycline, doxycycline, or chloramphenicol is also effective. Tetracycline or doxycycline should not be given to children younger than age 8 unless the benefits of use outweigh the risks of dental staining. Chloramphenicol is the preferred

treatment for plague meningitis. Antimicrobial treatment should be continued for 7–10 days or until several days after fever breaks. Drainage of abscessed buboes may be necessary; drainage material is infectious until effective antimicrobial therapy has been given.

Control measures. In addition to standard precautions, droplet precautions are indicated for all patients with suspected plague until pneumonia is excluded and appropriate therapy has been started. Special air handling is not indicated. In patients with pneumonic plague, droplet precautions should be continued for 48 hours after appropriate treatment has been started.

Postexposure prophylaxis should begin after confirmed or suspected exposure to *Y. pestis* and for postexposure management of health care workers and others who have had unprotected face-to-face contact with symptomatic patients. In children, prophylactic treatment with doxycycline (5 mg/kg/day, divided BID) or ciprofloxacin (20–30 mg/kg/day divided BID) is recommended and should be continued for 7 days after exposure or until exposure can be excluded. Household members and other people with intimate exposure to a patient with plague should report any fever or other illness to their physician.

Currently, no vaccine for plague is commercially available in the United States. Information concerning the availability of plague vaccines is available from the Division of Vector-Borne Infectious Diseases of the CDC.

Reporting. State public health authorities should be notified immediately of any suspected cases of plague in people. Initial suspicion of a bioterrorist event involving *Y. pestis* will likely involve identification of more than one case in a nonendemic area. If this occurs, immediately contact your local and State health departments and hospital infection control practitioner. If they are unavailable, contact the CDC at 770-488-7100.

For additional information, see http://jama.ama-assn.org/cgi/content/short/283/17/2281.

Smallpox

Variola, the virus that causes smallpox, is a member of the Poxviridae family (genus Orthopoxvirus). These DNA viruses are among the largest and most complex viruses known, and they differ from most other DNA viruses by multiplying in the cytoplasm. Monkeypox, vaccinia, and cowpox are other members of the genus and can cause zoonotic infection of people, but they usually do not spread from person to person. People are the only natural reservoir for variola virus. For additional information, see http://www.bt.cdc.gov/agent/smallpox/index.asp.

In 1980, the World Health Organization (WHO) declared that smallpox (variola) had been successfully eradicated worldwide. The last naturally occurring case of smallpox occurred in Somalia in 1977, followed by two cases attributable to laboratory exposure in 1978. The United States discontinued routine childhood immunization against smallpox in 1971 and routine immunization of health care workers in 1976. The U.S. military continued to immunize military personnel until 1990. Since 1980, the vaccine has been recommended only for people working with nonvariola orthopoxviruses. Two WHO reference laboratories were authorized to maintain stocks of variola virus. There is increasing concern that the virus and the expertise to use it as a weapon of bioterrorism may have been misappropriated.

Signs and symptoms. An individual infected with variola major develops a severe prodromal illness characterized by high fever (102°–104°F [38.9°–40.0°C]) and constitutional symptoms, including malaise, severe headache, backache, abdominal pain, and prostration, lasting 2–5 days. Infected children may have vomiting and seizures during this prodromal period. Most patients with smallpox tend to be severely ill and bedridden during the febrile prodrome. The prodromal period is followed by enanthemas that may not be noticed by the patient. This stage occurs <24 hr before the onset of rash, which is usually the first recognized manifestation of infectiousness. With the onset of enanthemas, the patient becomes infectious and remains so until all skin crust lesions have separated. The rash, or exanthem, typically begins on the face and rapidly progresses to involve the forearms, trunk, and legs in a centrifugal distribution (greatest concentration of lesions on the face and distal extremities). Many patients have lesions on the palms and soles of their feet. With rash onset, fever decreases, but the patient does not fully defervesce. Lesions begin as maculas that progress to papules, then firm vesicles, and then deep-seated, hard pustules described as "pearls of pus," with each stage lasting 1–2 days. By day 6 or 7 of the rash, lesions may begin to umbilicate or become confluent. Lesions increase in size for approximately 8–10 days, after which they begin to crust. Once all the lesions have separated, 3–4 weeks after the onset of rash, the patient is no longer infectious. Infected people sustain significant scarring after the crusts have separated. Because of the relatively slow and steady evolution of the rash lesions, all lesions on any one part of the body are in the same stage of development (see Figure 4.5, smallpox lesions).

Varicella (chickenpox) is the condition most likely to be mistaken for smallpox. Generally, children with varicella do not have a febrile prodrome; adults may have a brief, mild prodrome. Although the two diseases can be easily confused in the first few days of the rash, smallpox lesions develop into pustules that are firm and deeply embedded in the dermis, whereas varicella lesions develop into superficial vesicles. Because varicella erupts in crops of lesions that evolve quickly, lesions on any one part of the body are in different stages of development (papules, vesicles, and crusts; see Figure 4.6, varicella lesions). The distribution of the rash in the two diseases differs. Varicella most commonly affects the face and trunk with relative sparing of the extremities, and lesions on the palms or soles are rare (see Figure 4.4, distribution of smallpox lesions vs. varicella lesions).

In addition to the typical presentation of smallpox (≥90% of cases), there are two uncommon forms of variola major:

- Hemorrhagic, characterized by hemorrhage into skin lesions and disseminated intravascular coagulation.
- Malignant or flat type, in which the skin lesions do not progress to the pustular stage but remain flat and soft.

In the past, each variant occurred in approximately 5% of cases and was associated with a 95%–100% mortality rate. Hemorrhagic smallpox rash commonly was confused with meningococcemia. Flat-type (velvety) smallpox occurred more commonly in children. By contrast, variola minor, or alastrim, was associated with fewer lesions, more rapid progression of rash, and a much lower mortality rate (approximately 1%) than variola major, or typical smallpox.

Smallpox is spread most commonly in droplets from the oropharynx of infected individuals, although infrequent transmission from aerosol and direct contact with infected lesions, clothing, or bedding has been reported. Patients are not infectious during the incubation period or febrile prodrome but become infectious with the onset of mucosal lesions (enanthemas), which occur within hours of the rash. The first week of rash illness is regarded as the most infectious period, although patients remain infectious until all scabs have separated. Because most smallpox patients are extremely ill and bedridden, spread generally is limited to household contacts, hospital workers, and other health care professionals. Secondary household attack rates for smallpox were considerably lower than for measles and similar to or lower than rates for varicella. The incubation period is 7–17 days (mean 12 days).

Variola major in unimmunized people was associated with case fatality rates of approximately 30% during epidemics of smallpox. The mortality rate was highest in children younger than 1 year and adults older than 30. The potential for modern supportive therapy in improving outcome is not known. Death was most likely to occur during the second week of illness and was attributed to overwhelming viremia. Secondary bacterial infections occurred but were a less significant cause of mortality.

For help in evaluating a rash illness suspicious of smallpox, see http://www.bt.cdc.gov/agent/smallpox/diagnosis/riskalgorithm/index.asp.

Diagnosis. Variola virus can be detected in vesicular or pustular fluid by culture or by PCR assay. Electron microscopy can detect orthopoxvirus infection but cannot distinguish between viruses. Currently, variola diagnostic testing is conducted only at the CDC. Reports of patients classified by the CDC as at high risk of having smallpox will trigger a rapid response, with a team deployed to obtain specimens and advise on clinical management.

Treatment. There is no effective antiviral therapy available to treat smallpox. Infected patients should receive supportive care. Cidofovir, currently licensed for cytomegalovirus retinitis, has been suggested as having a role in smallpox therapy, but data to support its use in smallpox are not available. The drug must be given IV and is associated with significant renal toxicity. Vaccinia immune globulin (VIG) is reserved for certain complications of immunization and has no role in treatment of smallpox.

Control measures. If a patient is suspected of having smallpox, standard, contact, and airborne precautions should be implemented immediately, and the State and local health departments should be alerted at once. Hospital infection control personnel should be notified when the patient is admitted, and the patient should be placed in a private, airborne infection isolation room equipped with negative-pressure ventilation with high-efficiency particulate air filtration. Anyone entering the room must wear an N95 or higher-quality respirator, gloves, and gown, even if there is a history of recent successful immunization. If the patient is moved from the room, he or she should wear a mask and be covered with sheets or gowns to decrease the risk of fomite transmission. Rooms vacated by patients should be decontaminated using standard hospital disinfectants, such as sodium hypochlorite or quaternary ammonia solutions. Laundry

and waste should be discarded into biohazard bags and autoclaved, and bedding and clothing should be washed in hot water with laundry detergent followed by hot-air drying or incinerated.

Vaccination. Postexposure immunization (within 3–4 days of exposure) provides some protection against disease and significant protection against a fatal outcome. Any person who has had significant exposure to a patient with confirmed smallpox during the infectious stage of illness should be immunized as soon after exposure as possible, but within 4 days of first exposure. Because infected individuals are not contagious until the rash (and/or enanthema) appears, individuals exposed only during the prodromal period are not at risk.

Vaccinia immune globulin. Vaccinia immune globulin (VIG) prepared from plasma of immunized individuals was used in the past to prevent or modify smallpox when administered within 24 hours of a known exposure. Current supplies of VIG are used in the treatment of complications of smallpox immunization. The CDC is the only source of VIG in the United States. Supplies may be obtained by calling the CDC Smallpox Vaccine Adverse Events Clinical Information Line at 877-554-4625 for physicians in civilian medical facilities.

Reporting. Cases of febrile rash illness for which smallpox is being considered in the differential diagnosis should be reported immediately to local or State health departments. After evaluation by the State or local health department, if smallpox laboratory diagnostic testing is considered necessary, the CDC Rash Illness Evaluation Team should be consulted at 770-488-7100. Laboratory confirmation of smallpox is available only from the CDC. (See Figure 4.8, evaluating febrile rash illness in patients suspected of having smallpox [algorithm]).

Tularemia

Tularemia is caused by *Francisella tularensis*, a small, nonmotile, aerobic, gram-negative coccobacillus. *Francisella tularensis* is one of the most infectious pathogens known; inoculation with or inhalation of as few as 10 organisms can cause disease. It is found in diverse animal hosts and can be recovered from contaminated water, soil, and vegetation. Small mammals, including voles, mice, water rats, squirrels, rabbits, and hares, are natural reservoirs. They acquire infection through tick, fly, or mosquito bites and by contact with contaminated environments. Natural infection in people occurs through bites of infected arthropods; handling infectious animal tissues or fluids; direct contact with or ingestion of contaminated food, water or soil; or inhalation of infective aerosols. Person-to-person transmission does not occur.

Aerosol release of *F. tularensis* as a bioterrorist event would be expected to cause primarily pleuropneumonitis, but some exposures might result in ocular tularemia, ulceroglandular or glandular disease, or oropharyngeal disease with cervical lymphadenitis. Release in a densely populated area would be expected to result in an abrupt onset of large numbers of people with acute, nonspecific febrile illness beginning 3–5 days later (incubation period is 1–14 days), with pleuropneumonitis developing in a significant proportion of cases during the ensuing days and weeks.

Signs and symptoms. *Francisella tularensis* is a facultative intracellular bacterium that multiplies within macrophages. Major target organs are the lymph nodes, lungs and pleura, spleen, liver, and kidney. Bacteremia may be common in early stages. Initial tissue reaction is a

focal, intensely suppurative necrosis that becomes granulomatous. After inhalational exposure, hemorrhagic inflammation of the airways develops and progresses to bronchopneumonia. Pleuritis with adhesions, and effusion and hilar lymphadenopathy are common.

Illness begins with fever, headache, chills and rigors, generalized body aches, coryza, and sore throat. There may be a dry or slightly productive cough and substernal pain or tightness with or without objective signs of pneumonia. These findings are followed by sweats, fever, chills, progressive weakness, malaise, anorexia, and weight loss. These signs and symptoms would be similar to those caused by Q fever, but the progression of illness would be expected to be slower and the case-fatality rate lower than in inhalational plague or anthrax.

Diagnosis. *Francisella tularensis* can be isolated from respiratory secretions and, sometimes, from blood in cases of inhalational infection. Gram stain, fluorescent antibody, or immunohistochemical stains (performed in designated reference laboratories in the National Public Health Laboratory Network) may demonstrate the organism in secretions, exudates, or biopsy specimens. If tularemia is suspected, the laboratory should be informed to minimize risks of transmission to laboratory personnel. Routine diagnostic procedures can be performed in Biosafety Level 2 conditions. Cultures in which *F. tularensis* is suspected should be examined in a biological safety cabinet. Manipulation of cultures and other procedures that might produce aerosols or droplets (e.g., grinding, centrifuging, vigorous shaking, animal studies) should be conducted under Biosafety Level 3 conditions. Bodies of patients who die of tularemia should be handled using standard precautions. Autopsy procedures likely to produce aerosols or droplets should be avoided. Clothing or linens contaminated with body fluids of patients with tularemia should be disinfected per standard hospital procedure.

Treatment. In case of a bioterrorist event, antimicrobial susceptibility testing of isolates should be conducted quickly and treatment altered according to test results and clinical response. For treatment recommendations in children before test results are known, see Table 4.6.

Control measures. Treatment with streptomycin, gentamicin, doxycycline, or ciprofloxacin started during the incubation period of tularemia and continued daily for 14 days can protect against symptomatic infection. Therefore, if an attack is discovered before individuals become ill, those who have been exposed should be treated prophylactically with oral doxycycline or ciprofloxacin for 14 days. If an attack is discovered only after individuals become ill, a fever watch should begin for those who potentially have been exposed. Treatment (as outlined above) should begin in those who develop an otherwise unexplained fever or flu-like illness within 14 days of presumed exposure.

Postexposure prophylactic treatment of those in close contact with tularemia patients is not recommended because person-to-person transmission is not known to occur. Standard precautions should be used in caring for hospitalized patients.

Reporting. Initial suspicion of a bioterrorist event involving *F. tularensis* will likely involve identification of more than one case in a nonendemic area. If this happens, immediately contact the local and State health departments and hospital infection control practitioner. If they are unavailable, contact the CDC at 770-488-7100.

For additional information, see http://www.bt.cdc.gov/agent/tularemia/facts.asp and http://www.bt.cdc.gov/agent/tularemia/faq.asp.

Viral Hemorrhagic Fevers

The term "viral hemorrhagic fevers (VHFs)" refers to a group of illnesses that are caused by several distinct families of viruses. In general, the term "viral hemorrhagic fever" is used to describe a severe multisystemic syndrome. Characteristically, the overall vascular system is damaged, and the body's ability to regulate itself is impaired. Although some types of hemorrhagic fever viruses cause relatively mild illnesses, many of these viruses cause severe, life-threatening disease.

VHFs are caused by RNA viruses of four distinct families:

- Arenaviruses (including Lassa fever).
- Filoviruses (including Rift Valley fever and hantavirus).
- Bunyaviruses (including Ebola and Marburg hemorrhagic fever).
- Flaviviruses (including tick-borne encephalitis).

In nature, the survival of these viruses depends on an animal or insect host called the natural reservoir. They are geographically restricted to the areas where their host species lives, and people are not the natural reservoir for any of these viruses. People may become infected when they come into contact with infected hosts, and in some cases, people can transmit the virus to one another. With a few exceptions, there is no cure or established drug treatment for VHFs.

Signs and symptoms. Specific signs and symptoms vary by the type of VHF, but initial signs and symptoms often include marked fever, fatigue, dizziness, muscle aches, loss of strength, and exhaustion. Other signs and symptoms can include vomiting, diarrhea, abdominal pain, chest pain, cough, and pharyngitis. A maculopapular rash, predominantly on the trunk, develops in many patients about 5 days after the onset of symptoms. Patients with severe VHF often show signs of bleeding under the skin, in internal organs, or from body orifices like the mouth, eyes, or ears. However, although they may bleed from many sites around the body, patients rarely die because of blood loss. Severely ill patients may go into shock with nervous system malfunction, coma, delirium, and seizures. Some types of VHF are associated with renal failure.

The incubation period is 4–21 days. The mortality rate varies depending on the specific virus involved.

Diagnosis. Diagnosis of VHF introduced through bioterrorism is likely to be recognized only after a cluster of patients present with similar, severe illness. Clinical suspicion should prompt notification of infection control and State health officials. Serum for antibody testing and tissue samples should be sent through your State health department to the CDC.

Treatment. In general, there is no specific treatment or established cure for VHFs. Treatment is supportive. Ribavirin has been effective in treating some individuals with Lassa fever or hemorrhagic fever with renal syndrome. Treatment with convalescent-phase plasma has been used with success in some patients with Argentinean hemorrhagic fever.

Control measures. Some viruses that cause hemorrhagic fever—including Ebola, Marburg, Lassa fever, and Crimean-Congo hemorrhagic fever viruses—can spread from one person to another (once an initial person has become infected). This type of secondary transmission of the virus can occur directly through close contact with infected people or their blood or other body fluids. Contaminated syringes and needles have been involved in the spread of infection in outbreaks of Ebola hemorrhagic fever and Lassa fever.

Both standard precautions and contact precautions should be used in caring for patients with suspected or confirmed VHF. A surgical mask and eye protection should also be worn by those coming within 3 feet of a patient with suspected or confirmed Lassa fever, Crimean-Congo hemorrhagic fever, or filovirus infections. Airborne isolation, including use of a HEPA-filtered respirator, should be used if patients with these conditions have prominent cough, vomiting, diarrhea, or hemorrhage. Decontamination should be performed using hypochlorite or phenolic disinfectants.

There are no vaccines to protect against these diseases, except for yellow fever and Argentinean hemorrhagic fever. For more information about specific VHF illnesses and their management, see http://www.cdc.gov/ncidod/dvrd/spb/mnpages/vhfmanual/anx2.pdf.

Reporting. These viruses are highly pathogenic and require handling in special laboratory facilities designed to contain them (Biosafety Level 4 facilities). If VHF is suspected, contact your State and local health departments immediately. If local and State health departments are unavailable, contact the CDC at 770-488-7100.

Category B and C Agents

Ricin

Ricin is a potent cytotoxin that can be easily extracted from the beans of the castor plant (*Ricinus communis*). Castor beans are processed worldwide in production of castor oil, and ricin-rich waste mash is a by-product. Ricin can be prepared in liquid, crystalline, or powder form; as an agent of terrorism, it could be disseminated as an aerosol, injected, or used to contaminate food or water. Symptoms depend on the route of exposure: respiratory, enteral, or parenteral. Compared with other biological toxins (e.g., botulinum toxin), ricin has low toxicity, and large quantities would be required to affect large numbers of people.

Signs and symptoms. Ricin inhibits cellular protein synthesis. Aerosol exposure results in fever, chest tightness, cough, dyspnea, nausea, and arthralgias after a delay of 4–8 hours. Death after aerosol exposure has not been reported in people, but animals develop necrosis and severe alveolar fluid collection. Ingestion of ricin causes necrosis of the GI epithelium with local necrosis of muscle and regional lymph nodes. Intravascular injection causes minimal pulmonary perivascular edema.

Diagnosis. Ricin exposure should be suspected if a geographic cluster of individuals develop acute lung injury. Pulmonary edema develops 1–3 days after exposure (compared with about 12 hours after *Staphylococcus* enterotoxin B exposure and about 6 hours after phosgene exposure).

Ricin provokes a specific antibody response, and acute and convalescent sera should be obtained for antibody titer. Tissue can also be stained using immunohistochemical methods.

Treatment. Treatment involves supportive care, including appropriate respiratory support and treatment for pulmonary edema if required. Enteral exposure should be treated by vigorous gastric lavage and use of cathartics.

Control measures. Protective masks are effective in preventing exposure. No vaccine is available.

Reporting. If ricin exposure is suspected, contact your State and local health departments. If they are unavailable, contact the CDC at 770-488-7100.

Q Fever

Q fever is caused by *Coxiella burnetti*, a rickettsial organism that causes usually asymptomatic infection in farm animals (cattle, sheep, goats). It can also infect dogs, cats, rodents, and some birds. Natural infection in people is rare. When it does occur, it usually is transmitted by aerosolized organisms from the tissues, fluids, or excreta of infected animals. Exposure through terrorism would likely involve aerosolization, and resulting disease would likely be similar to naturally occurring disease.

Signs and symptoms. The incubation period is 9–39 days after exposure. Initial symptoms include sudden onset of fever, chills, headache, weakness, lethargy, anorexia, and profuse sweating. Approximately 50% of infected individuals have pneumonia. Liver function tests are often abnormal—a result of granulomatous hepatitis—but jaundice is rare. Neuropathies sometimes develop. Transmission to the fetus is common when pregnant women are infected. The infection becomes chronic in approximately 1% of infected individuals and can manifest as endocarditis or hepatitis.

Diagnosis. Clinically, Q fever is not easily distinguished from other causes of flu-like symptoms and pneumonia. *Coxiella* can be isolated from blood cultures; however, if Q fever is suspected, blood cultures are not recommended because of the risk of exposure of laboratory personnel. PCR assays can identify the organism in tissue or environmental samples. Acute and convalescent sera should be submitted for antibody titers; antibody concentration may rise only after 2–3 weeks of illness.

Treatment and prophylaxis. Most infections resolve without specific therapy. Treatment with doxycycline may hasten recovery in acute infection. Chronic infection may require prolonged or repeated treatment. Chloramphenicol and ciprofloxacin are alternate choices for children <8 years of age and for pregnant women, respectively. The same antibiotics may be used for prophylaxis after known or suspected exposure. Prophylaxis should be delayed for 8–12 days after exposure and should be given for 5–7 days. Earlier prophylactic treatment may delay but not prevent disease.

Control measures. Person-to-person transmission is not known to occur, although transmission from contaminated clothing has been reported. Soap and water or a 0.5% chlorine solution can be used for decontamination.

Reporting. If Q fever is suspected, contact your local and State health departments. If they are unavailable, contact the CDC at 770-488-7100.

Staphylococcal Enterotoxin B

Staphylococcus enterotoxin B (SEB) is an exotoxin that acts on the intestine to produce a brisk cascade of pro-inflammatory cytokines, resulting in an intense inflammatory response. Food poisoning due to SEB results from ingestion of improperly handled food that contains enterotoxin.

Signs and symptoms. Natural disease is generally localized to the GI tract. After a brief incubation period (30 minutes to 8 hours, usually 2–4 hours), the exposed individual experiences abrupt and sometimes violent onset of severe nausea, abdominal cramps, vomiting, and prostration, often accompanied by diarrhea. There may be an associated low-grade fever or subnormal temperature. Symptoms typically last 1–2 days. Inhalational exposure, as might be expected in an incident of bioterrorism, results in predominantly respiratory symptoms, including nonproductive cough, retrosternal chest pain, and dyspnea. GI symptoms may be seen as well if toxin is inadvertently swallowed. Fever (103°–106°F) is likely and may last up to 5 days with chills and prostration. There may be conjunctival injection, and fluid losses may lead to postural hypotension. Chest radiographs are likely to be normal, but overt pulmonary edema can occur.

Diagnosis. Clinically, symptoms of staphylococcal enterotoxin ingestion can be distinguished from other causes of food poisoning except those due to *Bacillus cereus*. Illness due to *Clostridium perfringens* has a shorter incubation period and rarely is accompanied by vomiting. Foodborne *Salmonella* or *Shigella* infection usually is accompanied by fever.

Symptoms of inhalation of SEB are similar to those caused by many other respiratory pathogens. However, the clinical condition of respiratory disease due to inhaled SEB would be expected to stabilize without specific therapy, unlike that caused by tularemia, anthrax, or pneumonic plague. The absence of infiltrates on chest radiographs should also help distinguish respiratory tract disease caused by SEB from that caused by other likely agents of bioterrorism.

SEB antigen can be detected in urine, serum, or respiratory secretions. In addition, acute and convalescent serum samples should be submitted for antibody titer.

Treatment. Supportive care should include close attention to hydration and oxygenation.

Control measures. SEB is not absorbed through intact skin, and secondary aerosolization from affected patients is not hazardous. Environmental surfaces may be decontaminated using soap and water. Contaminated foodstuffs should be destroyed.

Reporting. If disease due to SEB is suspected, contact your local and State health departments. If they are unavailable, contact the CDC at 770-488-7100.

Brucella

Brucella species that infect people include *B. abortus*, *B. melitensis*, *B. suis*, and rarely, *B. canis*. *Brucella* species are small, gram-negative coccobacilli. People contract disease naturally through direct contact with infected animals and their carcasses or secretions or by ingestion of unpasteurized milk or milk products. *Brucella* species, particularly *B. melitensis* and *B. suis*, are potential terrorist agents. Aerosolization also can result in human infection.

Signs and symptoms. Most infected individuals become ill within 3–4 weeks of exposure, but the incubation period may vary from <1 week to several months. Clinical features after natural exposure are extremely variable and nonspecific. They include flu-like symptoms—i.e., fever, sweats, malaise, anorexia, headache, myalgia, and back pain. Disease in children is commonly mild and self-limited, but in adults the illness is more chronic. Physical findings may include lymphadenopathy, hepatosplenomegaly, and occasionally, arthritis. Serious complications include meningitis, endocarditis, and osteomyelitis.

Diagnosis. *Brucella* can be recovered in culture from blood, bone marrow, or other tissues. Specimens should be incubated for a minimum of 4 weeks. Serum samples collected at least 2 weeks apart can confirm the diagnosis with a four-fold rise in antibody titers. A polymerase chain reaction (PCR) test has been developed but is available only in reference laboratories.

Treatment and prophylaxis. Oral doxycycline (2-4 mg/kg/day, max 200 mg/day, divided BID) should be administered for 4–6 weeks. For children younger than 8 years, trimethoprim-sulfamethoxazole may be used (trimethoprim, 10 mg/kg/day, max 480 mg/day; sulfamethoxazole 50 mg/kg/day, maximum 2.4 g/day) divided BID for 4–6 weeks. Rifampin (15–20 mg/kg/day, max 600–900 mg/day), divided BID, should be added to prevent relapse. For patients who have endocarditis, osteomyelitis, or meningitis, therapy is often extended for several months with streptomycin sulfate or gentamicin sulfate added to the above regimens.
Prophylaxis after suspected exposure should be provided using doxycycline and rifampin.

Control measures. Standard precautions provide adequate protection from spread of infection, except that contact precautions should be added for patients with draining wounds.

Reporting. Reporting suspected *Brucella* infection, regardless of mechanism of exposure, is mandated throughout the United States. You should contact your local and State health departments. If they are unavailable, contact the CDC at 770-488-7100.

Burkholderia mallei (Glanders)

Glanders is caused by the gram-negative bacillus *Burkholderia mallei*. Most cases occur in horses, mules, or donkeys, but people may become infected through handling infected animals. There have been no naturally acquired cases of glanders in people in the United States in more than half a century; most human cases occur in Asia, the Middle East, or South America. It is believed that this organism was used during WWI and WWII as a weapon of terrorism to infect horses, mules, and people. Human cases diagnosed in the absence of animal cases should raise suspicion of terrorism.

Signs and symptoms. The incubation period after exposure ranges from 1 to 14 days. Acute and chronic presentation is possible, but acute illness is most likely after a bioterrorist event. Disease may be localized (e.g., pneumonia) or disseminated (fulminant sepsis). Most commonly, symptoms include high fever, mucositis, and abscesses in multiple organs, predominantly the lungs, liver, and spleen.

Symptoms and signs associated with acute septicemia include fever, rigors, headache, muscle pain, night sweats, pleuritic chest pain, jaundice, sensitivity to light, and diarrhea. Diffuse erythroderma may be accompanied by necrotizing lesions. Cervical adenopathy, tachycardia, and mild hepatomegaly or splenomegaly may be present.

Acute localized disease may involve the lungs (after inhalation of particles or through hematogenous spread). In addition to the signs and symptoms associated with acute septicemia (above), miliary lesions and/or bilateral upper lobe infiltrates with or without consolidation or cavitation may be noted on chest radiograph. Mucous membrane involvement begins with nasal ulcers and nodules that secrete bloody discharge and often lead to sepsis. A papular and/or pustular rash, similar in appearance to the smallpox rash, may develop. Liver and spleen abscesses may be present. Septic shock usually follows.

Diagnosis. Small bacilli may be seen on methylene blue or Wright stain of exudates. Both *B. mallei* and *B. pseudomallei* can be grown and identified from standard cultures.

Treatment and prophylaxis. Without effective antibiotic therapy, mortality nears 100%. Localized disease may be treated successfully with oral therapy for 60-150 days, while systemic illness requires parenteral therapy. Definitive antibiotic therapy should be based on susceptibility testing. Presumptive therapy can be provided using amoxicillin/clavulanate (60 mg/kg/day, PO, divided TID), tetracycline (40 mg/kg/day, PO, divided TID), or trimethoprim-sulfamethoxazole (trimethoprim 4 mg/kg/day; sulfamethoxazole 20 mg/kg/day, PO, divided BID) for localized disease.

The effectiveness of prophylactic, postexposure therapy is not known. Trimethoprim-sulfamethoxazole may be tried.

Control measures. Person-to-person transmission is unlikely after inhalational exposure as would be expected in disease due to terrorism. Transmission from direct contact between nonintact skin or mucous membranes and infected animal tissue is the usual means of natural infection. Standard precautions are adequate for most patients, while contact precautions should be added for patients with skin lesions. Environmental decontamination using 0.5% hypochlorite solution (bleach) is effective.

Reporting. If glanders is suspected, contact your local and State health departments. If they are unavailable, contact the CDC at 770-488-7100.

Encephalitis Viruses and Yellow Fever Virus

Arboviruses (arthropod-borne viruses) are spread by mosquitoes, ticks, or sand flies and produce four principal clinical syndromes:

- CNS infection (including encephalitis, aseptic meningitis, or myelitis).
- Undifferentiated febrile illness, often with rash.
- Acute polyarthropathy.
- Acute hemorrhagic fever, usually accompanied by hepatitis.

Infection with some arboviruses results in perinatal illness. Alpha viruses, including Eastern equine encephalitis (EEE) virus, Western equine encephalitis (WEE) virus, and Venezuelan equine encephalitis (VEE) virus, and yellow fever virus, a member of the Flavivirus genus, have been included in the CDC's list of potential agents of bioterrorism. In nature, disease due to these viral agents is limited to the geographic areas in which their arthropod vectors live.

Signs and symptoms. For many encephalitis viruses, asymptomatic infection is common. Clinical illness, when it occurs, ranges in severity from a self-limiting febrile illness with headache and vomiting to a syndrome of aseptic meningitis or acute encephalitis. EEE virus infection is typically a fulminant illness that leads to coma and death in one-third of cases and to serious neurologic sequelae in another third. The clinical severity of WEE virus infection is intermediate, with a case fatality rate of 5%; neurologic impairment is common in infants. VEE virus infection produces acute systemic febrile illness, with encephalitis developing in a small percentage (4% in children; <1% in adults). The incubation period for EEE and WEE encephalitis viruses is 2–10 days, while that for VEE virus infection is 1–4 days.

Yellow fever virus infection evolves through three periods from a nonspecific febrile illness (with headache, malaise, weakness, nausea, and vomiting) through a brief period of remission, to a hemorrhagic fever with GI tract bleeding and hematemesis, jaundice, hemorrhage, cardiovascular instability, albuminuria, oliguria, and myocarditis. The incubation period is 3–6 days, and 50% of cases are fatal.

Diagnosis. Diagnosis is made by serologic testing of cerebrospinal fluid (CSF) or serum or by viral isolation. Detection of virus-specific IgM antibody in CSF is confirmatory, and its presence in a serum sample is presumptive evidence of recent infection in a patient with acute CNS infection. Greater than four-fold change in serum antibody titer in paired serum samples obtained 2–4 weeks apart is confirmatory. A single increased antibody titer defines a case as presumptive. During the acute phase of yellow fever and VEE virus infection, virus can be isolated from blood and, in Venezuelan encephalitis, from the throat.

Treatment. Active clinical monitoring and supportive therapy may be life saving.

Control measures. Respiratory precautions are recommended when caring for patients with VEE virus infection. Patients with yellow fever may have active virus circulating in the blood, so they should be kept away from potential vector mosquitoes that could feed on them and subsequently transmit infection to others. Standard precautions are recommended for patients with EEE and WEE virus infection. A live-attenuated yellow fever vaccine is available and is

currently used for individuals traveling to countries where yellow fever is endemic (some parts of South America and Africa). The vaccine is currently available in the United States only at approved vaccination centers.

Reporting. If viral encephalitis or yellow fever is suspected, contact your local and State health departments. If they are unavailable, contact the CDC at 770-488-7100.

Clostridium perfringens

Food poisoning may be caused by a heat-labile toxin produced in vivo by *C. perfringens* type A; type C causes enteritis necroticans. Spores of *C. perfringens* may survive cooking. Spores germinate and multiply during slow cooling and storage at temperatures of 20°–60°C (68°–140°F). Once ingested, an enterotoxin produced by the organisms in the lower intestine is responsible for symptoms. Beef, poultry, gravies, and dried or precooked foods are common sources. Infection usually is acquired when food is prepared in large quantities and kept warm for prolonged periods (e.g., at banquets or institutions or from food provided by caterers or restaurants).

Signs and symptoms. Onset of watery diarrhea and moderate to severe, crampy, midepigastric pain is sudden. Vomiting and fever are uncommon. Symptoms usually resolve in 24 hours. The incubation period is usually 8–12 hours.

Diagnosis. The short incubation, short duration, and absence of fever in most patients differentiates *C. perfringens* foodborne disease from shigellosis and salmonellosis. The infrequency of vomiting and longer incubation period contrast with the clinical features of foodborne disease associated with heavy metals, *Staphylococcus aureus* enterotoxins, and fish and shellfish toxins. Diarrheal disease caused by *Bacillus cereus* enterotoxin may be indistinguishable from that caused by *C. perfringens*. Enteritis necroticans is a cause of severe illness and death attributable to *C. perfringens* food poisoning among children in Papua, New Guinea (where it is also known as pigbel).

Because the fecal flora of healthy people commonly includes *C. perfringens*, counts of 10^6/gram of feces obtained within 48 hours of onset of illness are required to support the diagnosis. The diagnosis can be suggested by detection of *C. perfringens* enterotoxin in feces by commercially available kits. To confirm *C. perfringens* as the cause, the concentration of organisms should be at least 10^5/gram in the epidemiologically implicated source of infection (food). Although *C. perfringens* is an anaerobe, special transport conditions are unnecessary because the spores are durable. Fecal samples, rather than rectal swab specimens, should be obtained.

Treatment. Oral rehydration or, occasionally, IV fluid and electrolyte replacement may be indicated to prevent or treat dehydration. Antimicrobial agents are not indicated.

Control measures. *Clostridium perfringens* food poisoning is not transmissible from person to person.

Reporting. If *C. perfringens* food poisoning is suspected, contact your local and State health departments. If they are unavailable, contact the CDC at 770-488-7100.

Bibliography

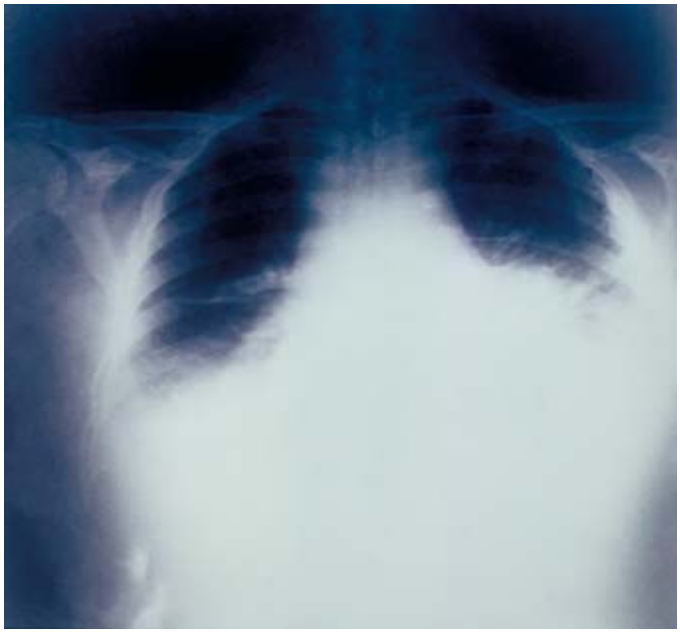
- Arnon SS, Schechter R, Inglesby TV, et al. Botulinum toxin as a biological weapon: medical and public health management. *JAMA.* 2001;285:1059–1070.
- Borio L, Inglesby T, Peters CJ, Schmaljohn, et al. Hemorrhagic fever viruses as biological weapons: medical and public health management. *JAMA.* 2002:287:2391–2405.
- Centers for Disease Control and Prevention. Acute, Generalized Vesicular or Pustular Rash Illness Protocol. An Algorithm for Evaluating Patients for Smallpox. Available at: http://www.bt.cdc.gov/agent/smallpox/diagnosis/evalposter.asp. Accessed July 12, 2006.
- Centers for Disease Control and Prevention. Anthrax. Available at: http://www.bt.cdc.gov/agent/anthrax/index.asp. Accessed July 13, 2006.
- Centers for Disease Control and Prevention. Bioterrorism Readiness Plan: A Template for Healthcare Facilities. April 1999. Available at: http://www.cdc.gov/ncidod/dhqp/pdf/bt/13apr99APIC-CDCBioterrorism.PDF. Accessed July 12, 2006.
- Centers for Disease Control and Prevention. Emergency Preparedness and Response. Agents, Diseases, & Other Threats. Available at: http://www.bt.cdc.gov/. Accessed July 13, 2006.
- Centers for Disease Control and Prevention. Emergency Preparedness and Response. Botulism. Available at: http://www.bt.cdc.gov/agent/botulism/index.asp. Accessed July 19, 2006.
- Centers for Disease Control and Prevention. Emergency Preparedness and Response. Clinician Registry for Terrorism and Emergency Response Updates and Training Opportunities. Available at: http://www.bt.cdc.gov/clinregistry/index.asp. Accessed July 12, 2006.
- Centers for Disease Control and Prevention. Emergency Preparedness and Response. Evaluate a Rash Illness Suspicious for Smallpox. Available at: http://www.bt.cdc.gov/agent/smallpox/diagnosis/riskalgorithm/index.asp. Accessed July 12, 2006.
- Centers for Disease Control and Prevention. Emergency Preparedness and Response. Frequently Asked Questions (FAQ) About Tularemia. Available at: http://www.bt.cdc.gov/agent/tularemia/faq.asp. Accessed August 17, 2006.
- Centers for Disease Control and Prevention. Emergency Preparedness and Response. Key Facts About Tularemia. Available at: http://www.bt.cdc.gov/agent/tularemia/facts.asp. Accessed July 19, 2006.
- Centers for Disease Control and Prevention. Emergency Preparedness and Response. Laboratory Information. Available at: http://www.bt.cdc.gov/labissues/index.asp. Accessed July 12, 2006.
- Centers for Disease Control and Prevention. Emergency Preparedness and Response. Smallpox Diagnosis and Evaluation. Available at: http://www.cdc.gov/ncidod/dvbid/plague/diagnosis.htm. Accessed July 19, 2006.
- Centers for Disease Control and Prevention. Guideline for Environmental Infection Control in Health-Care Facilities, 2003. Available at: http://www.cdc.gov/ncidod/dhqp/gl_environinfection.html.

Accessed July 12, 2006.
- Centers for Disease Control and Prevention. Guideline for Isolation Precautions in Hospitals. Available at: http://www.cdc.gov/ncidod/dhqp/gl_isolation.html. Accessed July 12, 2006.
- Centers for Disease Control and Prevention. The History of Bioterrorism (Video). Available at: http://www.bt.cdc.gov/training/historyofbt/index.asp. Accessed August 17, 2006.
- Centers for Disease Control and Prevention. Laboratory Response Network. Available at: http://www.bt.cdc.gov/lrn/. Accessed July 12, 2006.
- Centers for Disease Control and Prevention. Laboratory Response to Suspicious Substances. Available at: http://www.bt.cdc.gov/labissues/substanceresponse.asp. Accessed August 7, 2006.
- Centers for Disease Control and Prevention. Notice to readers: Update: interim recommendations for antimicrobial prophylaxis for children and breastfeeding mothers and treatment of children with anthrax. *MMWR.* 2001; 40(45):1014-1016.
- Centers for Disease Control and Prevention. Plague Diagnosis. Available at: http://www.cdc.gov/ncidod/dvbid/plague/diagnosis.htm. Accessed July 19, 2006.
- Centers for Disease Control and Prevention. Programs in Brief: Select Agent Program. Available at: http://www.cdc.gov/programs/php07.htm. Accessed August 17, 2006.
- Centers for Disease Control and Prevention. The Public Health Response to Biological and Chemical Terrorism. Interim Planning Guidance for State Public Health Officials, July 2001. Available at: http://www.bt.cdc.gov/Documents/Planning/PlanningGuidance.PDF. Accessed July 12, 2006.
- Centers for Disease Control and Prevention. Recognition of illness associated with the intentional release of a biologic agent. *MMWR.* 2001; 50(41):893-897.
- Centers for Disease Control and Prevention. Smallpox Response Plan and Guidelines (version 3.0). Available at: http://www.bt.cdc.gov/agent/smallpox/response-plan/index.asp. Accessed July 13, 2006.
- Centers for Disease Control and Prevention. Smallpox Vaccination. Information for Health Professionals. Available at: http://www.bt.cdc.gov/agent/smallpox/vaccination/index.asp. Accessed August 17,
2006.
- Centers for Disease Control and Prevention. Smallpox Vaccine Administration Video. Available at: http://www.bt.cdc.gov/agent/smallpox/vaccination/administration-video/index.asp. Accessed July 12, 2006.
- Centers for Disease Control and Prevention. Specimen Shipping and Packing. Available at: http://www.cdc.gov/ncidod/srp/specimens/shipping-packing.html. Accessed August 7, 2006.
- Centers for Disease Control and Prevention. State and Local Health Department Web Sites. Available at: http://www.cdc.gov/other.htm#states.
Accessed July 12, 2006.
- Centers for Disease Control and Prevention. Strategic National Stockpile. Available at: http://www.bt.cdc.gov/stockpile/. Accessed July 13, 2006.
- Centers for Disease Control and Prevention. Use of anthrax vaccine in the United States. *MMWR.* 2000; 49(RR-15):1-20.
- Centers for Disease Control and Prevention. Vaccinia (smallpox) vaccine. Recommendations of the Advisory Committee on Immunization Practices (ACIP), 2001. *MMWR.* 2001; 50(RR-10):1-25.

- Centers for Disease Control and Prevention. Special features of VHFs. Available at: http://www.cdc.gov/ncidod/dvrd/spb/mnpages/vhfmanual/anx2.pdf. Accessed July 19, 2006.
- Cono J, Casey CD, Bell DM. Smallpox vaccination and adverse events: Guidance for clinicians. *MMWR.* 2003; 52(RR-04):1-28.
- Council of State and Territorial Epidemiologists. Current State and Territorial Epidemiologists. Available at: http://www.cste.org/members/state_and_territorial_epi.asp. Accessed July 12, 2006.
- Davis CJ. Nuclear blindness: an overview of the biological weapons programs of the former Soviet Union and Iraq. *Emerg Infect Dis.* 1997;278(5):351–356.
- Dennis DT, Inglesby TV, Henderson DA, et al. Tularemia as a biological weapon: medical and public health management. *JAMA.* 2001;285:2763-2773.
- Franz DR, Jahrling PB, Friedlander AM, et al. Clinical recognition and management of patients exposed to biological warfare agents. *JAMA.* 1997:278; 399–411.
- Fritz SB, Singer AM, Revan VB, et al. Bioterrorism: relevance to allergy and immunology in clinical practice. *J Allergy Clin Immunol.* 2002;109(2):214–228.
- Garner JS, Hospital Infection Control Practices Advisory Committee. Guideline for isolation precautions in hospitals. *Infect Control Hosp Epidemiol.* 1996;17:53–80, and Am J Infect Control. 1996;24:24–52.
- Henderson DA, Inglesby TV, Bartlett JG, et al. Smallpox as a biological weapon: medical and public health management. *JAMA.* 1999;281:2127–2137.
- Inglesby TV, Henderson DA, Bartlett JG, et al. Anthrax as a biological weapon: medical and public health management. *JAMA.* 1999;281:1735–1745.
- Inglesby TV, Dennis DT, Henderson DA, et al. Plague as a biological weapon: medical and public health management. *JAMA.* 2000;283:2281–2290.
- Kadlec RP, Zelicoff AP, Vrtis AM. Biologic weapons control: prospects and implications for the future. *JAMA.* 1997;288:351–356.
- Kolavic SA, Kimura A, Simons SL, et al. An outbreak of *Shigella dysenteriae* Type 2 among laboratory workers due to intentional food contamination. *JAMA.* 1997;278:396–398.
- McGovern TW, Christopher GW, Eitzen EM. Cutaneous manifestations of biological warfare and related threat agents. *Arch Derm.* 1 999;135(3):311–322.
- Meffert, JJ. Biological warfare from a dermatologic perspective. *Curr Allergy Asthma Rep.* 2003; 3:304–310.
- National Child Traumatic Stress Network. Resources for Parents and Caregivers. Available at: http://www.nctsnet.org/nccts/nav.do?pid=ctr_aud_prnt. Accessed July 13, 2006.
- Noah DL, Huebner KD, Darling RG, et al. The history and threat of biological warfare and terrorism. *Emerg Med Clin No Am.* 2002;20:255–271.
- Olson KB. Aum Shinrikyo: once and future threat? *Emerg Infect Dis.* 1999;5(4):513–516.
- Patt HA, Feign RD. Diagnosis and management of suspected cases of bioterrorism: a pediatric perspective. *Pediatrics.* 2002;109(4);685–692.
- Public Health Security and Bioterrorism Preparedness and Response Act of 2002. Washington, DC: U.S. Congress; 2002. Available at: http://www.fda.gov/oc/bioterrorism/PL107-188.html. Accessed August 17, 2006.

- Roffey R, Tegnell A, Elg HF. Biological warfare in a historical perspective. *Clin Microbiol Infect.* 2002;8:450–454.
- Rotz LD, Khan AS, Lillibridge SR. Public health assessment of potential biological terrorism agents. *Emerg Infect Dis* [serial online] 2002 Feb;8(2). Available at: http://www.cdc.gov/ncidod/eid/vol8no2/01-0164.htm. Accessed August 17, 2006.
- Török TJ, Tauxe RV, Wise RP, et al. A large community outbreak of salmonellosis caused by intentional contamination of restaurant salad bars. *JAMA.* 1997;278:389–395.
- Treadwell TA, Koo D, Kurer K, et al. Epidemiologic clues to bioterrorism. *Pub Health Rep.* 2003; 18:92–98.

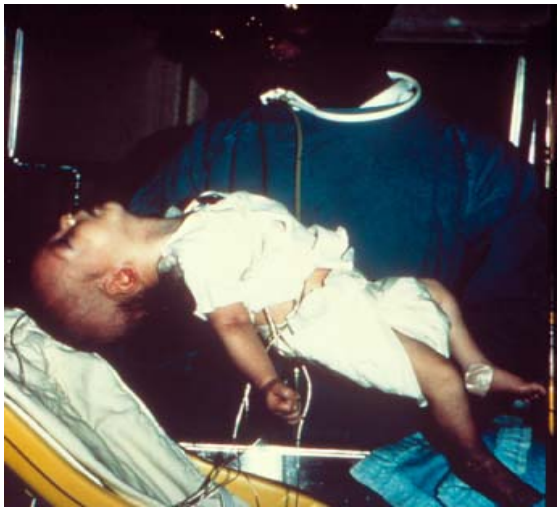
Figure 4.1. Chest radiograph taken 22 hours before death



Note: Film shows widened mediastinum due to inhalation anthrax.

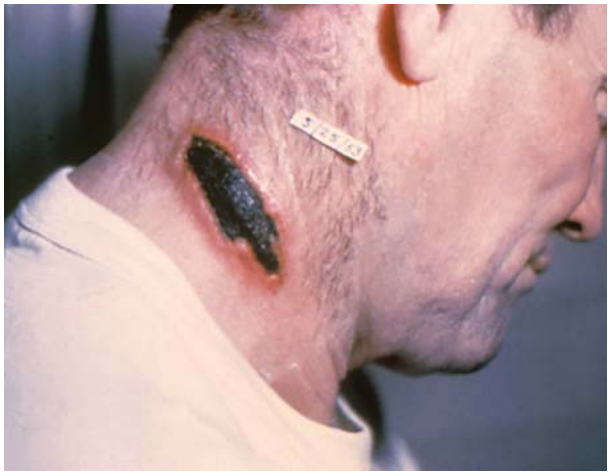
Source: Centers for Disease Control and Prevention. Available at http://www.bt.cdc.gov/agent/anthrax/.

Figure 4.2. Neurological signs, botulism, 6-week-old infant



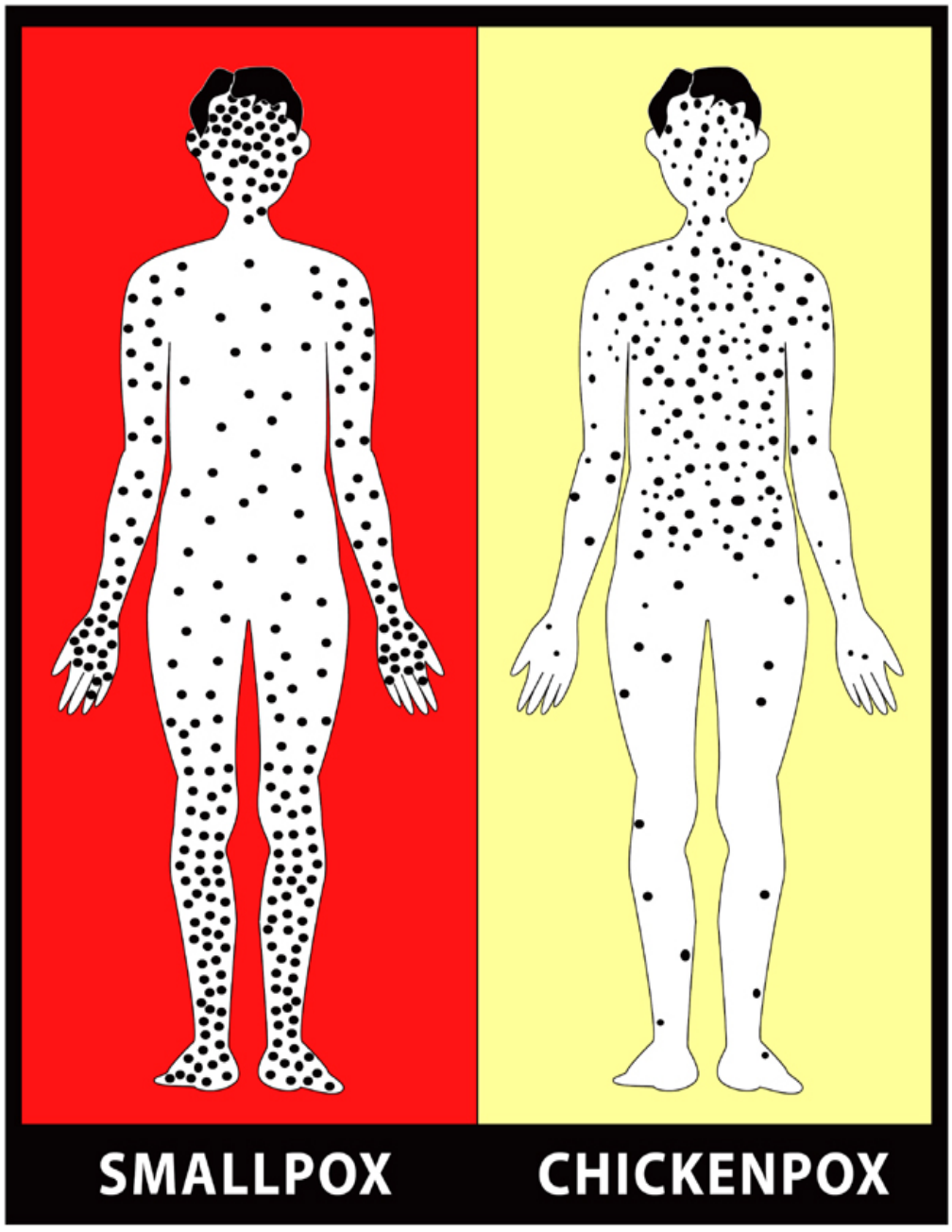
Source: Centers for Disease Control and Prevention. Available at http://www.bt.cdc.gov/bioterrorism/.

Figure 4.3. Cutaneous anthrax lesion with eschar on neck



Source: Centers for Disease Control and Prevention. Available at http://www.bt.cdc.gov/bioterrorism/.

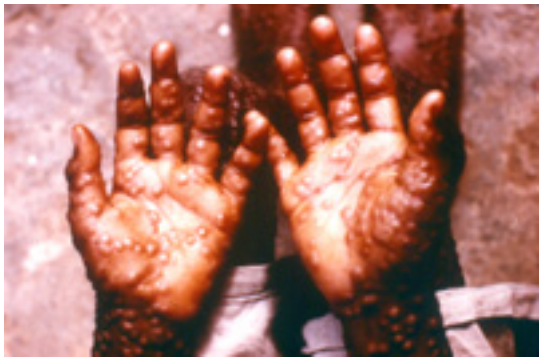
Figure 4.4. Physical distribution of smallpox lesions versus varicella lesions



Note: The smallpox lesions are concentrated on the face and extremities, including the palms and soles. In contrast, the varicella lesions are concentrated on the face and trunk

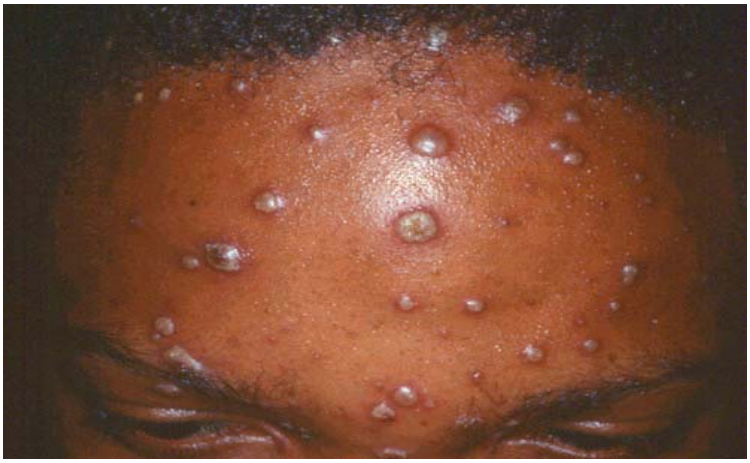
Source: Centers for Disease Control and Prevention. Available at http://www.bt.cdc.gov/agent/smallpox/.

Figure 4.5. Smallpox lesions



Note: The lesions are in the same stage of development, deep-seated, and umbilicated, and they appear to be firm.
Source. World Health Organization. Available at:
http://www.who.int/emc/diseases/smallpox/Africanseries/pages/WHO-Spx-Dx-Africa48-MedRes.htm.

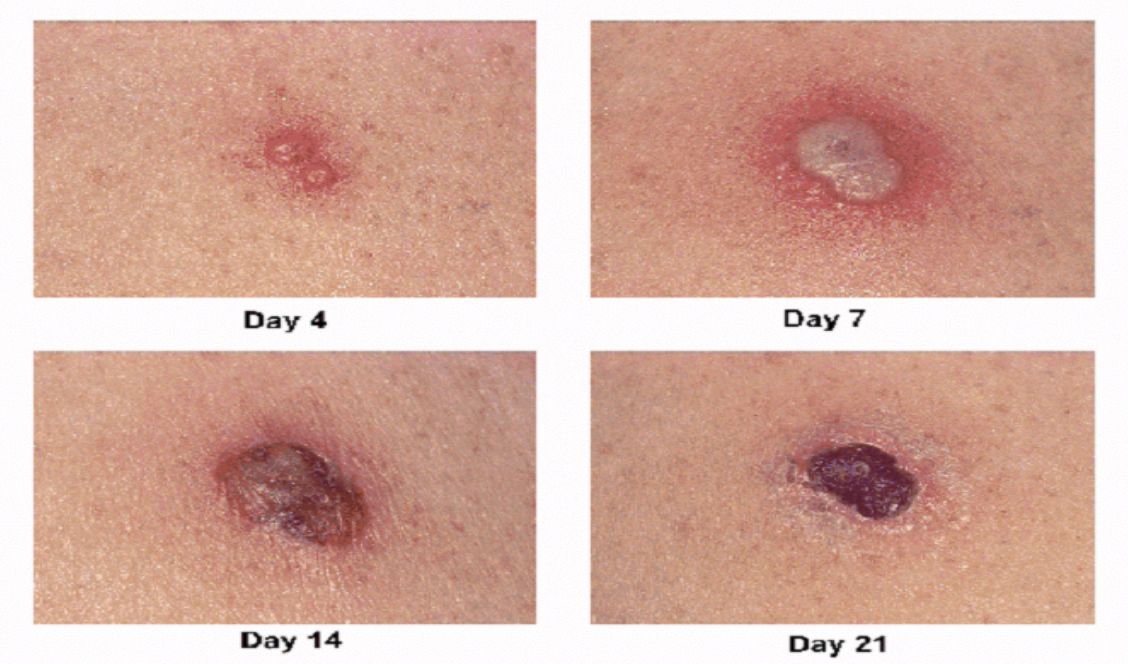
Figure 4.6. Varicella lesions



Note: The lesions are in varied stages of development, more superficial than those of smallpox, and irregular in shape.

Source: Centers for Disease Control and Prevention. Available at http://www.bt.cdc.gov/agent/smallpox/.

Figure 4.7. Expected smallpox vaccination site reaction (i.e., a "take") in a first-time vaccinate



Note: Figure demonstrates the progression from papule (day 4) to pustule (days 7-14) to scab (day 21).

Source: Centers for Disease Control and Prevention. Available at http://www.bt.cdc.gov/agent/smallpox/.

Figure 4.8a. Febrile rash illness algorithm for evaluating patients suspected of having smallpox

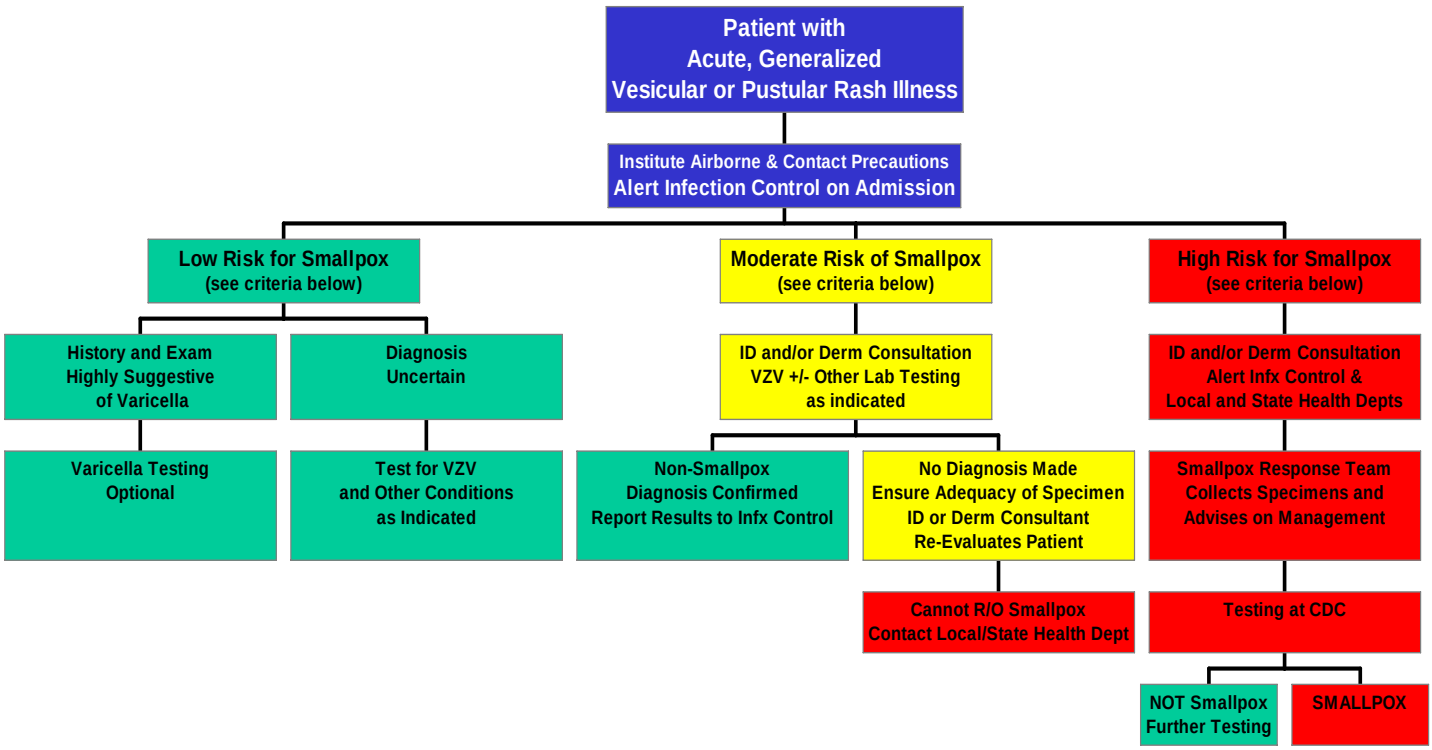


Figure 4.8b. Classification of risk – Febrile rash illness algorithm

- **High risk** (All three major criteria)
  - Febrile prodrome (1–4 days pre-rash, >101° F), ***and***
  - Classic smallpox lesions, ***and***
  - Same stage of development

- **Moderate risk**
  - Febrile prodrome, ***and***
  - 1 other major ***or*** ≥4 minor criteria

- **Low Risk**
  - No fever ***or***
  - Febrile prodrome ***and*** <4 minor criteria

Minor Criteria

- Greatest concentration of lesions on face and distal extremities
- Lesions first appeared on oral mucosa/palate, face, forearms
- Patient appears toxic or moribund
- Lesions evolve slowly from macules to papules to pustules over days
- Lesions on palms and soles

Source: CDC. Emergency Preparedness and Response. Poster: Evaluating patients for smallpox.

Note: Full protocol available at http://www.bt.cdc.gov/agent/smallpox/diagnosis/evalposter.asp.

Table 4.1. Early clinical signs and symptoms after exposure to selected bioterrorist agents

Clinical signs and symptoms*	Agent or disease
Respiratory	
Influenza-like illness ± atypical pneumonia	Tularemia Brucellosis Q fever Venezuelan equine encephalomyelitis Eastern equine encephalomyelitis Western equine encephalomyelitis
Influenza-like illness with cough and respiratory distress	Inhalational anthrax Pneumonic plague Inhalational tularemia Ricin Aerosol exposure to Staphylococcal enterotoxin B Hantavirus
Exudative pharyngitis and cervical lymphadenopathy	Oropharyngeal tularemia
Neurologic	
Flaccid paralysis	Botulism
Encephalitis	Venezuelan equine encephalomyelitis Eastern equine encephalomyelitis Western equine encephalomyelitis
Meningitis	Inhalational anthrax Septicemic and pneumonic plague Venezuelan equine encephalomyelitis Eastern equine encephalomyelitis Western equine encephalomyelitis
Gastrointestinal	
Diarrhea	*Salmonella* species *Shigella dysenteriae* *Escherichia coli* O157:H7 *Vibrio cholerae* *Cryptosporidium parvum*
Vomiting, abdominal pain, bloody diarrhea, hematemesis	GI anthrax
Dermatologic	
Vesicular rash† associated with fever, headache, malaise	Smallpox
Painless ulceration progressing to black eschar	Cutaneous anthrax
Ulcer plus painful regional lymphadenopathy and influenza-like illness	Ulceroglandular tularemia
Petechiae† with fever, myalgia, prostration	Viral hemorrhagic fever

Table 4.1. Early clinical signs and symptoms after exposure to selected bioterrorist agents, continued

Cardiovascular	
Shock after respiratory distress	Inhalational anthrax Ricin Viral hemorrhagic fever
Hematologic	
Thrombocytopenia	Brucellosis Viral hemorrhagic fever Hantavirus
Neutropenia	Viral hemorrhagic fever Venezuelan equine encephalomyelitis Eastern equine encephalomyelitis Western equine encephalomyelitis
Hemorrhage	Viral hemorrhagic fever
Disseminated vascular coagulation	Viral hemorrhagic fever
Renal	
Hemolytic-uremic syndrome, thrombotic thrombocytopenic purpura	*Escherichia coli* O157:H7 and other shiga toxin-producing *E. coli* *Shigella dysenteriae*
Oliguria, renal failure	Viral hemorrhagic fever Hantavirus
Other	
Painful lymphadenopathy	Bubonic plague
Purulent conjunctivitis with preauricular or cervical lymphadenopathy	Oculoglandular tularemia

* Based on route of exposure; likely to make someone seek medical attention; other manifestations (e.g., fever, headache, vomiting, diarrhea) possible and common early on in many illnesses.

† Rashes of diseases that cause petechiae or vesicular skin lesions may start as macular or papular lesions.

Table 4.2. Infection control transmission precautions for Category A agents

Agent	Transmission	Infection control	Special features
Anthrax	Contact (cutaneous form)	Standard	
Plague	Contact (bubonic form) Droplet (pneumonic form)	Standard, droplet	Masks
Tularemia	No	Standard	
Botulism	No	Standard	
Smallpox	Droplet and aerosol	Standard, airborne infection isolation	Fitted N-95 Negative-pressure room
Viral hemorrhagic fevers	Contact and droplet	Standard, contact, droplet	Masks

Table 4.3. Diagnostic procedures, isolation precautions, treatment, and postexposure prophylaxis for selected bioterrorist agents in children

Agent	Incubation period	Diagnostic specimens and procedures	Isolation precautions	Treatment	Postexposure prophylaxis[1]	Comments
Alphaviruses (VEE, EEE, and WEE)	2–10 days	CSF for viral isolation, antibody detection in CSF and acute and convalescent serum	Standard; respiratory precautions for WEE virus	Supportive	Protection from mosquito vectors	
Anthrax	1–60 days	Gram stain of buffy coat, CSF, pleural fluid, swab of skin lesion; culture of blood, CSF, pleural fluid, skin biopsy	Standard; contact for skin lesions	Ciprofloxacin[2] or doxycycline[3]; combine with one or two additional antimicrobial agents for inhalational, GI, or oropharyngeal disease[4]	Ciprofloxacin[2], doxycycline[3], or amoxicillin[5]; anthrax vaccine	Additional antimicrobial agents to be used for inhalational, GI, or oropharyngeal disease include rifampin, vancomycin, penicillin, ampicillin, chloramphenicol, imipenem, clindamycin, and clarithromycin
Botulism	Foodborne: 2 hr–8 days Inhalational: 24–72 hr	Toxin detection from serum, feces, enema fluid, gastric fluid, vomitus, or suspected food samples; culture of feces or gastric sections; nerve conduction testing	Standard	Supportive care; mechanical ventilation and parenteral nutrition may be required; equine botulism antitoxin given as soon as possible (CDC)[6]		Type-specific antitoxin should be administered when possible; antitoxin prevents additional nerve damage but does not reverse existing paralysis
Brucellosis	5–60 days	Culture of blood or bone marrow; acute and convalescent serum for antibody testing	Standard; contact for draining skin lesions	Doxycycline[3] and rifampin; if younger than 8 yr old, use TMP-SMX	Doxycycline[3] and rifampin	TMP-SMX may substitute for rifampin with doxycycline

100

Table 4.3. Diagnostic procedures, isolation precautions, treatment, and postexposure prophylaxis for selected bioterrorist agents in children, continued

Plague	2–4 days	Culture or fluorescent antibody staining of blood, sputum, lymph node aspirate	Droplet	Streptomycin sulfate or gentamicin sulfate; doxycycline[3] or tetracycline[3]	Doxycycline[3]; tetracycline[3]	TMP-SMX is an alternative; chloramphenicol for meningitis
Q fever	10–40 days	Acute and convalescent serum samples	Standard	Doxycycline[3] or tetracycline[3]	Doxycycline[3] or tetracycline[3]	Chloramphenicol is an alternative for treatment or prophylaxis
Smallpox	7–19 days	Culture of pharyngeal swab of skin lesions	Airborne, contact	Supportive care	Vaccine if administered within 4 days	
Staphylococcal enterotoxin B	3–12 hr	Serum, urine, and respiratory secretions for toxin; acute and convalescent serum for antibodies	Standard	Supportive care	None available	
Ricin	4–8 hr	Serum and/or respiratory secretions for enzyme immunoassay	Standard	Supportive care; gastric lavage and cathartics if toxin is ingested	Protective mask	
Viral hemorrhagic fevers	6–17 days	Culture and/or antigen detection of blood and other body tissues[7]; serum for acute and convalescent antibody detection	Standard, droplet, and contact precautions[8]	Ribavarin IV for Lassa fever; plasma from convalescent patients for Argentinean hemorrhagic fever; supportive care		

Table 4.3. Diagnostic procedures, isolation precautions, treatment, and postexposure prophylaxis for selected bioterrorist agents in children, continued

[1] Prophylaxis should be administered only after consultation with public health officials and only in situations in which exposure is highly likely. The duration of prophylaxis has not been determined for most agents.
[2] If susceptibility is unknown or indicates resistance to other agents. Ciprofloxacin is not licensed by the FDA for use in people younger than 18 yr but is indicated for potentially serious or life-threatening infections.
[3] Tetracyclines, including doxycycline, are not approved by the FDA for this indication and are usually contraindicated for children younger than 8 yr, but treatment is warranted for selected serious infections.
[4] Treatment should be administered parenterally initially but may be changed to oral therapy for cutaneous infection without dissemination.
[5] Amoxicillin may be used as prophylaxis only if the organism is known to be susceptible.
[6] Botulism antitoxin must be obtained from the CDC Drug Service, 404-639-3670 (weekdays, 8 am to 4:30 pm) or 404-639-2888 (weekends, nights, holidays).
[7] Isolation should be attempted only under Biosafety Level-4 conditions.
[8] Because of the risk of nosocomial transmission, the State health department and the CDC should be contacted for specific advice about management and diagnosis of suspected cases.

Table 4.4a. Diagnostic tests for anthrax

Anthrax type	Diagnostic tests
Cutaneous	Vesicular fluid and blood culture (anaerobic and aerobic)
Inhalational	Blood culture, CSF, chest radiograph, CT scan
Gastrointestinal	Blood culture

Table 4.4b. Adjunctive diagnostic tests for anthrax

Test	Comments
CBC	WBC may be normal or slightly increased. Increased neutrophils or band forms common. Leukopenia or lymphocytosis does not support diagnosis of anthrax.
Chest radiograph	Frequently abnormal in inhalational anthrax; may show signs of mediastinal widening, paratracheal or hilar fullness, pleural effusions, pulmonary infiltrates (uni- or multilobar), and mediastinal lymphandenopathy; changes may be subtle and better defined on chest CT.
Nasal swab	May assist in epidemiologic investigations but should not be relied on as a guide for prophylaxis or treatment of individual patients.

Table 4.5. Postexposure prophylaxis for anthrax

Patient	Prophylactic treatment
Adults (including pregnant women and immunocompromised people)	Ciprofloxacin 500 mg, PO, BID × 60 days ***or*** Doxycycline 100 mg, PO, BID × 60 days
Children	Ciprofloxacin 10–5 mg/kg, PO, BID × 60 days (total daily dose not to exceed 1000 mg) ***or*** Doxycycline at: >8 yr and >45 kg: 100 mg, PO, BID × 60 days >8 yr and <45 kg: 2.2 mg/kg, PO, BID × 60 days 8 yr or younger: 2.2 mg/kg, PO, BID ×60 days

Table 4.6. Treatment recommendations for tularemia in children before test results are known

Drug	Dosage
Preferred choices	
Streptomycin	15 mg/kg, IM, BID × 10 days (should not exceed 2 gram/day)
Gentamicin	2.5 mg/kg, IM or IV, TID × 10 days (not an FDA-approved use)
Alternative choices	
Doxycycline*	If weight ≥45 kg, 100 mg, IV × 14–21 days
	If weight <45 kg, give 2.2 mg/kg, IV, BID × 14–21 days
Chloramphenicol*	15 mg/kg, IV, 4 times daily × 14–21 days (not an FDA-approved use)
Ciprofloxacin*	15 mg/kg, IV, BID × 10 days (should not exceed 1 gram/day)

* Treatment with doxycycline, chloramphenicol, or ciprofloxacin given IM or IV can be switched to oral administration when clinically indicated.

Chapter 5. Chemical Terrorism

Introduction

Chemical terrorism is the intentional use of toxic chemicals to inflict mass casualties and mayhem on an unsuspecting civilian population, including children. Such an incident could potentially overwhelm the capacity of regional emergency medical services and pose extraordinary medical management challenges to pediatricians. However, careful community planning, robust research and development (by academic, private, and governmental collaborative efforts), and rigorous medical education could mitigate such a catastrophe.

The risk of chemical terrorism is more tangible since the events of September 11, 2001, and the subsequent intentional spread of anthrax through the U.S. mail. However, the specter of purposeful toxic exposures predates the September 11 attack. The 20th century witnessed Iraqi military attacks with nerve agents on civilian villages in Iran in the 1980s, the release of the nerve agent sarin in the Tokyo subway system in 1995, a chlorine bomb scare at Disneyland in 1995, and the finding of ricin in U.S. Senate office buildings in 2004.

Chemical terrorism often refers to the use of military chemical weapons that have been illicitly obtained or manufactured *de novo*. However, additional concerns might include the intentional explosion of an industrial chemical factory, a tanker car, or a transport truck in proximity to a civilian residential community, school, or worksite. These events underscore the need for all pediatricians to expand their working knowledge of the approach to mass casualty incidents involving traditional military chemical weapons and other toxic chemicals that might be used as "weapons of opportunity."

The medical consequences and epidemiology of a chemical terrorist attack mimic more conventional disasters but also reflect some distinct differences. Such an incident combines elements of both a traditional mass disaster (e.g., an earthquake) and a hazardous materials incident. Potential differences of a chemical terrorist attack compared with a "routine" hazardous materials incident include the following:

- Intent to cause mass casualties.
- Great toxicity of substances.
- Delayed initial identification of substance.
- Greater risk to first responders.
- Overwhelming numbers of patients.
- Many anxious individuals.
- Mass hysteria, panic.
- Discovery of dispersal device.

Casualties occur almost immediately, and the attack would likely be recognized rapidly. First responders are EMS, police, fire, and paramedic personnel. Decontamination and initial care of small children on-scene pose enormous management issues for personnel wearing bulky personal protective gear. In addition, many children who have been

exposed but not critically injured will be taken by parents to hospitals and pediatricians' offices without prior on-scene decontamination—thus posing similar challenges for and possibly personal risk to pediatric care providers themselves.

Specific Pediatric Vulnerabilities to Chemical Agents

Children have inherent physiologic, developmental, and psychological differences from adults that may enhance susceptibility and worsen prognosis after a chemical agent exposure (see also Chapter 1, Children Are Not Small Adults). Briefly, such physiologic differences include higher minute ventilation, increased skin permeability, relatively larger body surface area, less intravascular volume reserve in defense of hypovolemic shock, and shorter stature (which places children nearer to the greatest gas vapor density at ground level). Children who are pre-ambulatory or pre-verbal and those who have special needs are less able to evade danger or seek attention effectively. A chaotic atmosphere compounded by rescuers wearing unfamiliar garb may frighten children of all ages and potentially increase the posttraumatic response to stress. Those providing care for children are faced with additional complexities posed by developmental, age, and weight considerations beyond the general scope of the already enormous challenge.

Pediatric vulnerabilities become particularly significant when weapons of mass destruction are involved. A chemical agent will most likely be dispersed via an aerosol route or in combination with traditional warfare. Chemical exposures warrant expedient and thorough decontamination to limit continued primary and secondary exposures. Children's relatively large body surface area plays a key role in degree of contamination and in their ability to maintain thermal homeostasis after decontamination. Table 5.1 summarizes pediatric-specific vulnerabilities to chemical agents.

Chemical Injuries and Approach to the Unknown Chemical Attack

A listing of many of the most notable chemical agents of concern has been compiled by the CDC (see http://www.bt.cdc.gov/agent/agentlistchem.asp). Toxic effects from chemical agents usually follow dermal or inhalational exposure and may develop via injury to the skin, eyes, and respiratory epithelium, as well as via systemic absorption. The intensity and route of exposure to chemical agents affect both the rapidity of onset (seconds to hours) and the severity of symptoms. For example, a mild exposure to sarin vapor results in lacrimation, rhinorrhea, miosis, and slightly blurry vision; an intense exposure leads to seizures, apnea, and rapid death within minutes.

Clinical syndromes and management after exposure to various chemical agents (nerve agents, vesicants, pulmonary agents, cyanide, and riot-control agents) are summarized in Table 5.2 and detailed in the following sections. For in-depth discussions of general principles of supportive care for victims of chemical warfare agents, see Osterhoudt, et al, 2005, and Erickson, 2004.

Understanding the epidemiology of acute mass exposure to a toxin is helpful in recognizing a covert chemical attack with unknown agents. Mass exposure to a toxin will

likely manifest as an acute onset of illness (within seconds to minutes or within hours in the case of some of the vesicants and pulmonary agents). In more severe chemical incidents, numbers of people may collapse or die within minutes of exposure.

Chemical weapons can be categorized based on the predominant symptoms they cause:

- Neurologic (nerve agents or cyanide).
- Respiratory (phosgene or chlorine, high-dose riot-control agents, or sulfur mustard with a delay of several hours from time of exposure).
- Mucocutaneous syndromes (vesicants).

For additional advice on more definitive diagnosis and management strategies, contact public health authorities or the regional poison control center (1-800-222-1222).

The initial decision that will need to be made immediately will likely be the distinction of cyanide from nerve agent attack because the antidotal therapies are quite different. In both cases, large numbers of victims may suddenly collapse, have seizures, or go into a coma, and many deaths occur rapidly. Nerve agent casualties are likely to be cyanotic and have miotic pupils with altered vision, copious oral and nasal secretions, and acute bronchospasm and bronchorrhea.

The initial protection of everyone in a community exposed to a hazardous chemical requires safe evacuation or local sheltering. Circumstances may vary considerably, but it is expected that local and Federal authorities will decide and quickly advise on evacuation or local sheltering and broadcast their advice quickly and widely in the public media.

For the CDC guidelines for evacuation, see
http://www.bt.cdc.gov/planning/evacuationfacts.asp.
For the CDC guidelines for sheltering in place in a chemical emergency, see
http://www.bt.cdc.gov/planning/shelteringfacts.asp.

Initial Approach, Decontamination, and Triage

The general treatment of contaminated victims begins with extrication, triage, resuscitation as needed, and decontamination performed by rescue workers or health care providers wearing appropriate personal protective equipment (PPE). Ideally, decontamination would be done at the scene to avoid the considerable challenges posed by the arrival of contaminated patients, including children, at health care facilities. However, in a large-scale terrorist incident, it is far more likely that some victims will arrive at hospitals or other health care facilities without having been previously decontaminated. In this context, significantly contaminated victims should be decontaminated before they are allowed into the emergency department (ED). Even if decontamination has been done in the field, hospitals are likely to repeat decontamination procedures to protect the facility from contamination (which would result in closure or having to go “off line”); this would also address the possibility of cross-contamination moving from the scene. Decontamination to limit secondary exposures is especially important in exposures to nerve agents and vesicants.

Appropriate PPE for ED staff involved in patient decontamination is an important consideration. The amount of chemical agent believed to contaminate patients who arrive at the ED after a chemical terrorist attack would essentially consist of that on their skin and clothing (i.e., far lower concentration of chemicals than rescue workers would face at the scene of exposure). Most authorities believe that ED staff wearing level C PPE would be adequately protected. Level C PPE consists of a non-encapsulated, chemically resistant body suit, gloves, boots, and a PAPR mask containing a cartridge with both an organic-vapor filter for chemical gases and vapors and a HEPA filter to trap aerosols of biological and chemical agents. Such PPE is much less cumbersome to work in than level A or B outfits (which use self-contained breathing apparatus) and is also less expensive.

Cardiopulmonary and airway support, including endotracheal intubation, and emergent intramuscular antidotal therapy are provided as necessary and appropriate for the specific exposure. Contaminated clothing should be removed as soon as possible. The contamination hazard is reduced by as much as 80-90% simply by removing clothing. This is accompanied or immediately followed by more definitive decontamination. For vapor-exposed victims, decontamination may be accomplished primarily by clothing removal and washing of hair. In contrast, for victims with liquid dermal exposure, more thorough decontamination is required. Their skin and clothing pose considerable risk to ED personnel. Clothing should be carefully removed and disposed of in double bags. Victims with ocular exposure require eye irrigation with copious amounts of saline or water. Skin and hair should be washed thoroughly, but gently, with soap and tepid water. In the past, some authorities had recommended 0.5% sodium hypochlorite (dilute bleach) for skin decontamination of nerve agents and vesicants. However, this may be a skin irritant, thus increasing permeability to the agent. In addition, its use is time-consuming and has not been proven superior to washing with copious soap and water or water alone. Furthermore, there is little experience with this approach in infants and young children. A difficult question that remains is whether EMS and ED staff wearing bulky PPE will be able to provide significant advanced life support to small children before decontamination.

Ambulatory, asymptomatic victims may be able to be discharged from the scene, while those with minimal symptoms may be directed toward local shelters (e.g., American Red Cross stations, local schools, or other sites designated by local or State health departments) after decontamination for medical observation. These shelters may also serve as sites for reuniting children with their families, keeping track of all victims, and communicating with law enforcement agencies.

Industrial Chemicals

The potential of a terrorist attack on industrial sources of hazardous chemicals (e.g., factories, railroad and vehicular tank cars, or storage depots) expands the list of potential "chemical weapons" considerably. In general, many of the relevant industrial chemicals might be expected to induce respiratory effects analogous to those of chlorine or phosgene (see the section on pulmonary agents later in this chapter) or dermatologic injury from irritant or caustic properties, as well as more systemic effects in severe

exposures (Table 5.3). For an in-depth discussion of principles in managing such toxic injuries, see Osterhoudt, et al., 2005, and Erickson, 2004.

Community Preparedness

In the aftermath of September 11, 2001, many agencies are collaborating to ensure coordinated care of pediatric victims (see Chapter 2, Systems Issues). All pediatricians are encouraged to participate in disaster management training. The need to stock appropriate antidotes, practice decontamination strategies, and learn the use of PPE is apparent. Although perhaps not every practicing pediatrician needs to be competent in all aspects of disaster response, all in the community should work together to optimize the overall capacity for providing disaster care to chemically exposed children.

Successful planning and response to events involving chemical terrorism require strong collaboration and integrated functioning of many agencies and facilities, both governmental and nongovernmental, including local treatment facilities, local and State health departments, and Federal agencies (CDC, FEMA, FBI, etc.).

Nerve Agents

Nerve agents are organophosphorous compounds similar to the organophosphate insecticides used in agriculture or industry but far more toxic. Four compounds are currently regarded as nerve agents: tabun, sarin, soman, and VX ("Venom X"). All of these agents are hazardous by ingestion, inhalation, or cutaneous absorption, the latter being particularly true for VX. The toxic effects of nerve agent vapors depend on the concentration of the agent inhaled and on the time exposed to the agent. The toxicity of nerve agent liquid depends on the time exposed and the bodily site of exposure. Nerve agents exist as liquids at standard temperatures and pressures. In gaseous form, they are denser than air and vary in volatility, with some (e.g., VX) being more persistent than others (e.g., sarin).

Background

The Iran-Iraq War of the 1980s reportedly resulted in more than 100,000 casualties from chemical weapons. Iranian sources reported that the number of casualties caused by nerve agents was far greater than the number of casualties caused by mustard agent. Many nerve agent casualties that were only mildly to moderately affected were not counted.

A chemical warfare campaign by the Iraqi military on Kurdish civilians in the late 1980s caused thousands of deaths. The exact agents are not definitively known, but Iraq is known to have stockpiled tabun, sarin, and VX.

A Japanese religious cult that manufactured sarin deployed it in 1994 in attacks on a residential neighborhood of Matsumoto and again in 1995, in the Tokyo subway. Immediate mortality was low, but thousands of individuals arrived at emergency rooms. The lack of a decontamination process resulted in significant morbidity to health care personnel. The sarin was released by a relatively primitive method (punctured plastic

bags allowing sarin vapor to escape); many experts believe a more sophisticated delivery system might have resulted in far higher mortality.

Nerve agent exposures in the United States have been individual cases associated with industrial exposures.

Toxicology and Clinical Manifestations

Nerve agents inhibit the action of acetylcholinesterase at cholinergic neural synapses, where acetylcholine then accumulates markedly. The resulting cholinergic syndrome is classically divided into central, nicotinic (neuromuscular junction and sympathetic ganglia), and muscarinic (smooth muscle and exocrine gland) effects.

Clinical manifestations vary with the type of exposure. Symptoms after a vapor exposure appear suddenly with a full range of clinical effects, or there may be a partial expression of the syndrome. Symptoms after a liquid exposure may start with local sweating and then progress.

Central nervous system effects. Effects on the CNS include headache, seizures, coma, respiratory arrest, confusion, slurred speech, and respiratory depression. Although the seizures probably begin due to excess cholinergic stimulation, other effects (e.g., excitatory glutamate receptor stimulation and antagonism of inhibitory gamma-aminobutyric acid [GABA] receptors) may also play a role. Little experience with nerve agents is available to distinguish clinical effects in children from those in adults, although two cases of antichlolinesterase pesticide poisonings in children suggest a disproportionate degree of depressed sensorium and muscle weakness. Thus, children may manifest primarily central and/or neuromuscular effects after nerve agent exposure.

Autonomic nervous system effects. These include both nicotinic and muscarinic findings. Nicotinic effects on sympathetic activity can result in the following:

- Tachycardia.
- Hypertension.
- Metabolic aberrations (e.g., hyperglycemia, hypokalemia, and metabolic acidosis).

Muscarinic effects involve multiple systems:

- Ocular (miosis, eye pain, visual blurring, lacrimation).
- Respiratory (watery rhinorrhea, increased bronchial secretions and bronchospasm causing cough, wheezing, dyspnea, and cyanosis).
- Cardiovascular (bradycardia, hypotension, atrioventricular block).
- Dermal (flushing, sweating).
- Gastrointestinal (salivation, nausea, vomiting, diarrhea progressing to fecal incontinence, abdominal cramps).
- Urinary (frequency, urgency, incontinence).

Neuromuscular effects. At the neuromuscular junction, initial stimulation of cholinergic synaptic transmission is followed by paralysis. Thus, nicotinic effects include muscle fasciculations and twitching, followed by weakness progressing to flaccid paralysis and respiratory failure.

The clinical syndrome of organophosphate toxicity is summarized by various mnemonics, including "bag the puddles,"[1] "sludge" syndrome, and "dumbbels."

B = bronchoconstriction, bronchorrhea
A = apnea
G = graying/dimming of vision
P = pupillary constriction (miosis)
U = urination
D = diarrhea
D = diaphoresis
L = lacrimation
E = emesis
S = salivation, seizures

S = salivation, seizures
L = lacrimation
U = urination
D = diarrhea
G = graying/dimming of vision
E = emesis

D = diarrhea
U = urination
M = miosis
B = bronchoconstriction
B = bronchorrhea
E = emesis
L = lacrimation
S = salivation

Diagnostic Tests

The diagnosis of nerve agent toxicity is primarily based on clinical recognition and response to antidotal therapy. Measurements of acetylcholinesterase in plasma or red blood cells (RBCs) may confirm organophosphate poisoning, but correlation between cholinesterase levels and clinical toxicity is poor in some contexts; also, these analyses are rarely available on an emergent basis. RBC cholinesterase levels may help in monitoring recovery or in forensic investigations. In symptomatic patients, treatment is

[1] Adapted from Rotenberg JS, Newmark J. Nerve agent attacks on children: diagnosis and management. *Pediatrics* 2003; 112:648-58.

indicated without waiting for cholinesterase levels, while in exposed asymptomatic patients, antidotal therapy is not needed, even if cholinesterase is depressed.

Treatment

If recognized early, this is a treatable and reversible syndrome. Triage, resuscitation, and decontamination should begin at the scene and at accepting health care facilities (see Chapter 1). Individuals exposed to liquid should be observed for at least 18 hours.

Treatment focuses on airway and ventilatory support; aggressive use of antidotes, particularly atropine and pralidoxime (2-PAM); prompt control of seizures; and decontamination as necessary. Antidotal therapy is titrated according to clinical severity (Table 5.4).

Atropine, in relatively large doses, is used for its antimuscarinic effects, and pralidoxime chloride serves to reactivate acetylcholinesterase and thus enhance neuromuscular function. Atropine counters bronchospasm and increased bronchial secretions; bradycardia; GI effects of nausea, vomiting, diarrhea, and cramps; and may lessen seizure activity. Severely affected nerve agent casualties in the military have received 20-200 mg of atropine. Atropine should be administered until respiratory status improves, because tachycardia is not an absolute end-point for atropinization. Atropine cannot reverse neuromuscular symptoms, and paralysis may persist without pralidoxime.

Pralidoxime cleaves the organophosphate away from the cholinesterase, thus regenerating the intact enzyme if aging has not occurred. This effect is noted most at the neuromuscular junction, with improved muscle strength. Prompt use of pralidoxime is recommended in all serious cases.

Both atropine and pralidoxime should be administered IV in severe cases (intraosseous access is likely equivalent to IV). However, animal studies suggest that hypoxia should be corrected, if possible, before IV atropine use, to prevent arrhythmias; otherwise IM use might be preferable initially. Atropine has also been administered by the endotracheal or inhalational route in some contexts, and such use might have a beneficial effect. Experience with organophosphate pesticide poisoning in children suggests that continuous IV infusion of pralidoxime may be optimal. Nevertheless, the IM route is acceptable if IV access is not readily available. This may be of considerable relevance in a mass casualty incident involving children. In fact, most EMS programs in the United States now stock military IM auto-injector kits of atropine and 2-PAM. Similar kits with pediatric doses are currently not available in the United States. However, pediatric auto-injectors of atropine in 0.25 mg, 0.5 mg, and 1.0 mg sizes have recently been approved by the FDA. In dire circumstances, the adult 2-PAM auto-injector (600 mg) might be used in children older than 2–3 years or weighing more than 13 kg.

Seizures are primarily controlled with benzodiazepines. Diazepam is principally used by the U.S. military, but other benzodiazepines may be equally efficacious (e.g., midazolam or lorazepam). Midazolam is believed optimal for IM administration in the treatment of status epilepticus in general and so may be especially useful in nerve agent toxicity in

children. Finally, routine administration of anticonvulsant doses of benzodiazepines has been recommended in severe cases even without observed convulsive activity because animal studies have indicated some amelioration of subsequent seizures and morphologic brain damage with such use.

Supportive care is critical to patient outcome and includes the following:

- Protect airway/relieve bronchospasm/pulmonary toilet.
  - 100% oxygen, bronchodilators, nasogastric tubes.
- Monitor for cardiac arrhythmias.
- Treat complicating injuries and infections.
  - Wounds and foreign bodies may be contaminated.
  - Treat skin lesions.
- Provide fluids, electrolytes, and nutrition.
  - Nursing mothers should discard breast milk.
- Prevent hypothermia.
- Provide eye care.
  - Consider ophthalmic analgesics for ocular pain.
  - Consider topical mydriatics for miosis (atropine given systemically may not reverse miosis).
- Consider EEG and brain imaging for victims who do not promptly regain consciousness.

Isolation and Control Measures

Isolation is required only for potentially exposed victims before they are definitively decontaminated. Health care workers should wear PPE to treat victims before decontamination is complete.

Cyanide

Cyanide has long been used for sinister purposes, including as an agent of murder, suicide, chemical warfare, and judicial execution. In addition, it may pose an occupational hazard, and it has been ingested (usually in a precursor form) by children. Its efficacy as an agent of chemical terrorism is considered somewhat limited by its volatility in open air and relatively low lethality compared with nerve agents. However, if cyanide were released in a crowded, closed room, the effects could be devastating. This was more than amply illustrated by its notoriety as the chemical weapon used by the Nazis in the concentration camp gas chambers. More than 900 people ingested potassium cyanide salt in the 1978 Jonestown mass suicide incident. Chemical warfare agents involving cyanide include the liquids hydrocyanic acid (HCN, the form used by the Nazis, as “Zyclon B”) and cyanogen chloride (deployed during World War I), which rapidly vaporize after detonation. Cyanogen chloride may cause some initial eye, nose, throat, and airway irritation, but otherwise its effects are the same as those of hydrocyanic acid and result from systemic cyanide toxicity.

Toxicology

Cyanide has a strong affinity for the ferric iron (Fe^{3+}) of the heme ring and thus inhibits many heme-containing enzymes. Its primary effect in acute toxicity is inhibition of cytochrome a_3, thereby interfering with normal mitochondrial oxidative metabolism in the electron transport chain, causing cellular anoxia and lactic acidosis. It may also interfere with other important enzymes, including succinic acid dehydrogenase and superoxide dismutase, which may underlie some of its chronic toxicity. In addition, cyanide is believed to be a direct neurotoxin contributing to an excitatory injury in the brain, probably mediated by glutamate stimulation of N-methyl D-aspartate receptors. The primary human enzyme, rhodanese, detoxifies cyanide by combining it with a sulfate moiety such as thiosulfate to form the relatively nontoxic thiocyanate ion, which is then excreted by the kidneys. Therefore, exposure to a potentially lethal dose of cyanide that occurs slowly though continually over time may be tolerated, making it relatively unique among the agents of chemical terrorism.

Clinical Presentation

Clinical manifestations of cyanide toxicity vary considerably depending on dose, route of exposure, and acuteness of exposure but in general reflect the effects of cellular anoxia on organ systems. Thus, the most metabolically active tissues, the brain and heart, tend to be the most affected. With exposure to low concentrations of vapor, early findings include tachypnea and hyperpnea, tachycardia, flushing, dizziness, headache, diaphoresis, nausea, and vomiting. As exposure continues, symptoms may progress to those associated with exposures to high concentrations of vapor. The latter include rapid onset (within 15 seconds) of tachypnea and hyperpnea, followed by seizures (30 seconds), coma and apnea (2-4 minutes), and cardiac arrest (4-8 minutes). “Classical” signs of cyanide poisoning include severe dyspnea without cyanosis—or even with cherry-red skin (due to lack of peripheral oxygen use)—and a bitter almond odor to breath and body fluids. However, some patients do develop cyanosis (likely secondary to shock), and only about half the population is genetically capable of detecting the cyanide-induced bitter almond odor. Laboratory abnormalities in cyanide poisoning include metabolic acidosis with a high anion gap and increased serum lactate and an abnormally high mixed venous oxygen saturation (also due to decreased use of peripheral oxygen). Blood cyanide levels can be determined but not usually on an emergent basis.

In an aerosol attack using recognized military chemical weapons, if people are convulsing or dying within minutes of exposure, the weapon is likely to be either cyanide or a nerve agent. Although the symptoms of exposure to cyanide and nerve agents may be hard to distinguish, when there are high concentrations of cyanide, seizures begin within seconds and death within minutes, generally with little cyanosis or other findings. The course for lethal nerve agent toxicity is characteristically somewhat longer and accompanied by copious nasal secretions, miotic pupils, muscle fasciculations, and cyanosis before death.

Treatment

Management of cyanide poisoning begins with removing the victim from the contaminated environment to fresh air. Dermal decontamination is rarely necessary because these agents are so volatile but in case of contact with liquid agent, wet clothing should be removed and underlying skin washed. Ingested cyanide may be partially bound by activated charcoal.

Basic supportive intensive care is critical, including providing 100% oxygen, mechanical ventilation as needed, and circulatory support with crystalloid and vasopressors; correcting metabolic acidosis with IV sodium bicarbonate; and controlling seizures with benzodiazepines. Symptomatic patients, especially those who have lost consciousness or have other severe manifestations, may benefit further from antidotal therapy, which is a multistep process.

First, a methemoglobin-forming agent is administered, typically inhaled amyl nitrite or IV sodium nitrite because methemoglobin has a high affinity for cyanide and disassociates it from cytochrome oxidase. However, nitrite administration can be hazardous because it may cause hypotension, and overproduction of methemoglobin may compromise oxygen-carrying capacity. Thus, nitrite is probably not indicated for mild symptoms or if the diagnosis of cyanide poisoning is uncertain. Furthermore, people with cyanide poisoning who may have concomitant hypoxic insult (e.g., most victims of smoke inhalation) probably are not good candidates for nitrite therapy. Optimal nitrite dosing, especially when given parenterally, depends on body weight and hemoglobin concentration, which is of particular importance in pediatric patients, who have a broad range of hemoglobin concentrations. In the pre-hospital setting, or whenever IV access is not possible, amyl nitrite may be used to begin nitrite therapy. Amyl nitrite is provided in glass pearls, which are used by crushing the pearl and then either allowing spontaneous inhalation or introducing the vapor into a ventilation circuit, for 30 seconds of each minute. As soon as IV access is established, sodium nitrite may be given. The recommended pediatric dosage, assuming a hemoglobin concentration of 12 g/dL, is 0.33 mL (of the standard 3% solution)/kg, given slowly IV over 5-10 minutes (with a maximal, or adult, dose of 10 mL). Dosing may be adjusted for patients with significant anemia, although this would not likely be known in emergent treatment of a poisoned child in critical condition.

The second step is providing a sulfur donor, typically sodium thiosulfate, which is used as substrate by the rhodanese enzyme for conversion to thiocyanate. Thiosulfate treatment itself is believed efficacious and relatively benign, and thus it may be used alone empirically in cases in which the diagnosis is uncertain. (This approach has also been recommended, for example, in the management of the situation described above of cyanide toxicity complicating smoke inhalation, with likely concomitant lung injury and carbon monoxide poisoning). The recommended pediatric dosage of thiosulfate is 1.65 mL (of the standard 25% solution)/kg, IV (with a maximal, or adult, dose of 50 mL).

Each agent may be given a second time at up to half the original dose as needed, or in the case of thiosulfate, even a full dose would be unlikely to pose inherent toxicity. Both

these medications are packaged together in commercially available "cyanide antidote kits," along with amyl nitrite pearls. Additionally, most hospital pharmacies stock 25% sodium thiosulfate solution in vials containing sufficient volume (50 mL) to treat even adult patients. This has been used routinely in the preparation of nitroprusside infusions, premixed with thiosulfate, so as to obviate nitroprusside-induced cyanide toxicity.

Several alternative therapies and experimental antidotes have been used in Europe (cobalt salts, hydroxocobalamin) or are in clinical trials (aldehydes, aminophenol derivatives) or animal studies (dihydroxyacetone, alpha-ketoglutarate). Hydroxocobalamin, in particular, has been cited as a potentially quite useful antidote in civilian terrorism scenarios because of its relative safety compared with nitrites. However, it currently is not commercially available in the United States in a pharmacologically appropriate concentration for antidotal efficacy.

Vesicants

The term "vesicant" is commonly applied to chemical agents that cause blistering of the skin. Direct contact with these agents can also result in damage to the eyes and respiratory system. Systemic absorption may affect the GI, hematologic, and central nervous systems as well.

The four compounds historically included in this category—sulfur mustard, the nitrogen mustards, lewisite, and phosgene oxime—were all manufactured initially as potential chemical warfare agents. Phosgene oxime is technically not a true vesicant because the skin lesions it causes are urticarial as opposed to vesicular. The nitrogen mustards, although first synthesized in the 1930s for anticipated battlefield use, were found to be less effective for chemical warfare than the already existing sulfur mustard. Subsequent development for of nitrogen mustards for weapons use was therefore largely abandoned. However, one form of nitrogen mustard, HN2, became a highly used and effective chemotherapeutic agent. Lewisite was first synthesized during the latter part of World War I, but other than reports of its use by Japan against China between 1937 and 1944, it is not known to have ever been used on the battlefield. An antidote, British antilewisite (BAL, or dimercaprol), can minimize its effects if given promptly. Because so little is known about the toxicity and mechanisms of action of phosgene oxime and lewisite, and because anticipated medical management issues of these agents are somewhat similar, the following section focuses on the clinical effects and management issues regarding sulfur mustard exposure—historically the most frequently used and available of this class of chemical agent.

Sulfur mustard has been the most widely used of all chemical warfare agents over the last century. Approximately 80% of chemical casualties in World War I were due to sulfur mustard, and its use has been verified in multiple military conflicts since then. In addition, Iraq used sulfur mustard on numerous occasions during its war against Iran from 1980 to1988 and as a weapon of terror against thousands of Kurdish civilians, including children, by using aerially dispersed mustard bombs in 1988.

Sulfur mustard is stockpiled both in the United States and in several other countries as well. It is not difficult to manufacture, making it even more favorable for use by terrorists. In addition to its accessibility and ease of production, several other factors enhance its suitability as a terrorist or warfare agent. Although mortality associated with sulfur mustard is considerably lower than that caused by other chemical weapons such as nerve agents, sulfur mustard exposure results in significant and prolonged morbidity that may potentially overwhelm health care resources. The risk of direct contamination either from patient contact or from the agent's persistence in the environment may force health care providers to wear bulky protective gear, which makes it difficult to administer care, particularly to children. Although tissue damage occurs within minutes of exposure, clinical symptoms are delayed for hours, potentially rendering the victim ignorant of exposure until the opportunity for effective decontamination has passed. Lastly, unlike the case for lewisite, there is no known antidote for sulfur mustard exposure.

Characteristics

Sulfur mustard is an alkylating agent that is highly toxic to rapidly reproducing and poorly differentiated cells. Under normal environmental conditions, it is an oily liquid that varies in color from yellow to brown, depending on amounts and types of impurities. Its odor has been described as similar to garlic or to mustard itself. In warmer climates, mustard vapor is a particular concern due to its low volatility, while at lower temperatures (<14°C or 58°F); it becomes a solid and may persist in the environment for an extended time. On contact with tissue surfaces, mustard vapor or liquid is rapidly absorbed and exerts its cellular damage within minutes. Both vapor and liquid readily penetrate most clothing, although rubber overgarments may be protective for several hours.

Clinical Effects

After exposure to sulfur mustard, skin findings do not appear for 2–48 hours, depending on the mode of exposure, the sensitivity of the individual, and the environmental conditions (Table 5.5). The most common early sign in exposed areas is erythema resembling sunburn, which may coincide or even be preceded by significant pruritus. If the exposure is mild, this may be the only skin manifestation. More typically, yellowish blisters begin to form over the next 24 hours. Penetration of the agent is enhanced by thin skin, warmth, and surface moisture, rendering areas such as the groin, axillae, and neck particularly susceptible. Once they appear, the vesicles frequently coalesce to form bullae. Although largely painless, these fragile bullae commonly rupture, resulting in painful ulcers that may take weeks or months to heal. The fluid from the blisters does not contain free mustard and is therefore not hazardous. If skin exposure has been severe, these earlier stages of developing lesions may be bypassed altogether with the direct appearance—albeit delayed—of skin sloughing similar to that seen in a full-thickness thermal burn.

Although skin findings may be dramatic, the organ most sensitive to mustard exposure is the eye, with mild symptoms occurring at concentrations 10-fold lower than those needed to produce effects on the skin. Like the skin findings, ocular symptoms are also delayed,

usually for 4–6 hours. The first symptoms are usually pain and irritation, followed progressively by photophobia, worsening conjunctivitis, corneal ulceration, and perforation of the globe with severe exposures. Although visual impairment is common, it is usually transient and simply reflects eye closure from intense pain and reflex blepharospasm as opposed to true damage to the optic nerve. Severe lid edema caused by inflammation of soft tissue around the eyes is also common.

With inhalation of mustard vapor, both the proximal and distal respiratory tract may be affected. Proximal involvement usually manifests after several hours and consists of rhinorrhea, hoarseness, a dry and painful cough with expectoration, and eventually a characteristic toneless voice due to vocal cord damage. With more significant inhalational exposures, necrosis of the airway mucosa can lead to a sterile tracheobronchitis with the necrotic epithelium forming pseudomembranes that may obstruct the airway. Bacterial superinfection may develop as well, usually days later, facilitated by a weakened immune response. Respiratory failure can be the end result of either early mechanical obstruction from laryngospasm or pseudomembrane formation or later by overwhelming bacterial infection enhanced by the denuded respiratory mucosa and necrotic tissue. Early onset of dyspnea, along with other signs of impaired peripheral gas exchange, such as hypoxia, is a sign of severe inhalational exposure and indicates a poor prognosis.

All cellular elements of the bone marrow can be affected by sulfur mustard due to its DNA alkylating effects, which impair replication in rapidly dividing stem cells. Megakaryocytes and granulocyte precursors are more susceptible than those of the erythropoietic system, and therefore the presence of anemia along with leukopenia indicates significant exposure and a poorer outcome. During the first few days after exposure, there may be a reactive leukocytosis that may or may not progress to leukopenia, depending on the level of exposure.

Gastrointestinal symptoms can develop from the general cholinergic activity of sulfur mustard, resulting in nausea and vomiting that occurs after several hours and is rarely severe. Direct injury to the GI mucosa from ingestion of mustard either directly or from contaminated food or water can lead to a later onset of more severe vomiting, diarrhea, abdominal pain, and prostration.

Although historically a large percentage of battlefield victims have reported CNS findings such as lethargy, headaches, malaise, and depression, the role of the mustard agent itself in development of symptoms, as opposed to that of other environmental stressors, is unclear. Clinicians should be aware that, regardless of their etiology, these symptoms are a frequent presentation. In addition, absorption of high doses of sulfur mustard can result in CNS hyperexcitability, convulsions, abnormal muscular activity, and coma.

Treatment

The most effective treatment is decontamination, because once sulfur mustard penetrates tissues, its effects are irreversible. Unfortunately, sulfur mustard is rapidly absorbed on contact, usually exerting damage within 3–10 minutes of exposure. Effectiveness of

decontamination is therefore extremely time dependent. Self-decontamination may be the quickest method and should include removing clothing and physically eliminating any mustard residue on the skin.

Anyone providing aid to an exposed person should take proper precautions including ocular, respiratory, and skin protection, ideally with a chemical protection overgarment, rubber boots, and gloves. Exposed individuals should be washed with soap and warm water, or just rinsed with water, as soon as possible. The use of bleach is not recommended in children because it can cause liquefactive epithelial damage to their thin skin, which may in fact promote further penetration of the agent.

Other methods of physical removal, particularly if the mustard is predominately in solid form, include scraping or plucking the agent from the skin, as well as using adsorptive agents such as earth, powdered soap, or flour, followed by rinsing with water.

Regardless of decontamination method, the most important aspect is speed. While ideally all victims should be decontaminated before entering a medical treatment facility, if exposed individual arrive via personal transportation or on foot, they may first need to be taken to a separate area for decontamination. Even if delayed, decontamination should be done to protect others from exposure, to avoid further absorption, and to prevent spread to other areas of the body.

After decontamination and basic life-support issues and other life-threatening concomitant injuries have been addressed, it is important to remain aware of the latency of most symptoms of vesicant exposure. Even if no symptoms are seen at presentation, exposed patients should be observed for at least 8 hours before being discharged. Because of the lack of a specific antidote, the remainder of therapy is supportive.

Skin lesions are treated similarly to those of burn victims. However, fluid losses tend to be less. For this reason, traditional formulas for fluid replacement in burn victims often overestimate losses in vesicant-exposed patients. Erythema and symptoms such as pruritus should be treated with topical and systemic analgesia and antipruritics, as well as soothing lotions such as calamine. Small vesicles (<2 cm) should be left intact, but larger vesicles and bullae should be incised and treated with frequent irrigation and topical antibiotics such as silver sulfadiazine. Widespread and severe partial or full-thickness involvement should be managed in a burn unit if possible.

Eye treatment should center on removing the agent and on preventing scarring and infection. After irrigation of the eye with copious amounts of water, cyclopegic agents should be applied for comfort and to prevent formation of synechiae. Topical antibiotics should then be applied directly along with lubricating ointments such as petroleum jelly to the eyelids to prevent adhesions and subsequent scarring.

Mild respiratory symptoms involving the upper airway can be treated with cough suppressants, throat lozenges, and cool mist vapor. More severe lower respiratory involvement generally requires ventilation with positive end-expiratory pressure. The

patient should be intubated promptly if there are any signs of laryngeal spasm or edema. Direct bronschoscopy may be necessary for removal of obstructive pseudomembranes. The need for prolonged intubation (>5–10 days) is a sign of significant proximal airway damage and suggests a poor prognosis. The temptation to use systemic antibiotics during the first 3–4 days despite the not uncommon findings of fever, leukocytosis, and cough should be avoided to prevent the growth of resistant organisms. However, if these signs and symptoms persist beyond this period and there is radiographic evidence of consolidation, systemic antibiotics may then be indicated.

For severe GI effects, in addition to fluid replacement, antiemetics or anticholinergics may be helpful. In the rare case of vesicant ingestion, gastric lavage may be useful if performed within 30 minutes; vomiting should never be induced.

If anemia from bone marrow involvement is severe, blood transfusions may be of benefit. Other therapies, such as administration of hematopoietic growth factors and bone marrow transplantation, although used successfully in animal studies, have never been used in people exposed to vesicants.

Pediatric Considerations

The unique susceptibilities of children (see Chapter 1, Children Are Not Small Adults) emphasize the need to consider a number of practical treatment issues after vesicant exposure. The first consideration is the time from exposure to onset of skin manifestations, which is shorter in children than in adults. As a result, children may be overrepresented in the initial index cases in a mass civilian exposure. Because a child's skin is more delicate, the caustic effects of decontamination agents (such as bleach) on an already damaged skin surface are potentially much greater; consequently, these agents should probably be avoided altogether in children. Soap and water used for washing and rinsing should be warmed if possible to prevent the greater likelihood of hypothermia in children. In addition, low water pressure (60 psi preferred; if not available, <100 psi) should be used if possible to minimize potential further penetration of the agent into the thinner skin of the child.

Because mustard vapor is denser than air, it tends to settle close to the ground, which has obvious ramifications for small children. In addition to more common facial and eye involvement, pulmonary involvement can also be more extensive, ostensibly from the lower breathing zones and increased respiratory rates of children. Therefore, intubation may be needed earlier, and more aggressive ventilatory management may be necessary in the vapor-exposed child who has lower respiratory tract symptoms.

Fluid replacement may also need to be more aggressive in children because of the greater potential for dehydration secondary to their lower volume reserve. Pain management is another important consideration, particularly in the very young child who is preverbal.

Pulmonary Agents

Toxic industrial chemicals used as terrorist weapons are a potentially significant threat to civilian populations. The Chemical Weapons Convention, a disarmament and nonproliferation treaty with 145 signatory countries, identifies 33 chemical and chemical precursors that can be used as weapons. Although some of the chemicals are well-known weapons (e.g., sarin, VX, sulfur mustard), others are more familiar as common industrial chemicals such as chlorine, phosgene, and others. In the United States today, millions of tons of these chemicals are manufactured yearly for the production of dyes, textiles, medicines, insecticides, solvents, paints, and plastics.

The potential terrorist threat posed by industrial chemicals is well known. A January 2002 report to Congress by the Central Intelligence Agency reports that terrorist groups "have expressed interest in many other toxic industrial chemicals—most of which are relatively easy to acquire and handle—and traditional chemical agents, including chlorine and phosgene." Although of clear interest to terrorist groups, traditional nerve agents require a greater degree of technical sophistication to manufacture and deliver as weapons.

Chlorine and Phosgene

As chemical weapons, chlorine and phosgene are commonly known as "pulmonary," "inhalational," or "choking" agents. These terms are ambiguous to the point of confusion, and it is better to conceptualize these compounds along a spectrum. Type I agents act primarily on the central, or tracheobronchial, components of the respiratory tract; Type II agents act primarily on the peripheral, or gas-exchange, regions (i.e., the respiratory bronchioles, alveolar ducts, and alveoli). Type I agents are typically water soluble and chemically reactive and attack the respiratory epithelium of the bronchi and larger bronchioles. The resultant pathologic effects are necrosis and denudation with or without the formation of pseudomembranes; the resultant clinical effects are mucosal irritation, with prominent components of noise (coughing, sneezing, hoarseness, inspiratory stridor, and wheezing). Type II agents cause noncardiogenic pulmonary edema initially manifested clinically by dyspnea without accompanying signs of either radiologic or laboratory anomalies.

Few agents are pure Type I or Type II agents, and high doses of either kind of agent can affect both central and peripheral compartments. For example, chlorine, which is intermediate in both aqueous solubility and chemical reactivity, typically produces a mix of both central and peripheral effects. Phosgene, however, has few Type I effects except at moderately high doses. Sulfur mustard has poor aqueous solubility, but once dissolved, it cyclizes to form such a powerfully reactive cyclic ethylene sulfonium oxide that it acts in the airways primarily as a Type I agent at low to moderate doses. Used as weapons of mass destruction, agents such as hydrogen cyanide and hydrogen sulfide would most likely be released as vapors or gases and, in that respect, would be "inhalational" agents. However, in contrast to Type I and Type II agents—the major pathologic and clinical effects of which are local on respiratory epithelium or alveolar septae—cyanides are widely distributed via the blood throughout the body and therefore merit a separate

classification as Type III (systemically distributed) agents. Finally, some agents such as sulfur mustard exhibit both local (in this case, initially Type I) and systemic (Type III) effects, although the systemic effects of mustard (which may include bone-marrow depression and resulting pancytopenia) become clinically significant only after a delay.

Although not stockpiled in the United States for military purposes, chlorine, phosgene, and hydrogen cyanide are common components in industrial manufacturing. Primarily liquids, they are easily vaporized, allowing for widespread gaseous dispersion.

Clinical Effects

The significant morbidity from pulmonary agents is caused by pulmonary edema. With chlorine, edema may appear within 2–4 hours or even sooner with more significant exposures. Radiologic signs lag behind clinical symptoms: pulmonary interstitial fluid must be increased 5- to 6-fold to produce Kerley B lines on a chest radiograph. Pulmonary edema may be exceptionally profuse; in a study from the 1940s, pulmonary sequestration of plasma-derived fluid could reach volumes of up to 1 L/hr. This problem may be exceptionally profound in children, who have less fluid reserve and are at increased risk of rapid dehydration or frank shock with the pulmonary edema. Additionally, because children have a faster respiratory rate, there is exposure to a relatively higher toxic dose.

Chlorine. Chlorine is a greenish yellow gas that is denser than air and, therefore, settles closer to the ground and low-lying areas. This may have significant consequences for small children and infants, who would be exposed to higher concentrations of the vapor and thus receive higher inhaled doses of the agent. Chlorine has a strong, pungent odor that most people associate with swimming pools. Because the odor threshold (at 0.08 ppm) is less than the toxicity threshold, the odor may warn individuals that exposure is occurring.

The initial complaints in chlorine exposure may be either intense irritation or the sensation of suffocation, or both; the suffocating feeling is what led to its characterization as a “choking” agent. Low-level exposures to chlorine result in mucosal irritation of the eyes, nose, and upper airways. Higher doses lead to respiratory symptoms that progress from choking and coughing to hoarseness, aphonia, and stridor—classically Type I effects. Dyspnea after chlorine exposures indicates damage to the peripheral compartment (Type II insult) and incipient pulmonary edema.

Phosgene. Like chlorine, phosgene is also heavier than air, thus posing an increased risk for children who are exposed. Phosgene itself is colorless, but associated condensation of atmospheric water produces a dense white cloud that settles low to the ground. It has the characteristic odor of newly mown hay. However, the odor threshold for phosgene (at 1.5 ppm) is higher than the toxicity threshold, and unlike the case with chlorine, detection of the odor would be inadequate and too late to serve as a warning against toxic exposure.

Phosgene is primarily associated with the development of pulmonary edema. However, because in low to moderate doses it does not cause the mucosal irritation associated with

Type I agents, the significance of the exposure may be underestimated. Exposure to progressively higher doses produces mild cough, sneezing, and other effects on the central compartment. Dyspnea is seldom present initially except when doses have been massive; instead, there is a clinically asymptomatic, or latent, period usually of several hours and inversely correlated with dose. Dyspnea and associated clinical deterioration have in several instances been triggered by slight to moderate exertion.

Treatment

Decontamination. Decontamination consists primarily of removing the victim from the source of the pulmonary agent to fresh air. For first responders such as paramedics and fire-rescue workers, PPE with self-contained breathing apparatus is required; however, because the gases are volatile, cross-contamination is unlikely. Victims of chlorine exposure may require copious water irrigation of the skin, eyes, and mucosal membranes to prevent continued irritation and injury.

Management. Management is primarily supportive; there are no antidotes or specific postexposure treatments for inhalational agents. Victims should be observed and monitored for both central (Type I) and peripheral (Type II) acute effects, including development of pulmonary edema. Most deaths are due to respiratory failure and usually occur within the first 24 hours. Because of the delay in onset of pulmonary edema, prolonged observation of victims of phosgene and chlorine attacks is warranted.

Treatment of central, or Type I, damage involves administering warm, moist air and supplemental oxygen, and treating bronchospasm either produced *de novo* by the toxicant in normal airways or resulting from toxicant-induced exacerbation of airway hyperresponsiveness in individuals with underlying pathology such as asthma or reactive airways. Aggressive bronchodilator therapy with beta-agonists is appropriate. The value of corticosteroids is less clear, but they may be efficacious in victims with severe bronchospasm or a history of asthma. Nebulized lidocaine (4% topical solution) has been recommended to provide analgesia and reduce coughing. The possibility of laryngospasm should always be anticipated and the necessity and timing of intubation carefully assessed. Associated central damage from inhaled particles of smoke in situations involving fire should also be considered. Pseudomembrane formation may lead to airway obstruction and may require bronchoscopic identification and removal of pseudomembranous debris. Necrotic debris from central damage provides an excellent culture medium for secondary bacterial colonization and infection, and bacterial superinfections are commonly seen 3–5 days after exposure. Early aggressive antibiotic therapy directed against culture-identified organisms is imperative. Prophylactic antibiotics are of no value.

Treatment of peripheral (Type II) damage from pulmonary agents includes adequate oxygenation, establishment of effective intra-alveolar pressure gradients using positive end-expiratory pressure (for example, in conscious patients, with continuous positive airway pressure, or CPAP), and careful attention to fluid balance. In cases of florid pulmonary edema, using a central line to monitor hemodynamics in critically ill children may be necessary. The length of the latent period in a dyspneic patient can provide

clinically valuable information about the intensity of exposure; patients who develop breathing difficulty within the first 4 hours after exposure may face a grave prognosis, and even patients with mild dyspnea, because of the timing of the dyspnea, may be candidates for urgent or priority evacuation. All patients at risk of pulmonary edema induced by pulmonary agents should be maintained on strict bed rest to avoid cardiopulmonary decompensation associated with exertion.

Riot Control Agents

Modern riot control agents comprise a heterogeneous group of chemical compounds that have been used widely around the world since the 1950s (Table 5.6). These agents have the ability to incapacitate at low aerosol concentrations and have a high safety ratio (ratio of lethal dose to effective dose). However, prolonged exposure or release in enclosed areas can intensify the physical effects of these agents. CS (2-chlorobenzylidene), CN (1-chloroacetophenone, Mace®), and pepper spray (*Oleoresin capsicum*) are commercially available to the public in the United States.

Transmission and Pathogenesis

Mode of transmission varies by agent. Common means include spraying a solution, release of pressurized canisters, explosive dispersion (smoke "grenades"), and burning. Explosive modes of transmission may cause traumatic injuries in addition to the incapacitating effects. CS is very flammable and poses a fire hazard. Most agents disperse soon after release, although persistent forms of CS exist. Riot control agents may contaminate clothing, buildings, and furniture and may cause ongoing symptoms in continued or repeat exposure.

When dispersed, riot control agents are chemical irritants of the skin and mucous membranes of the eyes, nose, mouth, airways, and GI tract. CS and CN incapacitate through direct chemical irritation, acting like chemical thorns. Due to its low vapor pressure, CR (dibenzoxazepine) has limited effects on the respiratory tract. In addition to direct irritation, pepper spray also induces local release of the neurotransmitter substance P in peripheral afferent sensory nerves. This mechanism causes pain, capillary leakage, and vasodilation.

Clinical Manifestations

Riot control agents have specific effects on the eyes, nose, mouth, and airway, with variation in intensity depending on mode of exposure and agent used. Symptoms occur quickly after exposure and typically resolve in 1–2 hours once the victim has been removed from the agent. On contact, these agents induce eye burning, eye pain, tearing, conjunctival infection, blepharospasm, periorbital edema, and photophobia. Exposures at close range, particularly to exploding CS and CN grenades or canisters, may cause serious damage to the eye including corneal edema, conjunctival laceration, hyphema, vitreous hemorrhage, and secondary glaucoma. Permanent effects such as cataracts and traumatic optic neuropathy may also be seen.

After dispersal of riot control agents, nasal burning and pain, copious rhinorrhea, and persistent sneezing begin along with oral irritation and salivation. Pulmonary effects include chest tightness and burning, bronchorrhea, bronchospasm, and coughing. Gagging, retching, and vomiting frequently accompany mucosal and airway irritation. Exposed skin stings and may progress to erythema, vesiculation, and bullae depending on the conditions of exposure; prolonged exposure, high ambient temperature, and humidity favor worsening skin effects. These manifestations may occur hours to days after exposure to CS. Skin exposed to CR may become painful in water for up to 2 days after exposure. CN and CS can cause allergic contact dermatitis in people who are repeatedly exposed.

Severe clinical effects from riot control agents are uncommon. Intense exposure to CS, CN, and pepper spray has caused laryngospasm, pneumonitis, bronchospasm, and noncardiogenic pulmonary edema. Often, the agent was released in an enclosed space, or the victim was not able to leave the vicinity of the agent. Individuals with asthma are predisposed to serious pulmonary symptoms. Experience with a 4-week-old infant who was unintentionally exposed to pepper spray at close range suggests that severe lung injury from *Oleoresin capsicum* is reversible in young children, provided that intensive medical support is provided. Deaths caused by pulmonary effects have occurred after CN exposure in victims who had previously normal lung function. Pepper spray was implicated in the death of one asthmatic prisoner in custody. Prolonged reactive airway disease has also been described after CS exposure in a previously healthy person with no prior history of atopy. In general, riot control agents are incapacitating but rarely lethal, especially relative to other deployable chemical agents such as the nerve agents, vesicants, and pulmonary agents.

Diagnosis

Some physical characteristics of the compounds can assist in detection when riot control agents are used. The most common agents (CS, CN, and pepper spray) are deployed in identifiable canisters. CS and pepper spray have a pungent pepper odor. CN has a flowery apple odor. Pepper spray frequently contains fluorescein dye that becomes readily apparent on exposed skin under a Wood's lamp. No environmental monitoring systems currently exist for riot control agents.

Differentiation of clinical effects caused by riot control agents from those of other chemicals can be a challenge during early management. Tearing, salivation, bronchorrhea, bronchospasm, and vomiting suggest the cholinergic effects of nerve agent exposure. Intense exposure to riot control agents with pneumonitis and pulmonary edema mimic symptoms of exposure to pulmonary agents, such as chlorine and phosgene. The potential for delayed skin effects, including vesiculation and bullae, with riot control agents makes them similar to vesicants such as sulfur mustard. However, symptoms rapidly resolve once contact with the agent ceases. Lack of progression to more severe symptoms such as bone marrow failure, paralysis, and seizures, combined with negative results from field detection systems and the physical characteristics mentioned above make identification of riot control agent release ultimately possible.

Treatment and Control

Decontamination requires that all victims be moved to a well-ventilated, uncontaminated space and have their outer clothing removed. Clothing should be double bagged to prevent secondary exposure. Medical treatment of riot control agent exposure focuses on ending contact, assessing for serious pulmonary effects, and addressing ongoing eye and skin irritation (Table 5.7).

In most instances, clinical signs and symptoms resolve over 30–60 minutes, and specific medical treatment is not needed. Pulmonary effects may be delayed. Victims who exhibit prolonged dyspnea or have other objective lung findings should be admitted to a medical facility for ongoing monitoring and treatment.

All first responders should wear PPE including, but not limited to, a full-face gas mask, properly rated outer clothing, gloves, and boots. Field incident command should identify a hot zone, decontamination area, and cold zone. Ideally, decontamination should begin in the field and be complete before entry into a medical facility.

Bibliography

Introduction

- American Academy of Pediatrics: Chemical and biological terrorism and its impact on children; a subject review. *Pediatrics.* 2000;105:662–70.
- Brennan RJ, Waeckerle JF, Sharp T, et al. Chemical warfare agents: emergency medical and emergency public health issues. *Ann Emerg Med.* 1999;34:191–204.
- Centers for Disease Control and Prevention. Emergency Preparedness and Response. Chemical Agents. Available at: www.bt.cdc.gov/agent/agentlistchem.asp. Accessed July 20, 2006.
- Erickson TB. Toxicology: ingestions and smoke inhalation. In: Gausche-Hill M, Fuchs S, Yamamoto L (eds). APLS *The Pediatric Emergency Medicine Resource,* 4th ed. American Academy of Pediatrics and American College of Emergency Medicine. Subury, MA: Jones and Bartlett; 2004. p. 234–267.
- Henretig FM, Cieslak TJ, Madsen JM, et al. The emergency department response to incidents of biological and chemical terrorism. In: Fleisher GF, Ludwig S, Henretig FM, ed. *Textbook of Pediatric Emergency Medicine,* 5th ed. Philadelphia: Lippincott Williams & Wilkins; 2005.
- Henretig FM. Biological and chemical terrorism defense: a view from the "front lines" of public health. *Am J Public Health.* 2001;91:718–720.
- Henretig FM, Cieslak TJ, Eitzen EM Jr. Biological and chemical terrorism. *J Pediatr.* 2002;141:311–326.
- Henretig FM, McKee MR. Preparedness for acts of nuclear, biological and chemical terrorism. In: Gausche-Hill M, Fuchs S, Yamamoto L (eds). APLS *The Pediatric Emergency Medicine Resource*, 4th ed. American Academy of Pediatrics and American College of Emergency Medicine. Sudbury MA: Jones and Bartlett; 2004. p. 568–591.

- Kales SN, Christiani DC. Acute chemical emergencies. *N Engl J Med*. 2004;350:800–808.
- Macintyre AG, Christopher GW, Eitzen E Jr, et al. Weapons of mass destruction events with contaminated casualties: effective planning for health care facilities. *JAMA*. 2000;283:242–249.
- Okumura T, Takasu N, Ishimatasu S, et al. Report on 640 victims of the Tokyo subway sarin attack. *Ann Emerg Med.* 1996;28: p.129–135.
- Osterhoudt KC, Ewald, MB, Shannon M, Henretig FM. Toxicologic emergencies. In: Fleisher GF, Ludwig S, Henretig FM (eds). *Textbook of Pediatric Emergency Medicine*, 5th ed. Philadelphia: Lippincott Williams & Wilkins; 2005.
- U.S.Army Medical Research Institute of Chemical Defense. *Medical Management of Chemical Casualties*, 3rd ed. Aberdeen Proving Ground, MD: US Army Medical Research Institute of Chemical Defense; 1999.

Nerve Agents

- Amitai Y, Almog S, Singer R, et al. Atropine poisoning in children during the Persian Gulf crisis. A national survey in Israel. *JAMA.* 1992;268:630–632.
- Holstege CP, Kirk M, Sidell FR. Chemical warfare: nerve agent poisoning. *Crit Care Clin* 1997;13:923–942.
- Lee EC. Clinical manifestations of sarin nerve gas exposure. *JAMA.* 2003; 290:659–662.
- McDonough JH Jr, Shih TM. Neuropharmacological mechanisms of nerve agent-induced seizure and neuropathology. *Neurosci Behav Rev.* 1997;15:559–579.
- Okumura T, Takasu N, Ishimatsu S, et al. Report on 640 victims of the Tokyo subway sarin attack. *Ann Emerg Med*. 1996;28:129–135.
- Rotenberg JS, Newmark J. Nerve agent attacks on children: diagnosis and management. *Pediatrics*. 2003; 112:648–658.
- Sidell FR, Borak J. Chemical warfare agents: II. nerve agents. *Ann Emerg Med.* 1992;21:865–871.
- Sofer S, Tal A, Shahak E. Carbamate and organophosphate poisoning in early childhood. *Pediatr Emerg Care.* 1989;5:222–225.
- US Army Medical Research Institute of Chemical Defense. *Medical Management of Chemical Casualties-Handbook*. Aberdeen Proving Grounds, MD: US Army Medical Research Institute of Chemical Defense; 1999.

Cyanide

- Berlin CM. The treatment of cyanide poisoning in children. *Pediatrics.* 1970;46:793–796.
- Beasley DMG, Glass WI. Cyanide poisoning: pathophysiology and treatment recommendations. *Occup Med.* 1998;48:427–431.
- Sauer SW, Keim ME. Hydroxocobalamin: improved public health readiness for cyanide disasters. *Ann Emerg Med*. 2001;37;635–641.
- Kerns W II, Isom G, Kirk MA. Cyanide and Hydrogen Sulfide. In: Goldfrank LR, Flomenbaum NE, Lewin NA, Howland MA, Hoffman RS, Nelson LS (eds):

Goldfrank's Toxicologic Emergencies, 7th ed. Norwalk, CT: Appleton & Lange; 2002. p.1498–1510.
- Rotenberg JS. Cyanide as a weapon of terror. *Pediatr Annals* 2003;32:236–240.
- Sidell FR. What to do in case of an unthinkable chemical warfare attack or accident. *Postgrad Med* 1990;88:70–84.

Vesicants

- Andreassi L. Chemical warfare and the skin. *Int J Dermatol.* 1991;30.
- Armada M, Mendelson M. Chemical terrorism update: vesicants. *Emerg Med.* 2002:34(9):51–57.
- Barranco VP. Mustard gas and the dermatologist. *Int J Dermatol.* 1991;30;684–686.
- Briggs SM, Brinsfield KH. *Advanced Disaster Medical Response. Manual for Providers*. Boston, MA: Harvard Medical International Trauma and Disaster Institute, 2003.
- Hu H, Cook-Deegan R, Shukri A. The use of chemical weapons: conducting an investigation using survey epidemiology. *JAMA.* 1989;262;640–643.
- Marshall E. Chemical genocide in Iraq? *Science.* 1988;241:1752.
- Momeni A, Arminjavaheri M. Skin manifestations of mustard gas in a group of 14 children and teenagers: a clinical study. *Int J Dermatol.* 1994;33:184–187.
- Momeni A, Enshaeih S, Meghdadi M, et al. Skin manifestations of mustard gas. *Arch Dermatol.* 1992;128:775–780.
- Safarinejad MR, Moosavi SA, Montazeri B. Ocular injuries caused by mustard gas. Diagnosis, treatment, and medical defense. *Military Med.* 2001;166;67–70.
- Sidwell FR, Takafuji ET, Franz DR, eds. *Textbook of Military Medicine Part 1: Medical Aspects of Chemical and Biological Warfare.* Washington DC: Office of the Surgeon General, Walter Reed Army Medical Center; 1997.
- Smith WJ, Dunn MA. Medical defense against blistering chemical warfare agents. *Arch Dermatol.* 1991;127:1207–1213.
- U.S. Army Medical Research Institute of Chemical Defense. *Medical Management of Chemical Casualties*, 3rd ed. Aberdeen Proving Ground, MD: US Army Medical Research Institute of Chemical Defense; 2000.

Pulmonary Agents

- Bunting H. Clinical Findings in Acute Chlorine Poisoning. In: *Respiratory Tract.* Vol 2. In: *Fasciculus on Chemical Warfare Medicine*. Washington, DC: Committee on Treatment of Gas Casualties, National Research Council; 1945: 51–60.
- Central Intelligence Agency. Unclassified Report to Congress on the Acquisition of Technology Relating to Weapons of Mass Destruction and Advanced Conventional Munitions, 1 January through 30 June 2001. Washington, DC Central Intelligence Agency: Available at: https://www.cia.gov/cia/reports/721_reports/jan_jun2001.htm. Accessed August 18, 2006.

- Gray EA, Love JW, Arnold JL. CBRNE—Lung-damaging Agents, Phosgene. In: *eMedicine: Emergency Medicine* (Textbook Online). St. Petersburg, FL: WebMD; June 13, 2006.
- Grimaldi JV and Gugliotta G. Chemical plants are feared as targets: views differ on ways to avert catastrophe. *The Washington Post.* December 16, 2001:A1.
- Henretig FM, Cieslak TJ, Madsen JM, et al. The Emergency Department Response to Incidents of Biological and Chemical Terrorism. In: Fleisher GR, Ludwig S, Henretig FM, eds. *Textbook of Pediatric Emergency Medicine*, 5th ed. Philadelphia: Lippincott Williams and Wilkins, 2005.
- Noltkamper D, O'Malley GF. CBRNE—Lung-damaging Agents, Chlorine. In: eMedicine: *Emergency Medicine.* (Textbook Online). St. Petersburg, FL: WebMD.
- Organization for the Prohibition of Chemical Weapons. Chemical Weapons Convention. Available at: http://www.opcw.org/html/glance/index.html.
- Sidell FR and Franz DR. Overview: Defense Against the Effects of Chemical and Biological Warfare Agents. In: Sidell FR, Takafuji ET, Franz DR, eds. *Textbook of Military Medicine. Part 1: Medical Aspects of Chemical and Biological Warfare.* Washington DC: Office of the Surgeon General, Walter Reed Army Medical Center; 1997.
- Urbanetti JS. Toxic Inhalational Agents. In: Sidell FR, Takafuji ET, Franz DR, eds. *Textbook of Military Medicine. Part 1: Medical Aspects of Chemical and Biological Warfare*. Washington, DC: Office of the Surgeon General, Walter Reed Army Medical Center; 1997.

Riot Control Agents

- Billmire DF, Robinson NB, Panitch HB, et al. Pepper-spray induced respiratory failure treated with extracorporeal membrane oxygenation. *Pediatrics.* 1996;98:961–963.
- Park S, Glammons ST. Toxic effects of tear gas on an infant following prolonged exposure. *Amer J Dis Child.* 1972;123:245–247.
- Sidell FR. Riot Control Agents. In: Sidell FR, Takafuji ET, Franz DR, eds. *Textbook of Military Medicine: Medical Aspects of Chemical and Biological Warfare.* Washington DC: Office of the Surgeon General, Walter Reed Army Medical Center; 1997. 450–476.
- Smith J, Greaves I. The use of chemical incapacitant sprays: a review. *J Trauma*. 2002;52:595–600.
- Weir E. The health impact of crowd-control agents. *CMAJ*. 2001;164:1889–1890.

Table 5.1. Pediatric vulnerabilities to chemical terrorism

Realm	Potential vulnerability	Potential response
Physiologic	Increased respiratory exposure (higher minute ventilation, live "closer to the ground)	Early warning, sheltering* (gas masks not advised because of risk of poor fit, suffocation)
	Increased dermal exposure (thinner, more permeable skin; larger body surface area/mass ratio)	Protective clothing, early decontamination[1]
	Increased risk of dehydration, shock with illness-induced vomiting, diarrhea (decreased fluid reserves, larger body surface area/mass ratio)	Recognition, aggressive fluid therapy
	Increased risk of hypothermia during decontamination (larger body surface area/mass ratio)	Warm water decontamination
	More fulminant disease; (possible) physiologic detoxification immaturity; more permeable blood-brain barrier	Pediatric-specific research for early diagnosis and treatment of chemical weapons victims[1]
Developmental	Less ability to escape attack site, take appropriate evasive actions (developmental immaturity, normal dependence on adult caregivers who might be injured or dead)	?
Psychological	Less coping skill of children who suffer injury or witness parental, sibling death (psychological immaturity)	Child psychiatry involvement, research for preventing pediatric post-traumatic stress disorder[1]
	Greater anxiety over reported incidents, hoaxes, media coverage, etc	Pediatric counseling of parents and children†
EMS	Less capacity to cope with influx of critical pediatric patients	Community and regional planning with significant pediatric input
	Loss of routine hospital transfer protocols	
	Limited ability to expand pediatric hospital bed capacity through NDMS	

* Plausible, but unproved or unstudied, and/or not intuitively obvious
† For AAP and AACAP resources for parents and pediatricians, *see* http://www.aap.org/advocacy/releases/disastercomm.htm and http://www.aacap.org/publications/factsfam/disaster.htm.

Source: Adapted from Henretig FM, Cieslak TJ, Eitzen EM Jr. Biological and chemical terrorism. *J Pediatr* 141:311–326, © 2002, with permission from Elsevier.

Table 5.2. Chemical weapons – Summary of pediatric management considerations

Agent	Toxicity	Clinical findings	Onset	Decontamination*	Management
Nerve Agents					
Tabun **Sarin** **Soman** **VX**	Anticholinesterase: muscarinic, nicotinic, and CNS effects	*Vapor:* miosis, rhinorrhea, dyspnea *Liquid:* diaphoresis, vomiting *Both:* coma, paralysis, seizures, apnea	*Vapor:* seconds *Liquid:* minutes-hours	*Vapor:* fresh air, remove clothes, wash hair *Liquid:* remove clothes, copious washing of skin and hair with soap and water, ocular irrigation	ABCs Atropine: 0.05 mg/kg IV†, IM‡ (min 0.1 mg, max 5 mg), repeat q2–5 min prn for marked secretions, bronchospasm Pralidoxime: 25 mg/kg IV, IM (max 1 g IV; 2 g IM), may repeat within 30–60 min prn, then again every hour for 1 or 2 doses prn for persistent weakness, high atropine requirement Diazepam: 0.3 mg/kg (max 10 mg) IV; lorazepam: 0.1 mg/kg IV, IM (max 4 mg); midazolam: 0.2 mg/kg (max 10 mg) IM prn for seizures or severe exposure
Vesicants					
Mustard	Alkylation	Skin erythema, vesicles, ocular inflammation, respiratory tract inflammation	Hours	Wash skin with soap and water, ocular irrigation (major impact only if done within min of exposure)	Symptomatic care
Lewisite	Arsenical		Immediate pain		Possibly BAL 3 mg/kg IM q4–6hr for systemic effects in severe cases
Pulmonary Agents					
Chlorine Phosgene	Liberate HCL, alkylation	Eyes, nose, throat irritation (especially chlorine); bronchospasm, pulmonary edema (especially phosgene)	Minutes: eyes, nose, throat irritation; bronchospasm Hours: pulmonary edema	Fresh air, wash skin with water	Symptomatic care

Table 5.2. Chemical weapons – Summary of pediatric management considerations, continued

Cyanide					
Cyanide	Cytochrome oxidase inhibition: cellular anoxia, lactic acidosis	Tachypnea, coma, seizures, apnea	Seconds	Fresh air, wash skin with soap and water	ABCs, 100% oxygen Sodium bicarbonate prn for metabolic acidosis Sodium nitrite (3%): *Dosage (mL/kg)* *Estimated Hgb (g/dL)* 0.27 10 0.33 12 (est. for avg. child) 0.39 14 (max 10 mL) Sodium thiosulfate (25%): 1.65 mL/kg (max 50 mL)
Riot Control Agents					
CS CN (eg, Mace®) Capsaicin (pepper spray)	Neuropeptide substance P release; alkylation	Ocular pain, tearing, blepharospasm; nose and throat irritation; pulmonary failure (rare)	Seconds	Fresh air, ocular irrigation	Topical ophthalmics, symptomatic care

* Should be performed by health care providers garbed in adequate personal protective equipment, especially if victims have had significant exposure to nerve agents or vesicants. For emergency department staff, adequate PPE consists of a non-encapsulated, chemically resistant body suit, boots, and gloves with a full-face air purifier mask/hood.

† Intraosseous route likely equivalent to intravenous.

‡ Atropine via endotracheal tube or inhalation, or aerosolized ipratropium of possible benefit.

Note: ABCs = airway, breathing, and circulatory support; BAL= British anti-lewisite; Hgb= hemoglobin concentration; prn = as needed

Adapted from Henretig FM, Cieslak TJ, Eitzen EM Jr. Biological and chemical terrorism. *J Pediatr* 141:311-326 © 2002, with permission from Elsevier.

Table 5.3 Representative classes of industrial chemicals – Summary of pediatric management considerations

Agent	Clinical findings	Onset	Decontamination	Management
Strong acids/bases	*Eye:* caustic injury *Skin:* chemical burns *GI:* chemical burns of mouth, larynx, esophagus, stomach	Rapid	*Eye, skin:* immediate copious water irrigation *GI:* defer, immediate emergency department referral	Supportive care, early endoscopy for significant ingestion; antibiotics and steroids controversial, should be individualized, consult Poison Control Center*
Respiratory tract irritants (e.g., ammonia, HCl and HF gases)	EENT and respiratory tract irritation with cough, chest pain, dyspnea, wheeze (possible pulmonary edema in severe cases)	Rapid	Move to fresh air	Supportive respiratory care (consider nebulized calcium gluconate solution for HF, consult Poison Control Center)
Fentanyl and other opioids	CNS and respiratory depression, miosis	Rapid	Move to fresh air (for aerosol exposure), consider AC for ingestion, consult Poison Control Center	Supportive care, naloxone (0.01-0.1 mg/kg)
Cellular asphyxiants (e.g., phosphine, sodium azide)	Cough, dyspnea, headache, dizziness, vomiting, tachycardia, hypotension, severe metabolic acidosis; may progress to coma, seizures, death; may have delayed onset pulmonary edema with phosphine	Rapid (except pulmonary edema with phosphine)	Move to fresh air (consider AC for ingested sodium azide—caution with vomitus, which may emit toxic hydrazoic acid fumes; consult Poison Control Center)	Airway, breathing, and circulatory support; 100% oxygen
Arsine	Severe hemolysis	2–4 hr	Move to fresh air	Supportive care, enhance urine flow, consider alkalinization, consult Poison Control Center

* Contact Poison Control Center at 1-800-222-1222.

Note: EENT = eye, ear, nose, and throat; HCl = hydrogen chloride; HF = hydrogen fluoride; AC = activated charcoal (1g/kg orally or nasogastric).

Table 5.3. Representative classes of industrial chemicals – Summary of pediatric management considerations, continued

Adapted from: Henretig FM, Cieslak TJ, Madsen JM, Eitzen EM Jr, Fleisher GF. The emergency department response to incidents of biological and chemical terrorism. In: Fleisher GF, Ludwig S, Henretig FM (eds). *Textbook of Pediatric Emergency Medicine*, 5th ed. Lippincott Williams & Wilkins, Philadelphia, in press, 2005.

Table 5.4. Nerve agent triage and dosing

Symptoms	Triage level: disposition	Anticholinergics	Oxime-pralidoxime chloride (2-PAM)	Benzodiazepines*
Asymptomatic	Delayed: observe	None	None	None
Miosis, mild rhinnorhea	Delayed: admit or observe	None	None	None
Miosis and any other symptom	Immediate: admit	Atropine 0.05 mg/kg IV, IM, IO, to max 4 mg; repeat as needed q 5–10 min until pulmonary resistance improves or secretions resolve; correct hypoxia before IV use; increased risk of ventricular fibrillation Alternatives: Consider scopolamine for nervous system and peripheral effects; glycopyrrolate for peripheral effects only	2-PAM 25–50 mg/kg IV, IM, to max 1,800 mg; repeat q 1 hr prn; watch for muscle rigidity, laryngospasm, tachycardia, hypertension	If neurologic symptoms or rapid progression: Midazolam 0.15–0.2 mg/kg, IM, IV, repeat as necessary or start continuous IV drip; less likely to cause apnea by IM route Diazepam IV, prn (see below) Lorazepam IV at 0.05–0.1 mg/kg (IM absorption variable) If IV or IM is not available, consider midazolam given sublingual or intranasal at 0.2 mg/kg or diazepam prn or lorazepam prn

Table 5.4. Nerve agent triage and dosing, continued

Apnea, convulsions, cardiopulmonary arrest	Immediate: admit, intensive-care status	Atropine 0.05–0.10 mg/kg IV, IM, IO Repeat q 5–10 min as above (no max) Endotracheal tube: increase dose 2–3 times, mix with 3–5 mL normal saline and introduce via suction catheter, flush 3–5 mL normal saline	2-PAM 25–50 mg/kg IV, IM, as above	Midazolam, as above Diazepam 30 days to 5 yr old: 0.05–0.3 mg/kg, IV, max 5 mg/dose ≥5 yr old: 0.05–0.3 mg/kg, IV, max 10 mg/dose Repeat q 15-30 min, prn Lorazepam IV, IM
Autoinjector		Atropine 2 mg for ≥40 kg 1 mg for ≥20 kg 0.5 mg for ≥10 kg (0.05 mg/kg/dose)	2-PAM 600 mg for ≥12 kg (50 mg/kg/dose)	Diazepam 10 mg for ≥30 kg (0.3 mg/kg/dose)

*Monitor respiratory status and blood pressure.

Note: Remember airway, breathing, circulation, decontamination/drugs. Consider oxygen, bronchodilators, nasogastric tube/drainage, ophthalmic analgesia, mydriatics, temperature control. If prolonged impairment of consciousness, EEG (to rule out nonconvulsive status epilepticus) and imaging.

Adapted from Rotenberg JS, Newmark J. *Pediatrics* 2003; 112:648-58.

Table 5.5. Clinical effects from sulfur mustard exposure

	Eyes	Skin	Respiratory tract	Bone marrow	GI tract	CNS
Minimal	Tearing, burning, mild conjunctivitis, photophobia	Erythema	Rhinorrhea, hoarseness, hacking cough	Reactive leukocytosis	Nausea, vomiting	Apathy, depression, anxiety
Moderate	Severe conjunctivitis with blepharospasm, lid edema, pain, miosis	Blisters	Severe cough, expectoration, aphoria	Leukopenia (often preceded by leukocytosis)	As above	As above
Severe	Corneal edema, severe pain, ocular perforation	Deep burning with full-thickness skin loss	Dyspnea, pulmonary edema, asphyxia, bronchopneumonia	Severe leukopenia, aplastic anemia	Later nausea, vomiting (possible bloody), diarrhea	Agitation, hyper-excitability, abnormal muscular activity, coma

Table 5.6. Riot control agents

Chemical name	Abbreviated/common designation	Uses
2-Chlorobenzylidene	CS	Military, law enforcement, personal protection
1-Chloroacetophenone	CN (Mace®)	Military, law enforcement, personal protection
Dibenzoxazepine	CR	Military
Oleoresin capsicum	Pepper spray	Military, law enforcement, personal protection
Diphenylaminearsine	DM	Military (rare)
Bromobenzylcyanide	CA	Military (rare)

Table 5.7. Medical treatment of riot control agent exposure

Affected organ/symptoms	Treatment
Lungs	
Dyspnea	Oxygen
Bronchospasm	Albuterol 0.5% inhaled: <15 kg: 2.5 mg (0.5 mL in 2 mL normal saline) >15 kg: 5.0 mg (1.0 mL in 2 mL normal saline) Ipratropium bromide inhaled*: >2 yr: 500 µg (1 vial)
Bronchorrhea	Atropine, IV or IM: 0.02 mg/kg/dose (min 0.1 mg) Glycopyrrolate, IV or IM: 0.004 mg/kg/dose (max 0.1 mg)
Eyes	
Decontamination	Remove and discard contacts Copious irrigation with normal saline If CS powder present, blow out of eyes using fan, avoiding contamination of space downwind
Pain	Topical anesthetic (tetracaine 1%, proparacaine 1%)†: 1 drop to each eye (apply before irrigation)
Skin	
Decontamination	Copious irrigation with soap and water (may transiently increase symptoms) *Do not use* bleach (hypochlorite)‡
Pruritus	Oral antihistamines (H_1 receptor blockers): Diphenhydramine 5 mg/kg/day divided into 4 doses *or* Hydroxyzine 2 mg/kg/day divided into 4 doses *or* Equivalent medication
Erythema, dermatitis	Topical steroid preparation, apply sparingly BID to affected area Mild potency on face and genitalia (e.g., hydrocortisone 1% cream) Moderate to high potency on remainder of body (e.g., hydrocortisone 2% ointment, fluocinolone acetonide 0.025% ointment) Ensure proper decontamination
Vesicles, bullae	Burn dressings with topical antibiotic (silver sulfadiazine 1%)

* For severe bronchospasm. Do not give ipratropium bromide to patients with peanut allergy.
† Do not give to patients with allergy to local anesthetics.
‡ Bleach may increase riot agent skin exposure and exacerbate erythema, vesiculation, and blistering.

Chapter 6. Radiological and Nuclear Terrorism

Radiological Threats: Scope and Implications

Nuclear and radiological weapons pose a significant terrorist threat. In the past, terrorists have attacked discrete locations with explosive materials that are not inherently toxic. However, the tactics and technological sophistication of terrorists are continually evolving. The September 2001 terrorist attacks demonstrated sophistication and planning not previously encountered. Future attacks with radiological devices are a real possibility that is outside the experience of most local emergency and health officials. Radiological terrorism could include detonation of one or more nuclear weapons, deployment of a radiation-producing device or other isotopic weapon (e.g., "dirty bomb"), or simply placement of a radioactive source (e.g., nuclear waste material) in a public location. The probability and nature of injuries depends on the type of disaster involved (see Figure 6.1).

Such materials are sought by a variety of terrorist or criminal organizations and have been successfully seized by law enforcement personnel on many occasions. The increasing sophistication and inventiveness exhibited by transnational crime syndicates, drug cartels, anarchists, paramilitary warlords/insurgents and other groups challenge traditional law enforcement approaches. The danger posed by these various illicit organizations blurs the lines between international and domestic threats, as well as between criminal and military threats.

Incident Management

The National Council on Radiation Protection and Measurements (NCRP) suggests that attacks with radioactive materials can result in an area of contamination that is much larger than the immediate scene. Such "weaponized" hazardous materials are not readily amenable to local cleanup and require a paradigm shift in incident management. However, incident management will be difficult until the required and appropriate monitoring equipment is available, along with well-trained technical personnel. Incident response and the forensic investigation are also likely to be complicated by public fear and hysteria, need for personal protective equipment, potential contamination of evidence, and pressure for prompt cleanup and long-term site remediation. Consequently, police, fire, and EMS departments are training and equipping themselves to respond appropriately. State and Federal agencies are planning for how they will to coordinate transport and management of contaminated evidence for rapid diagnostic and forensic analysis, while still preserving safety and the evidentiary chain of custody.

There are several key public health and safety considerations in managing radiological incidents. These include the potential for both immediate and long-term health effects, depending on the specific radionuclide(s) and method of dispersal involved. Other concerns include protection of first responders (including forensic investigators) as well as the general public, casualty triage, decontamination, treatment, and management of emotional distress and fear associated with possible exposure to radiation. Key decision points include evacuation versus sheltering in place and potential restrictions on food and

water consumption. Initial response capabilities will be limited unless appropriate expertise, specialized equipment, and supplies are readily available.

Nuclear Weapons

The so-called "dirty bomb" disperses radioactive material and is relatively simple to deploy. A nuclear weapon would be much more difficult to deploy. Nevertheless, the potential for detonation of a nuclear weapon in a major city cannot be dismissed. A crude nuclear weapon constructed outside of a national program would likely be limited to a yield <10 kilotons (10,000 tons) of TNT equivalent. Theft or provision of a stockpile weapon from a nuclear nation could provide a more efficient and higher yield device.

The destructive action of nuclear weapons is mainly due to blast and heat, as in conventional explosives. However, there are several important differences between nuclear and conventional explosions:

- Nuclear explosions are hundreds to millions of times more powerful than conventional explosions.
- The temperatures attained by nuclear weapons are much higher (tens of millions of degrees versus a few thousand), causing much more of the explosive energy to be emitted as light and heat (thermal radiation). This can result in skin burns and fires at considerable distances from the detonation.
- Nuclear detonations release tremendous amounts of initial radiation.
- The various substances remaining after a nuclear explosion are radioactive and may emit radiation for an extended period of time (i.e., residual radioactivity).

Medical providers should be prepared to adequately treat injuries complicated by ionizing radiation exposure and radioactive contamination. Medical facilities in the immediate area will be nearly unusable due to heavy physical damage. Medical facilities in the adjacent areas will be severely compromised by downed power and phone lines; probable loss of all city utilities; and damage to electronics, communications, and HVAC control induced by the electromagnetic pulse produced by a nuclear blast. Patients who need more than basic medical care will require transport to functioning medical facilities well outside the immediate area of destruction.

Radiation from a nuclear detonation. A nuclear detonation produces four kinds of ionizing radiation: neutron, gamma, beta, and alpha (see section on Radiation Physics later in this chapter). Neutron and gamma radiation are emitted immediately at detonation, in a ratio that decreases with increasing weapon yield and distance from ground zero. The residual radiation is composed largely of alpha particles, beta particles, and gamma rays. This residual radiation comes from fission products, unfissioned residual nuclear material, and ground-zero materials made radioactive by neutrons from the initial radiation (i.e., neutron-activation).

For most weapons, blast and thermal injuries will outnumber radiation injuries. The types of injuries associated with the initial detonation vary with yield, distance from ground zero, time of day, and other factors. For weapons <10 kilotons (KT), most injuries requiring medical attention are caused by blast and ionizing radiation within a km or so

from ground zero. For weapons larger than 10 KT, thermal radiation is the primary cause of injury, as this extends over a greater distance than blast or radiation effects. Flash-blindness and retinal burns may occur out to 20 km during daytime and 50 km at night.

Fallout. Fallout is defined as radioactive material from a nuclear detonation that returns to the ground after the explosion. A detonation close to ground level (i.e., surface burst) will result in large quantities of earth and/or water being thermally vaporized and drawn up into a radioactive cloud. Much of this material may be blasted into the atmosphere and subsequently return to earth as fallout. This material becomes radioactive from either neutron activation or from condensing together with various radioactive isotopes. In highly contaminated areas, fallout can cause potentially lethal external radiation exposure, as well as provide a serious internal hazard from the inhalation or ingestion of radioactive materials, such as milk (see Figure 6.2).

A surface burst over the ground produces particles that range in size from submicron to several millimeters in diameter. The finer particles rise into the stratosphere and may be dispersed as worldwide fallout. The larger particles settle to earth within 24 hours as local radioactive fallout. The heaviest fallout usually occurs in the first 4 hours. In contrast, bursts over water are characterized by lighter and smaller particles, producing a smaller volume of fallout that extends over a larger area. These particles consist mostly of sea salts and water, which may produce a "cloud seeding" effect that results in areas of high local fallout as radioactive materials are washed out of the air.

The primary method of fallout protection is initial sheltering, with rapid evacuation from the contaminated area until the risk has been eliminated through decay and/or remediation. In general, it is very difficult to accurately predict the rate of radioactive decay for fallout. Thus, relevant decisions should be based on actual radiological survey data. The 7:10 rule can be used to estimate residual radiation decay after all fallout is on the ground and the dose rate is beginning to measurably decrease.

Radiological Dispersal Devices (Dirty Bombs)

A radiological dispersal device (RDD) is designed to spread radioactive material through detonation of conventional explosives or other (non-nuclear) means. These "dirty bombs" blast radioactive material into the area around the explosion, exposing people and buildings. The purpose of a dirty bomb is to frighten people and make buildings or land unusable for a long period of time.

An RDD is a weapon of "mass disruption" more than a weapon of mass destruction. However, an RDD can produce external contamination, an exposure hazard, and a risk of internal contamination (via inhalation, ingestion, or wounds) if basic safety and hygiene precautions are not followed. An RDD also may pose a radiation injury risk in the event that a strong source (e.g., Cs-137 or Co-60) is kept relatively concentrated. In this situation, the RDD acts as a high-intensity sealed gamma source.

The principal use of an RDD is to cause fear and to disrupt infrastructure. Use of an RDD also tends to generate panic and social and economic disruption from the physical and

psychological impacts of the attack. The severity of the psychological effects depends in part on the type of RDD material and the method of deployment. Mass psychosomatic symptoms resulting from an unrealistic fear of the effects of radioactive material could place unnecessary strain on the medical system, through medical screening of large numbers of anxious individuals. This would add to the chaos of identifying truly exposed cases, also mixed in among the existing cases of gastrointestinal, dermatologic, and respiratory illness that are prevalent in any population at baseline.

In the event an RDD is deployed, everything possible should be done to contain panic. It is very important to reassure the public that all necessary steps are being taken to safeguard their health and well being. This allows appropriate authorities to methodically contain and manage the incident in an atmosphere free of public interference. Public perception that appropriate treatment is not being provided, particularly for children and other vulnerable individuals, could markedly increase the psychological impact of the event. Medical authorities also need to manage the flow of casualties through proper layout and use of casualty reception stations, decontamination areas, and patient assessment and triage procedures.

Medical and Industrial Sources of Radiation

Radioactive materials used in medical or industrial settings can produce irradiation or contamination from accidental or intentional misuse. Irradiation and contamination are significantly different problems—a person can be irradiated from a distance without being contaminated by radioactive material. Radiological contamination is an issue because of the close proximity of the radionuclide to tissue, whether the contamination lies against the skin (external) or is inside the body (internal).

Powerful industrial radiography sealed sources used in the nondestructive testing of oil and water pipelines have caused severe exposures. Their bright stainless steel casings are eye-catching when the sources are accidentally left behind, and they have been stolen when positive control of the source was lost. They are a potential terrorist weapon and could present a serious localized radiation threat.

Another important medical/industrial source is Cs-137, which is an important decay product resulting from the fission of uranium and plutonium fuels. This isotope is used in both industrial sealed-gamma sources as well as medical therapeutic sealed sources. Cesium (Cs) is an alkaline metal that is metabolized much like potassium and excreted through the kidneys. It has a radioactive half-life of 30 years but a biological half-time in adults ranging from 68 to 165 days (average 109 days). The biological half-time is shorter in children, ranging from 12 days in infants to 57 days in older children. It is also shorter in women (84 ± 27days) than in men.

An example of the potential danger from an uncontrolled Cs-137 source occurred in 1987. An abandoned radiotherapy sealed source containing 1400 Curies (Ci) of Cs-137 powder was opened by looters in Goiania, Brazil. About 250 people were subsequently exposed. The victims included children who rubbed the glowing powder on their bodies and, in at least one case, ingested it. Resulting radiation doses were as high as 10 Sv

(1000 rem), resulting in four fatalities from acute radiation sickness (ARS). In this case, the Cs-137 had the exposures characteristic of both a sealed source (producing ARS) and an RDD causing both external and internal contamination. An estimated 120,000 concerned people had to be screened for possible contamination, with incident management costs in the millions of dollars.

Nuclear Power Plants

The United States has 104 nuclear reactors licensed to provide electric power, as well as 36 reactors licensed for other uses. The U.S. Nuclear Regulatory Commission (NRC) has stringent physical protection requirements against sabotage, which cover both plant design and security protection features. Unlike the design of some foreign reactors, designs for power reactors operating in the United States incorporate a layered system of physical shields and walls, including a potentially pressurized containment vessel. Consequently, there have been relatively few mishaps involving American-designed power reactors.

The primary down-wind hazard from destruction or sabotage of a nuclear reactor is the venting of radioactive iodine gas. Power reactors cannot detonate like a nuclear bomb, because reactor fuel does not contain the highly enriched uranium needed for detonation.

The only significant U.S. catastrophic reactor failure occurred in March 1979, at the Three Mile Island (TMI) nuclear power facility in Pennsylvania. At that time, one of the TMI reactors experienced overheating and a meltdown of a portion of its fuel rods. This event resulted in a small release of radioactive gas to the environment, primarily Iodine-131. The radiation produced negligible doses to people residing near the plant (estimated maximum dose of 0.001 Sievert [Sv] [100 mrem]), which is equivalent to a routine chest radiograph). There has been no evidence to date of public injury resulting from the small amount of nuclear material that was vented as a result of this incident.

Pediatricians may be asked about the safety of consuming milk after a reactor accident. Iodine-131 fallout on vegetation has an effective half-life of about 5 days (combination of radioactive decay half-life [8 days] and vegetation half-life). An infant consuming 1 L of milk per day contaminated with 1 microCurie (μCi)/L would receive a total cumulative dose to the thyroid of about 16 rem. Therefore, locally produced milk, fruit, and vegetables should be declared fit for consumption only after clearance by appropriately-trained health inspectors. Emergency reference doses of 0.25 μCi/L peak level in milk and 1.5 $\mu Ci/m^2$ on pasture can be used to guide consumption (exposure) countermeasures. (See Medical Treatment section later in this chapter for information on radioactive iodine and thyroid issues.)

Historical Overview of Radiation Injury

Radiation in the form of “x-rays” was discovered in 1895 by the German physicist Wilhelm Roentgen. Radioactivity in ore was discovered by Antoine Henri Becquerel in 1898. Within a few years of discovery, ionizing radiation was used for a wide variety of medical diagnostic and treatment purposes. Radiation was found to be an extremely

useful modality for medical imaging and an effective means for treating a variety of conditions from acne to malignancies. However, some of the side effects of radiation became known within a few years. Some early radiologists developed radiation skin injury and leukemia from their exposure to ionizing radiation.

Since 1940, there have been over 300 significant radiation accidents in the United States alone. These include both medical and industrial errors, as well as accidents involving the production and storage of nuclear weapons. Some of the most significant worldwide events associated with radiation injury are described below.

Hiroshima and Nagasaki, Japan — 1945

The atomic bomb blasts over Hiroshima and Nagasaki in 1945 during World War II resulted in massive firestorms that obliterated much of the city. They also provided information on the medical effects of uncontrolled ionizing radiation over a wide dose range among those casualties who initially survived the blast and firestorm. The bomb detonated over Hiroshima was equivalent to 15,000 tons (15 KT) of TNT, while the Nagasaki weapon was equivalent to 22 KT of TNT.

It has been estimated that 50% of those exposed to a 2.7–3.1 Gray (Gy) bone marrow dose died within 60 days from their radiation exposure, as little medical assistance was available. Radioactive fallout was very limited, because both of the detonations were airbursts, in which the actual fireball does not reach the ground. The estimates of maximum dose due to fallout are 1–3 centigray (cGy) in Hiroshima and 20–40 cGy in Nagasaki.

The principal effects of radiation in unborn infants in Hiroshima and Nagasaki were small head size (microcephaly) and mental retardation. Of the 1,600 children exposed before birth who were followed, 30 were found to have severe mental retardation. Those with severe mental retardation were noted to have received radiation exposure between 8 weeks and 25 weeks gestation. The most sensitive time of the gestational period was found to be 8–15 weeks after conception.

Ionizing radiation is a relatively weak carcinogen. Among 86,000 atomic-bomb survivors at Hiroshima and Nagasaki followed from 1950 to 1990, there were 7,578 deaths from solid cancer (versus 7,244 expected) and 249 deaths from leukemia (versus 162 expected), representing 421 additional deaths from cancer. The minimum elapsed time between radiation exposure and clinical disease was 2–3 years for leukemia, 3–4 years for bone cancer, 4–5 years for thyroid cancer, and 10 years for other solid tumors.

Mayak, Russia, Former Soviet Union — 1948-1990

The Mayak Production Association (Mayak) is an industrial complex in the Southern Urals of Russia, where the former Soviet Union produced tons of plutonium for nuclear weapons. Between 1948 and 1956, radioactive waste was poured directly into the Techa River, which was the source of drinking water for many villages. It is reported that 124,000 people were exposed to medium and high levels of radiation. In 1957, one of the

cooling systems at Mayak exploded, and more than half the amount of radioactive waste released at Chernobyl went into the atmosphere. Another incident occurred after a reservoir for waste storage evaporated during a dry hot summer. Windstorms then carried 600,000 curies of radioactive dust over 2,700 square kilometers. According to one source, the radiation accidents and radioactive discharge at Mayak killed thousands and made many more ill. The implications for children who were living in this area are enormous, although health data for this population are not readily available.

Marshall Islands — 1954

Fallout played a large role in the Marshall Islands after a 1954 nuclear weapons test on Bikini Island caused fallout on nearby islands, resulting in significant health effects in children. Acute effects included skin injuries from beta radiation (so-called "beta burns"), especially of the feet. The most heavily exposed were the 64 people of Rongelap Island, who were exposed to 190 cGy of external radiation plus 1000–5000 cGy to the thyroid from radioactive iodine. Of 32 individuals who were younger than 20 years old when exposed to the radioactive fallout, 4 developed thyroid cancer and 1 developed leukemia. Two individuals who were younger than 1 year old at exposure developed myxedema and short stature.

Three Mile Island, PA — 1979

On March 28, 1979, a nuclear power plant at Three Mile Island (TMI), PA, had a near meltdown (overheating of the fuel rods and a release of radioactive material). The accident produced negligible doses among people living nearby, with a maximum dose of 0.001 Sievert (Sv) (100 mrem) to the community. Immediate administration of potassium iodide (KI) was recommended for those living near TMI, but this drug was not available. There were no biological effects from the exposure, although significant psychological sequelae occurred.

Chernobyl, Ukraine, Former Soviet Union — 1986

In April 1986, a power plant in Chernobyl (aka Chornobyl), Ukraine, had a mishap that produced a meltdown and hydrogen gas explosion within the reactor core. The area around the reactor was heavily contaminated with plutonium, cesium, and radioactive iodine because there was no containment vessel designed into the facility. An estimated 120 million curies (Ci) of radioactive material was released, contaminating more than 21,000 square kilometers of land. The greatest areas of fallout occurred in Ukraine, Belarus, and the Russian Federation. Approximately 135,000 people were permanently evacuated.

Almost 17 million people, including 2.5 million children younger than age 5, were exposed to excess radiation. The first delayed health effects were noted 4 years after exposure, with a sharp increase in the occurrence of thyroid cancers in children and adolescents, especially among those younger than 4 years at the time of the accident. As of 2002, there were more than 2,000 excess cases of childhood thyroid cancer in the

exposed population. A large area remains uninhabitable because of environmental contamination that exceeds the legally allowable limits.

Goiania, Brazil — 1987

An abandoned radiation therapy facility in Goiania, Brazil, was looted on September 13, 1987. The looters took a canister containing 1,400 Ci of radioactive Cs-137, which was subsequently opened and distributed to a junkyard owner and several families. Children played with the material, rubbing it on their bodies so that they glowed in the dark.

As word of this event spread, thousands of people became anxious about their possible exposure. A stadium was used to screen self-referred potential casualties. Approximately 112,000 people were evaluated, and 249 individuals were found to have either external or internal exposure. Radiation doses were determined to be as high as 10 Sievert (1,000 rem) using cytogenetic techniques (see Radiation Biology and Dosimetry later in this chapter). Twenty individuals needed hospitalization, and four died of acute radiation sickness. Victims developed radiation-associated illnesses that ranged from significant skin irradiation injury to acute radiation sickness and long-term health problems.

Treatment for victims with significant exposure included Prussian Blue, which binds cesium and helps eliminate it from the body, and granulocyte macrophage colony stimulating factor (GM-CSF), which stimulates white blood cell production for victims with radiation-induced neutropenia. Mitigation efforts required the removal of 6,000 tons of dirt, furniture, clothing, and other materials.

Radiological Dispersal Devices (Dirty Bombs)

Terrorists are clearly interested in obtaining RDDs, although a dirty bomb has not yet been exploded. Some incidents involving RDDs are described below.

- November 1995: Chechen rebels in Moscow, Russia, informed a Russian television station that they had buried a cache of radiological materials in a park. Authorities found a partially buried source of cesium.
- March 1998, Greensboro, NC: 19 tubes of Cs-137 were stolen from a hospital.
- December 1998, Argun, Chechnya: A container filled with radioactive material was found attached to an explosive mine near a railway line.
- June 2002, Chicago, IL: An American citizen was arrested on suspicion of planning to build and detonate a dirty bomb in an American city.
- January 2003, Herat, Afghanistan: British intelligence agents reported that Al Qaeda had succeeded in constructing a small dirty bomb.

Other Radiation Uses and Injuries

Prior to the 1960s, ionizing radiation was deemed nearly innocuous and was often believed to be beneficial. Individual exposures to low-level radiation commonly occurred from cosmetics, luminous paints, medical and dental x-ray machines, and gadgets for fitting shoes in retail stores. Fluoroscopy was widely used, as in the routine monthly well-baby visits of at least one large pediatric practice, and to shrink the thymus gland in other

pediatric practices. This put infants at risk of thyroid cancer years later. Radium-224 was given intravenously to treat tuberculosis of the bone, resulting in an excess incidence of bone malignancies. Thorotrast® was also used as an intravenous radiographic contrast medium in Europe, Japan, and the United States, producing excess malignancies of the liver, bile ducts, spleen, brain, and bone in all age groups. Children and adults were also subjected to radiation exposure as part of experimental research in people that would not have met today's ethical standards.

In 1956, the U.S. National Academy of Sciences—National Research Council and the U.K. Medical Research Council published similar conclusions about the potential hazards and the late effects of radiation. Since that time, the uses of ionizing radiation have come under increasing scrutiny and regulation.

Physical Principles of Ionizing Radiation

Atomic Structure

Atoms have two distinct parts that are made up of three main particles. Protons and neutrons are found in the nucleus, which is the tiny, dense center that contains almost the entire mass of the atom. Electrons surround the nucleus, moving in paths called orbitals.

Protons. Protons have a mass of about 1.7×10^{-27} kg, which is assigned the value of 1 atomic mass unit (AMU). Protons carry an electrical charge, which is assigned a value of +1. The density of an atom is determined by the number of protons in the nucleus. For example, uranium has 92 protons, making it much denser than hydrogen, which has only one proton.

Neutrons. Neutrons also have a mass of about 1 AMU, differing from the proton by <0.14%. Unlike the proton, the neutron has no charge. While the number of protons determines the density/identity of an atom, the number of neutrons can vary. Atoms with the same number of protons but different numbers or neutrons are called isotopes of each other. For example, 235U and 237U are two different isotopes of uranium.

Electrons. The mass of an electron is about 1/1800th of an AMU, or about 9×10^{-31} kg. The electron charge is –1. In a neutral atom, the number of electrons and protons match, with their charges canceling each other out. An ion is an atom that has either gained or lost electrons and hence carries a net negative or positive charge, respectively.

Stability and Radiation

Several factors can influence the stability of an element, leading to the potential for radioactivity. An electron or nucleus can become excited by radiation, causing it to become unstable and to give up energy in the form of photons. For example, technetium-99m (99mTc) is an unstable (i.e., metastable) form of technetium that is used in medical settings. When 99mTc goes from an excited state to a non-excited state, it gives off a photon and becomes 99Tc.

The ratio of protons to neutrons in the nucleus is also important to stability. The lighter elements are usually stable, with a ratio of about 1:1. As elements get heavier, the ratio reaches about 2:3, increasing the chances of instability. Whenever the ratio shifts from a stable configuration to an unstable one (for whatever reason), the nucleus attempts to regain stability by emitting radiation.

When an unstable atom emits radiation, it is said to decay. The half-life of a radionuclide is the average amount of time that it takes for half of the atoms in a sample to decay. Half-lives can vary from fractions of a second to millions of years. When an atom decays, it does not necessarily become stable. Rather, it is quite common for decay to be a series process, in which a given atom might decay dozens of times in different ways before becoming stable. Radon gas is a common example of this, with a very long decay chain. Radioactive decay can be associated with several different types of radiation.

Photons (x-rays and gamma rays). Photons are (nearly) massless "bundles" of energy. The energy determines the wavelength, which can be classified into various categories for convenience. Radio waves, visible light, ultraviolet light, x-rays, and gamma rays are all photons of various energies. X-rays and gamma rays differ only in their origin. An electron losing energy yields an x-ray photon, while loss of energy from a nucleus exciting produces a gamma photon. X-rays and gamma rays with the same energy are otherwise identical.

Gamma or x-rays are often released to remove residual energy that remains after other forms of decay (e.g., alpha or beta particles). For example, 60Co, a common industrial and medical radionuclide, decays by beta emission, while simultaneously giving off two powerful gamma rays during its transformation into the stable element 60Ni.

Beta particles (electrons). One of the ways that an unstable nucleus can adjust its proton:neutron ratio is to emit an electron, effectively transforming a neutron into a proton and an electron. This electron is then ejected from the nucleus as a "beta particle," which in all respects is identical to any other electron. Beta particles can cause damage to tissue directly or through secondary processes.

Bremstrahlung. When an electron decelerates (or turns), it loses energy in the form of photons, which is a process called "bremstrahlung." The quicker the deceleration, the greater the energy imparted to the photons. Electron paths tend to curve near heavy nuclei, so that electrons lose energy when interacting with dense matter. This is one of the reasons that lightweight materials are preferred as protection against beta radiation. For example, in old televisions, leaded glass was used for the picture tube. When the electrons from the electron guns hit the back of the screen, they produced bremstrahlung that were roughly equivalent to weak x-rays. Newer televisions are designed to produce minimal bremstrahlung.

Positrons. Some nuclei (e.g., 12N or 124I) emit an anti-electron or positron, converting a proton to a neutron. The ejected positron is identical to an electron but has a +1 charge rather than a –1 charge. Because it is an anti-particle to an electron, the two will annihilate when they interact. The annihilation completely converts both particles to

energy, releasing two photons of exactly 0.511 MeV. Positron emission and the detection of the annihilation photons form the basis of positron emission tomography (PET), a fairly common medical imaging procedure.

Alpha particles. Alpha particles are stable helium nuclei (two protons and two neutrons) ejected mainly (but not exclusively) by heavy nuclei during adjustment of improper proton:neutron ratios. The two protons and two neutrons are emitted, often with considerable energy, and the particle has a +2 charge. Alpha particles are the most massive and highly charged of the common types of radiation. Both of these properties make them potentially very damaging but also very easy to shield.

Neutrons. Neutrons are rarely emitted as decay products. Rather, they are more commonly emitted as by-products of nuclear fission or fusion. As neutrons have no charge, they are able to interact readily with the nucleus of an atom, possibly being absorbed into it. This affects the proton:neutron ratio and can lead to the atom becoming radioactive or otherwise unstable.

Radiation Interactions

Radioactive particles can interact in many ways, leading to potentially hazardous processes and by-products.

Electron excitation. The simplest way for a radiation particle (including a photon) to interact with a target atom is for it to impart some energy to the electrons of the atom. If enough energy is imparted, the electron will be knocked out, and an ion will be formed. If the energy is not sufficient to form an ion, the electron will merely be excited, giving back this extra energy in the form of a photon. This photon can be in the form of visible light (e.g., the phosphors in a television) or as a more hazardous x-ray.

Ionization. The primary radiation effect of medical concern is ionization, in which an electron is completely removed from an atom. Ionization can directly or indirectly break chemical bonds, leading to tissue damage. Furthermore, if an electron is removed from the inner shell of an atom, the cascade of electrons dropping to fill the gap may produce a series of x-rays, which can carry the damage beyond the original track of the incident particle. Similarly, if the electron that was removed has sufficient energy, it may induce its own ionization events, again carrying the effects away from the original track. These follow-on events are called secondary radiation.

Nuclear excitation. If an incident particle interacts with the nucleus of an atom, several outcomes are possible. Nuclear fission or fusion is possible, although the simplest outcome is excitation. As noted above, when a nucleus goes from an excited to an unexcited state, it will usually emit a photon in the form of a gamma ray.

Nuclear fission. When certain heavy nuclei become unstable with respect to their proton:neutron ratio, they can undergo a splitting process termed fission. Typically, this process is induced by the absorption of a neutron into the nucleus of the target atom. The nucleus then breaks into two or more fission fragments, including lighter elements, subatomic particles, and energy.

The subatomic particles that can be released during fission include more neutrons, which can in turn induce new fissions if the conditions are appropriate. This latter process is called a chain reaction and is the heart of nuclear reactors and weapons. Uranium, thorium, and plutonium have isotopes that can undergo fission. A large number of different possible fission fragments can be created, and many of them are themselves unstable and hence radioactive. Some of the most common and hazardous byproducts of fission include 131I, 137Cs, and 90Sr.

Activation. Neutrons lack a charge and can therefore readily reach the nucleus of an atom. If the neutron is absorbed by the nucleus, the new nucleus may have an unstable proton:neutron ratio as well as excess energy. This new configuration may or may not be stable. If it is not stable, it will eventually decay. In other words, the new nucleus may be radioactive. This process of creating a new radioactive radionuclide is called activation and is mainly associated with nuclear reactors and nuclear detonations.

Secondary radiation. The radiation "particle" that initially enters a volume/space is considered primary radiation. Any electrons or photons (or anything else) liberated with enough energy to produce new ionizations are considered secondary radiation. Secondary radiation does not necessarily travel in the same direction as the primary energy, leading to secondary damage in new spaces. Furthermore, the secondary radiation is not necessarily of the same type as the primary radiation, leading to different types of effects. For example, a neutron will liberate electrons and numerous gamma and x-rays as it decelerates. Very high energy radiation (e.g., cosmic radiation) travels quickly through a given space (e.g., human tissue), producing only a small number of interactions. However, each of these interactions can transfer large amounts of energy as secondary radiation, which can then distribute throughout the given space.

Radiation Damage and Protection

Linear energy transfer (LET). As a radiation "particle" passes through matter, it loses energy through the various mechanisms discussed above. The rate at which it loses that energy is the LET, which is measured in energy per unit distance (e.g., MeV per micron). A particle with high LET transfers energy very quickly, slowing down rapidly and depositing energy on a dense "track" in the target volume. A particle with low LET interacts much less often and slows down only gradually. For example, photons are low LET radiation because they have no mass or charge, while alpha particles are high-LET radiation due their large mass and +2 charge. Beta particles and neutrons are intermediate, with neutrons having the higher LET of the two.

Penetration and radiation shielding. The more rapidly a given radiation particle loses energy (high LET), the more quickly and easily it is stopped. Therefore, the LET and the degree to which a particle is considered penetrating are inversely related—i.e., the higher the LET, the less penetrating the particle. For example, an alpha particle deposits very large amounts of energy in a very short distance, which can result in considerable damage to living cells. However, alpha particles cannot penetrate even the thickness of a piece of paper or the dead layer of skin on your body. Therefore, alpha particles generally do not

need to be shielded, although the alpha-emitting materials should be prevented from entering the airways (e.g., with filtering masks) to protect delicate lung tissue. On the other hand, high-energy gamma rays need to be shielded with very thick lead or concrete because they interact so little and penetrate so deeply. As noted earlier, lightweight materials such as Plexiglas® or aluminum are preferred as shielding for beta particles to reduce bremstrahlung.

Radiation Units

The measurement of radiation required the creation of new units to describe various aspects that were new to science. The major units used to describe radiation and its effects are discussed below. This discussion will include both the older American nomenclature and the newer nomenclature under the System International (SI), because both are still widely used.

Radioactivity; curie/becquerel. The units used to describe radioactivity are the curie and the becquerel (SI). Activity represents the number of radioactive decays that take place in 1 second. One curie (Ci) is the number of decays that occur in 1 gram of pure radium-226 in 1 second (i.e., 3.7×10^{10} disintegrations/second). The becquerel (Bq) is one disintegration per second, so that there are 3.7×10^{10} Bq per Ci.

The specific activity (activity per unit of mass) varies from radionuclide to radionuclide, so that the same weight of different materials does not necessary have the same level of radioactivity. For example, 1 curie of 226Ra weighs about 1 gram, as noted above, while 1 Ci of 232Th weighs just under 10 million grams (ten metric tons).

Exposure; roentgen. Formally, the roentgen (R) is a unit of electrical charge liberated by photons per kilogram of air. This unit is specific to x- and gamma rays and cannot be applied to any other form of radiation or to any other medium. It does not have an SI equivalent.

Absorbed dose; Rad/Gray. "Absorbed dose" is the amount of energy absorbed per unit of mass, which applies to any type of radiation in any type of matter. The Rad (radiation absorbed dose) is defined as 100 erg per gram. The SI equivalent is the Gray (Gy), defined as 1 joule per kilogram. One Gy is equivalent to approximately 100 Rad. Often the unit of centiGray (cGy) is used because it is equal to 1 Rad.

Effective dose. Absorbed dose is a very useful concept, but it does not account for the different biological effects of different types of radiation on different types of tissues. Two artificial weighting factors are used to represent the impact of these differences in "effective dose."

Relative biological effectiveness. This weighting factor accounts for how strongly a given radiation interacts with the particular mix of elements that makes up the human body. This factor does not account for shielding and is partially dependent on energy. Note that this weighting factor seems to match up reasonably well with the discussion of LET. For

example, alpha particles and certain neutrons have the greatest effectiveness, while photons and beta particles have the lowest effectiveness (see Table 6.1).

Tissue weighting factors. Different tissues in the human body have differing sensitivities to radiation. Nerve tissue, for example, is fairly insensitive to radiation, while the lining of the GI tract is very sensitive. These weighting factors apply only to cancer risk and cannot be correctly applied to other risks. The sum of all the weighting factors is one. A whole body exposure receives a weighting factor of one, while a partial body exposure would receive the appropriate fraction.

Dose equivalent. The dose equivalent is the product of absorbed dose and the various weighting factors in units of energy per unit mass. The traditional American unit is the REM, which stands for roentgen equivalent man. The REM is defined as 100 ergs per gram, just like the Rad. The SI equivalent is the Sievert (Sv), which is defined as 1 joule per kilogram and is equal to 100 REM. The absorbed dose and the dose equivalent are not the same. The former is a measurable physical quantity, while the latter is the product of consensus weighting factors with a special focus on biological effects.

Radiation Background Exposure

Americans typically receive about 360 millirem of background radiation per year. Of this, approximately 295 millirem is considered natural (cosmic rays, radon, etc.), and the other 65 millirem is considered manmade (x-rays, etc). The main sources of radiation exposure are discussed below.

Natural exposures. There are four sources of natural exposure: radon, cosmic radiation, internal radiation, and terrestrial exposure.

Radon. Radon gas is a natural by-product of the decay of uranium and thorium in rocks and soil. Remember that decay can occur in chains of radionuclides. Radon is part of a long chain. Because it is a gas, it "percolates" up through the soil and into the atmosphere. If it happens to come up under a building, it can become trapped and accumulate in the basement or first floor, leading to sometimes appreciable radiation doses to the occupants. Radon decays eight times before becoming stable, and many of the decays produce very high-energy alpha particles. Because radon is a gas, it can enter the lungs where there is no dead layer of skin to block the alpha particles. Radon and its daughter products account for about 200 millirem of exposure per person per year.

Cosmic radiation. Cosmic radiation, as the name implies, originates from space, either from the sun or from extra-solar sources like supernovae and quasars. Much cosmic radiation is of exceedingly high energy and does not interact at all with the human body. Much more is blocked either by the solar atmosphere or by the earth's atmosphere. The annual cosmic radiation dose at sea level is about 27 millirem, while people living or working at higher elevations (e.g., airline pilots) receive more.

Internal radiation. A number of materials are present in food, water, etc., that expose us to small doses of radiation. Most commonly considered is 40K, a common, naturally

radioactive isotope of potassium. Bananas and some nuts contain elevated levels of radioactivity from this radionuclide. The total dose due to naturally occurring radioactive material incorporated into the body is about 39 millirem per year.

Terrestrial exposure. The earth itself contains a number of radioactive elements. Uranium and potassium have already been mentioned. The annual dose from external radiation by rocks and soils is about 28 millirem.

Manmade exposures. Manmade exposures may occur through medical procedures, consumer products, and/or fallout/nuclear power fallout and waste.

Medical. By far the largest manmade exposure is from medical procedures, such as radiology, radiation therapy, and so forth. For example, the ubiquitous chest x-ray exposes a patient to about 8 millirem. The average annual dose from all medical procedures is about 53 millirem.

Consumer products. A variety of small radiation doses come from consumer products. Cathode ray tubes, radium dials, airport x-rays, etc., all contribute to the 10 millirem annual dose.

Fallout and nuclear power. Combined, fallout from weapons testing and waste from the nuclear power industry produce an annual dose of <1 millirem.

Radiation Biology and Dosimetry

Radiation Biology

Ionizing radiation interacts with biological systems through discrete energy deposition events referred to as spurs, blobs, and tracks. This can occur by direct interaction with cellular and tissue component targets or through the indirect production of free radicals (e.g., hydroxyl radicals, etc.) or other harmful molecules. Radiation-induced damage to DNA and the cellular capacity to repair DNA/chromosome damage have a major influence on the deleterious cellular effects. Additional cellular-specific parameters (e.g., oxygen tension, cell-cycle distribution, proliferative status or rate of cell division, extent of cell differentiation, etc.) and radiation-specific parameters (dose, type of radiation, dose rate, etc.) can modulate the degree of cellular and tissue injury.

The consequences of radiation exposure to cells and tissues are highly variable, including both acute effects (e.g., delay in cell division, cell death) and late effects (e.g., cataracts, mutagenesis, carcinogenesis, etc.). Tissues vary in sensitivity to radiation; various tissues in order of most to least sensitive are lymphoid, gastrointestinal, reproductive, dermal, bone marrow, and nervous system. The response to radiation of self-renewing tissues is dynamic and influenced by tissue-specific transit times. Transit time includes both the time course for stem-cell differentiation and for the lifespan of mature, functioning cells.

Biodosimetry

Effective medical management for suspected radiation overexposure necessitates the recording of dynamic medical data and the measurement of level of injury using appropriate radiation bioassays. Biodosimetric assays can provide diagnostic information to the attending physician, as well as an estimate of the dose for personnel radiation protection records. Radiation biodosimetry usually encompasses multiple parameters, including the following:

- Radioactivity measurements and monitoring of the exposed individual.
- Observation and recording of prodromal signs/symptoms and erythema.
- Complete blood counts with WBC differential.
- Chromosome-aberration cytogenetic bioassay, using the "gold standard" dicentric assay for dose assessment.
- Bioassay sampling (if appropriate) to determine radioactivity contamination.
- Other available approaches.

The biological response to an absorbed dose of ionizing radiation should be assessed to predict the medical consequences. Physical dosimeters (e.g., film badges) may misrepresent the actual radiation dose and may not be available in a radiological accident or terrorism incident. Multiparameter dose assessments represent the current approach on which to base medical treatment and management decisions. Radiation syndromes are generally characterized into three phases: prodromal, manifest, and latent. The early or prodromal signs and symptoms will be emphasized later in this chapter.

Medical recording. Radiation-induced signs, symptoms, and erythema should be recorded during the course of medical management for radiation casualties to help triage patients and to guide medical management of casualties. Default standard mechanisms and procedures include recording of medical data by the respective medical responders and health care providers.

Additional medical guidance available to first responders and health care providers includes the following:

- International Atomic Energy Agency's (IAEA) Generic Procedures for Medical Response during Nuclear and Radiological Emergency (http://www-pub.iaea.org/MTCD/publications/PDF/EPR-MEDICAL-2005_web.pdf).
- Armed Forces Radiobiology Research Institute's (AFRRI) radiation casualty management software application (i.e., Biodosimetry Assessment Tool or BAT), which was developed for radiation casualty management. It is distributed to authorized users who have access to the AFRRI Web site.

The BAT software application equips health care providers with diagnostic information (e.g., clinical signs and symptoms, physical dosimetry, etc.) relevant to the management of radiation casualties. Designed primarily for prompt use after a radiation incident, the program facilitates the collection, integration, and archiving of data obtained from exposed individuals. Data collected in templates are compared with established radiation dose responses obtained from the literature to provide multiparameter dose assessments.

An integrated, interactive human body map permits convenient documentation of the location of a personnel dosimeter (if worn by the individual at the time of exposure), radiation-induced erythema, and radioactivity detected by an appropriate radiation detection device.

Radioactivity counting and bioassay. In the case of suspected internal radioactive contamination, the following processes are typically performed concurrently:

- Decontamination to minimize the local radiation dose to the potential wound site.
- Removal and collection of metallic (or other) fragments for isotope identification.
- Biological sample collection for determination of committed dose.

Monitoring/sampling of wounds (i.e., wound swabs or wound cleansing wipes), body orifices (i.e., nasal swabs, oral swabs), and skin surfaces (i.e., wipes) should be performed for confirmation of internal contamination. Although not strictly considered biological samples, metal fragments, bandages/dressings, and clothing should be retained for isotope identification.

Biological specimens should also be collected to determine possible internal radionuclide deposition. Urinalysis and fecal sample analysis are the primary in vitro methods for determining internal dose. Urine is generally a high priority for bioassay, and therefore, "spot" urine as well as 24-hour urine specimens should be collected from accident victims.

In the special case of an improvised nuclear device or any event involving nuclear fission, there is also a need to collect whole blood specimens for biodosimetry. The blood specimens should be labeled, packaged, and shipped to the network of radionuclide bioassay laboratories for neutron activation analysis to estimate individual neutron doses for the casualties.

Prodromal signs, symptoms, and erythema. The pattern of biological responses associated with acute radiation sickness (ARS) can provide initial diagnostic indices and help with triage of potentially exposed individuals. The early clinical responses associated with the radiation prodromal phase include the following:

- Nausea.
- Vomiting.
- Headache.
- Fever.
- Tachycardia.
- Fatigue.
- Weakness.
- Abdominal pain.
- Parotid pain.
- Erythema.

For the dose-dependent signs and symptoms associated with the prodromal phase of whole-body exposure to photon-equivalent radiation, see Table 6.2. An increase in the

exposure dose is associated with a parallel increase in both the constellation of prodromal signs and symptoms and the percentage of exposed individuals affected. For example, only 50% of individuals exposed to 2-Gy acute photon radiation will exhibit emesis. Radiation doses above 2 Gy will result in a progressive increase in the percentage of individuals who vomit and also express the broad range of prodromal symptoms.

Hematology. Radiation exposure produces a predictable pattern of changes in blood-forming tissues. A crude estimate of absorbed dose can be obtained by serial measurement of peripheral lymphocyte blood counts. Therefore, a CBC with WBC differential should be obtained immediately after exposure, three times a day for the next 2–3 days, and then twice a day for the next 3–6 days. Lymphocyte cell counts and lymphocyte depletion kinetics can be used to estimate exposures between 1 and 10 Gy photon equivalent dose range. This biodosimeter is useful only for a few days (<10) after exposure because of the transient nature of radiation-induced lymphocyte depletion.

Cytogenetic bioassay. The analysis of chromosomal aberrations, particularly the incidence of chromosomes with two centromeres (dicentrics) in peripheral blood lymphocyte cultures, is widely used to assess dose after exposure to radiation. Human T lymphocytes have a long half-life, and a small proportion survive for decades. The frequency of dicentrics after exposure to radiation remains fairly stable for up to a few weeks. After acute partial-body exposure, the irradiated lymphocytes rapidly mix with unirradiated blood, reaching equilibrium within 24 hours.

Blood for cytogenetic bioassay should generally be collected within 24 hours of the irradiation incident. Peripheral blood (10 mL) from the exposed person is collected in a lithium-heparin collection tube, although an EDTA tube can be substituted if necessary. The blood must be transported immediately to a cytogenetic laboratory that is familiar with biodosimetry according to internationally accepted guidelines. During transport, the sample should be chilled with cold packs sufficient to keep the sample cool (4^{o}C) but not frozen. The blood lymphocytes are then isolated and stimulated to grow in culture. Cell proliferation is arrested in the first metaphase, and metaphase spreads are observed under a microscope. The observed level of dicentrics can then be used to estimate dose by comparison with an established dose-response curve.

High-dose partial-body radiation exposures represent a common clinical scenario after accidents. Differences of 10% in absorbed dose can produce clearly observable variations in biological response. Hematological recovery in heavily irradiated areas of the body will be possible if a sufficient number of stem cells survive in unirradiated or mildly irradiated portions of the hematopoietic system. Knowledge of the heterogeneity of the absorbed dose is particularly important in making appropriate medical treatment decisions for patients exhibiting radiation-induced bone marrow syndrome. Cytokine therapy will stimulate proliferation of spared stem cells, but in cases of whole-body stem-cell sterility, bone-marrow transplant or alternative therapeutic measures may become necessary. In high-dose partial-body exposure scenarios, chromosome damage measured in peripheral blood lymphocytes helps determine the absorbed dose.

Biodosimetry and Radiological Terrorism

Effective medical management of radiation casualties after a terrorist incident requires a coordinated and adequate biodosimetric assessment for individuals suspected of exposure. A Medical Field Card (Figure 6.3) should be kept with the patient and used to record early signs and symptoms. This is consistent with an all-hazard approach for medical first responders and health care providers. Emphasis should be placed on recording the characteristic early signs and symptoms of radiation exposure (Table 6.3). Table 6.2 illustrates several of the quantitative multiparameter biodosimetry indices (stratified by 100 cGy dose windows from 1 to 10 Gy) that could be used for early diagnosis and triage of suspect radiation casualties.

There are two important issues to consider when collecting appropriate specimens for radioactivity counting and bioassay in a mass casualty situation.

1. A vigorous screening process should be implemented at an assessment center(s). This helps determine the most likely candidates for bioassay of internal contamination by external counting, using hand-held or portal radiation detectors. Effective screening assures that appropriate victims are assayed, while avoiding the default decision of assaying everyone. The latter could overwhelm the limited resources available for performing the analyses.
2. Specimens must be shipped to confirmed qualified laboratories. It is imperative to have established laboratory standards for bioassay procedures for assessment of internal radionuclide deposition, as well as a robust certification process for the laboratory network that will be conducting the analyses. The Department of Energy (DOE) performs bioassay and dosimetry at a number of sites and also has a program (DOE Laboratory Accreditation Program) to certify these laboratories.

The above laboratories will probably be different from those providing blood cell counts and cytogenetic biodosimetry. During the first 3 days of a mass radiological casualty situation, each potentially exposed person should have a minimum of five CBCs with WBC differentials. To adequately address this requirement, additional assistance (i.e., deployable hematology laboratory capacity) will likely be required to supplement local medical resources.

Cytogenetic confirmation (lymphocyte dicentric assay) of clinical triage has been proposed to help deal with mass radiological casualties or when there is an urgent need for rapid results. Confirmation of clinical triage can generally be accomplished using a simplified assay that scores only 20–50 metaphase spreads per subject, as compared with a typical analysis of 500–1,000 spreads. However, additional scoring is recommended after the initial results are communicated to the physician to resolve potential conflicts in dose assessment and to assist physicians considering marrow-stem-cell transfusions to mitigate bone marrow ablation caused by high doses.

Pediatric Issues

Compared with adults, children have a significantly higher risk of developing cancer from radiation exposure. For this reason, pediatricians should be prepared for any special

medical needs supporting biodosimetry assessments. There is limited knowledge in the available literature about biological dosimetry in pediatric populations. Current models to determine committed dose based on internal contamination are generally based on adult population parameters. However, the procedures applied to the general population are probably applicable to children in most cases. Special containers are required for collecting radioactivity counting and bioassay samples from infants and children (e.g., pediatric urine collection device).

With today's microtechnology, as little as 1 mL of blood can be collected for blood cell counts and cytogenetic biodosimetry in children, given their smaller blood volume and concerns about blood loss. The kinetic lymphocyte depletion method is preferred over single blood lymphocyte counts of dose assessment, given that normal lymphocyte counts in children decrease with advancing age.

The cytogenetic bioassay (dicentrics in metaphase spreads) can also be used in pediatric populations, although normal values may need to be adjusted. The consensus background frequency of dicentrics is 1 in 1000 metaphase spreads for the healthy adult population. Children generally show a lower frequency of dicentrics, and older adults (older than 60 years) a slightly higher frequency, compared with the normal adult background. There is one report in the literature that children younger than 1 year of age may be relatively more radiosensitive. This study found that dicentric yields after in vitro irradiation of blood from children are higher than in blood from adults exposed to similar radiation levels.

Medical Diagnosis: Acute Radiation Syndrome

Acute radiation syndrome (ARS) can occur from radiation exposures during peacetime or as a result of war or terrorism. A case of ARS can be caused by an accident with a military or civilian radioactive source or as a complication of medical treatment. A radiation dispersal device (RDD) would probably not result in exposures high enough to cause ARS, although it is possible. This section describes ARS as it has occurred since the 1940s and as it may occur in the future.

Acute radiation syndrome is also termed acute radiation sickness or radiation toxicity. It is an acute illness that presents as a combination of clinical signs and symptoms. These generally occur in stages during the hours to weeks after acute exposure to a high dose of penetrating radiation (e.g., ≥0.7 Gy). This syndrome generally occurs after irradiation of the whole body (or most of the body), although the signs and symptoms evolve as injury to various tissues and organs is expressed. ARS follows a predictable course after a high or potentially fatal dose of penetrating radiation (e.g., gamma, neutron, or high energy x-rays). ARS is usually associated with prompt exposure or exposure within minutes at a high dose rate, although fractionated doses can also induce ARS.

Pathophysiology

High doses of ionizing radiation cause depletion of stem cell lines and microvascular injury, which lead to the clinical features of ARS. The most radiosensitive cells are

primitive/progenitor cells and other rapidly dividing cells, while slower growing and more mature cells are generally radio-resistant. The most radiosensitive mammalian cells, in decreasing order, include the following:

- Spermatogonia.
- Lymphocytes and oocytes.
- Erythroblasts.
- Other hematopoietic tissue.
- Small-intestine crypt cells.
- Hair follicles.

All of these cells contain rapidly dividing cell lines. Clinicians are familiar with the effects of radiotherapy and chemotherapy on these tissues, as sterility, bone marrow damage, diarrhea, and hair loss all involve radiosensitive stem cell lines. With survivable radiation doses, some stem cells survive, and their cell lines regenerate. Microvascular injury can result in dramatic systemic symptoms and in permanent and irreversible damage such as local radiation injury.

Clinically detectable effects first appear at doses >0.2 Gy (≥20 cGy or 20 rad). These effects include decreased sperm count, chromosome abnormalities, and mild bone marrow depression. Whole-body radiation doses >0.7 Gy can cause clinical illness. The lethal dose 50 (LD_{50}) for penetrating radiation is approximately 3.5 Gy for untreated patients and 5 Gy for those receiving full medical treatment. The LD_{50} is the dose of radiation that will kill half the exposed population.

Clinical Stages

All health effects from radiation exposure tend to follow a similar clinical pattern that can be divided into a series of time-dependent stages: prodrome, latent period, and manifest illness. At higher radiation doses, these stages are associated with shorter time of onset, more severe signs and symptoms, and decreased survival. However, there is individual variation, and symptoms may not occur in all patients.

Prodrome. The initial stage of prodromal symptoms (prodrome) begins within the first few hours to 2 days after exposure. Symptoms include nausea and vomiting, with subsequent malaise, fatigue, and weakness. This is a nonspecific clinical response to acute radiation exposure caused by the cell membrane and free-radical effects of radiation energy, as mediator chemicals such as histamine, interleukins, and cytokines are released.

Latent period. On recovery from the prodrome, there is usually a latent period during which most symptoms subside, although fatigue and weakness may remain.

Manifest illness. This is the full disease picture that develops from the clinical signs and symptoms associated with damage to major organ systems (e.g., blood-forming elements, intestine, cardiovascular, CNS). The molecular cause of disease is DNA damage. Death is usually caused by sepsis.

Syndromes

ARS is not a single syndrome but rather a series of sub-syndromes, each of which evolves over time. The first syndrome involves the hematopoietic system, usually from doses as low as 0.7 Gy. The GI system is affected next, followed by the cardiovascular system and the CNS. These sub-syndromes are progressive and additive with increasing dose, as each organ system is damaged in turn. The cardiovascular and CNS sub-syndromes are usually discussed together because both are rapidly lethal, caused by microvascular injury, and without effective treatment.

Hematopoietic sub-syndrome. The hematopoietic system is affected at doses >0.7 Gy. Although the dose range has been stated as 1–5 Gy, the hematopoietic system actually begins to show damage at doses below 1 Gy, and damage continues at all higher doses. The stem cells of all bone marrow cell lines are affected, so that all production of all blood cells is reduced or stopped. The clinical severity increases with dose, with ancytopenia occurring above about 2 Gy.

The prodrome begins 3–26 hours after doses of 1 to 5 Gy, lasting 48 hours or less. The latent period is mostly asymptomatic, except for possible mild weakness, fatigue, and anorexia that lasts 3–4 weeks. Hair loss (which requires about 3 Gy) and weight loss appear at about day 14. The manifest illness phase sets in at 3–5 weeks, sooner at higher doses. Bone marrow atrophy with pancytopenia (Figure 6.4) can lead to hemorrhage and infection, similar to that which occurs in chemotherapy patients.

Uncomplicated cases can survive with treatment, which includes bone marrow resuscitation and prevention of infections and hemorrhage. Marrow irradiated to 3 Gy shows depletion of cells, which are replaced with fat. Many remaining cells undergo pyknotic death or look grossly abnormal, containing large, bizarre nuclei.

Typical changes in the peripheral blood profile occurs as early as 24 hours after irradiation. Figure 6.5 shows the pattern of blood counts over time after 3 Gy of exposure. Lymphocyte levels fall immediately on day 1. At about 4 weeks, cell counts are at their lowest. The depleted white cells and platelets predispose to hemorrhage and overwhelming infection. Treatment is aimed at protecting patients from infection and restoring blood-forming elements.

Gastrointestinal sub-syndrome. This sub-syndrome is also termed radiation enteropathy and is seen occasionally in the setting of radiation oncology or among victims of a high-dose regional abdominal exposure. Major injury of the GI tract is clinically evident at absorbed, whole-body doses of >5 Gy. The prognosis is grave at doses >6 Gy. Doses >8 Gy are generally lethal.

The prodrome begins abruptly in 1–4 hours (usually 1–2 hours), can last >48 hours, and can be severe. The latent period is 5–7 days, with symptoms of malaise and weakness severe enough to be disabling. The clinical course can be stormy during the manifest illness phase, with complete paralytic ileus as the mucosa breaks down. This is marked by abdominal distention, vomiting, diarrhea, and GI collapse. The damaged mucosa

permits bacterial translocation and sepsis, which is the usual cause of death. If patients survive long enough, hematopoietic syndrome will develop concurrently.

Cardiovascular and CNS sub-syndromes. The radiation dose affecting the cardiovascular system and the CNS begins at >20 Gy, with the full syndrome occurring above 50 Gy. Such extremely high doses have been rare, such as during nuclear fuel handling accidents, in which victims were near a critical mass that suddenly formed ("criticality event"). The key pathological insult is acute microvascular injury with increased endothelial membrane permeability, especially in the brain, leading to cerebral edema and its subsequent clinical effects. The prodrome begins in as little as 5–10 minutes, with rapid onset of uncontrollable nausea, explosive vomiting and diarrhea, and CNS signs that include epileptic seizures and altered mental status. The brief latent period lasts several hours to 2 days. During the latent period, victims have generally been lucid, even euphoric, with a clear sensorium. However, orthostatic hypotension and weakness are often present. The manifest illness phase is marked by rapidly deteriorating CNS status and reduced consciousness, with or without epileptic seizures and a measurable increase in intracranial pressure. Watery diarrhea, respiratory distress, and uncontrollable swings in systemic blood pressure are also common during manifest illness. Coma and death from cerebral edema occur in 2–3 days.

Miscellaneous sub-syndromes. Other radiation-induced clinical sub-syndromes can be observed in special cases or severe exposures. These include radiation pneumonitis, as well as cutaneous and local injuries. Pulmonary effects often play a major role in patients who sustain lethal-dose exposures of >5 Gy, then survive for several weeks with hematopoietic and GI sub-syndromes. With high radiation skin doses, there is acute skin injury, often termed a radiation burn. This is the result of high doses limited to the skin, usually from poorly penetrating radiation (e.g., beta particles) or whole-body irradiation doses >6 Gy. Radiation burns are also a common complication of radiation therapy. Acute local injury can result from external radiation exposures in which parts of the body are shielded. For example, local injury can occur after exposure to a collimated radiation beam or to a highly radioactive material (e.g., radiotherapy or industrial source) placed in close proximity to tissue.

Emergency Care

In an ARS patient, life-threatening medical complications should be addressed first (as in any life-threatening emergency). The other aspects of ARS can be treated after the patient has been stabilized. Airway/breathing/circulation (ABCs) of BLS, ACLS, should be addressed, along with other required emergency resuscitation. After the patient has been stabilized, the need for external decontamination can be addressed.

Diagnostic Steps

The first diagnostic step is to assess the dose of radiation exposure, which may not be obvious. A dose assessment involves collection and analysis of the medical data discussed in this section, as well as consultation with health physicists and subject matter experts. Clinicians and health physicists calculate or determine an initial estimate of dose,

which is revised over time as more clinical and dosimetric data become available (Table 6.4).

Rapid diagnosis. The initial diagnosis provides an initial determination of dose that guides early treatment. Diagnostic steps can begin in an emergency department, in any acute-care setting, or even at a field hospital. The best and quickest diagnostic indicators are:

- The time to onset of vomiting (prodrome).
- The speed of lymphocyte depletion (serial lymphocyte counts).

The time to onset of vomiting provides an estimate of the prodrome and suggests a dose range. The clinician should then observe the onset and duration of the subsequent latency. These various times combine to provide an estimate of dose, which suggests a treatment plan (Table 6.5). For example, prodromal symptoms of vomiting that began 1–2 hours after exposure, with duration of approximately 24 hours, suggests radiation exposure in the range of 3–5 Gy (Table 6.6).

The most useful early laboratory test is the CBC, including an absolute WBC count. WBC counts should be performed every 6-8 hours during the first day after exposure and at least daily thereafter for the next week. Lymphocyte counts are the best rapid gauge of dose. The classic Andrews diagram for lymphocyte depletion curves (Figure 6.6) was published in 1965 and is still useful. Lymphocyte counts drop quickly with high radiation doses. A drop of 50% or more in 24 hours indicates a severe radiation injury.

Later diagnostic data. Later diagnostic data help to refine both the estimate of dose and the treatment plan. Data that should be gathered over time include physical dosimetry, biodosimetry, and clinical physical findings. These data should be recorded for all patients, regardless of symptoms or estimated level of exposure. Such group data may be crucial to clinical evaluation of individuals within the group. These data can also provide a pattern of illness for the group, which can lead to changes in the estimate of dose or even the diagnosis.

Physical dosimetry. The dose assessment method most familiar to medical personnel is physical dosimetry. This can be used for victims who wore a personal dosimeter (e.g., a film badge), thermoluminescent device (TLD), or pocket ionization chamber. However, it is unlikely that civilians—or even most police, firefighters, and military personnel—will have worn dosimeters. It is also important to remember that dosimeters can be damaged during the radiation event, rendering them unreliable. Therefore, clinicians should request dosimetric data from emergency response agencies or military units with trained technicians. Such specialized personnel may be able to estimate dose using radiation detection, indication and computation (RADIAC) equipment and isotope counters.

Biodosimetry. Biodosimetry can provide a good estimate of dose but requires specialized testing. This is performed at a few national centers, including AFRRI in Bethesda, MD, and the Radiation Emergency Action Center Training Site (REAC-TS) in Oak Ridge, TN.

To assess radiation dose, a researcher counts the radiation-induced chromosomal abnormalities in peripheral blood lymphocytes. The classic method is a standard genetic karyotype, which takes about 3 days but offers a good dose estimate. This approach counts the aberrations on a chromosome smear, especially aberrations involving two or more centromeres (dicentrics). The number of chromosomal abnormalities correlates directly with radiation dose (see Appendix E of the *AFRRI Handbook*, available at www.afrri.usuhs.mil).

Radiation-induced skin injury. Skin changes after radiation exposure offer a rough estimate of minimum dose, given that skin changes do not develop below about 3 Gy. Radiation-induced skin injury takes 10–14 days to appear and may resemble a skin burn. "Burns" that appear earlier may not be from radiation but rather from thermal or chemical exposure associated with the event. The severity of the burn increases with increasing dose.

Overall Dose Assessment

All of the above signs, symptoms, and tests can be used to provide an estimate of dose (Table 6.6). Dose assessment is an ongoing process that should include the techniques described above, as well as others recommended by experts. Close attention to dosimetric clues will eventually lead to a reasonably accurate estimate of dose. As the saying goes among health physicists, "Eventually the patient will tell you the dose."

Medical Diagnosis: External Contamination

Background

External contamination with radionuclides can occur in the same settings and situations that cause internal contamination. Any person who simply passes through a contaminated area without appropriate personal protective equipment (PPE), as well as any person who is injured in a contaminated area, will become externally contaminated. Civilians, emergency responders, and military personnel can encounter radionuclides both in peacetime and while waging the war on terrorism. Accidents in medical, industrial, institutional, military, and nuclear research and nuclear power settings can cause external contamination, as could an RDD (e.g., dirty bomb) or nuclear detonation. The largest amount of fallout is on the surface of the ground, so children and crawling infants are particularly prone to pick up this material on their bare skin.

Contaminated materials present as solid particulate matter or liquids that are on the ground or exposed surfaces. Health physicists have described these contaminants as "radioactive dirt" that can be washed off skin and hair. Radionuclides can be deposited on the skin and into body orifices during the immediate event or afterward through contact with contaminated surfaces or liquids. Liquids that contain radioactive materials can readily penetrate body orifices and non-protective clothing, allowing contamination of other external areas or internal contamination by absorption or ingestion. However, up to 95% of contamination is on the outer clothing and shoes. The body surfaces most likely to be contaminated include the hands, face, lower legs, and oral and nasal cavities.

Most radioactive contaminants will be alpha- and beta-particle emitters. Alpha particles do not penetrate the skin but can be a significant source of internal contamination. Beta emitters can cause full-thickness skin damage and subsequent scarring (so-called "radiation burns") when decontamination is delayed or performed improperly. Any radionuclide that emits a high level of gamma radiation can cause whole-body irradiation and ARS in a contaminated victim.

Protection of Medical and Emergency Personnel

Assessment and care of externally contaminated patients pose little to no risk to emergency or medical personnel. Proper technique and correct PPE allow medical personnel to treat radiation patients without fear of medical consequences to either themselves or the patient. First responders and hospital emergency department personnel may assume that care of such patients is dangerous, but history suggests otherwise. There has never been a major exposure among U.S. medical personnel treating radiation victims, with all measured secondary exposures being 14 mRem (0.14 mSv) or less. This dose of radiation is well within occupational standards. High-energy isotopes that pose an immediate medical threat can be detected at safe distances using radiation survey instruments (e.g., RADIACs).

Emergency Care

As with all other patients who present for emergency care, life-threatening complications should be addressed first. External contamination with radioactive agents is unlikely to cause acute injury, so emergency resuscitation and treatment of injuries come first. This is in contrast to external contamination with chemical agents, in which rapid decontamination may be more important. Emergency treatment should not be delayed out of fear of secondary contamination of health care providers because a living patient is unlikely to be a direct threat to health care personnel. The patient has already received a radiation dose several orders of magnitude higher than any caregiver, given that the patient is in direct contact with the contaminants for the longest time.

Decontamination can begin during emergency care but only after the patient is stable. Simply removing contaminated clothing will significantly reduce the subsequent exposure. For a decontamination technique, see the section on medical treatment later in this chapter.

Patient Evaluation

Recommended steps for patient evaluation are as follows:

1. Address life-threatening conditions and significant injuries.
   Note: External contamination has no early signs or symptoms.
2. Disrobe the patient and provide a clean gown and foot covers.
3. Obtain a history; this is crucial.
   What happened during the event?
   Where was the casualty?
   What has been done since the event that would change patient contamination

status?
4. Conduct an initial survey with RADIACs.
5. Collect samples – external swabs/swipes (orifices, wounds, "hot" areas).
6. Begin external decontamination.

History. Diagnosis of external contamination is usually revealed by medical history. A history of some exposure event, together with suspicion of contamination, should lead medical personnel to check the patient with detection instruments. External contamination would probably be asymptomatic at this time, unless the presenting complaint is a skin lesion occurring a considerable time after the incident.

Initial survey and samples. External monitoring and sample collection should begin at the accident scene if possible. This use of monitoring equipment to search for radioactivity is termed a radiological survey. This readily detects external contamination and guides decontamination efforts. First responders can be quickly taught basic survey techniques from health physicists, who are experienced users of survey equipment.

The initial survey involves passing a RADIAC slowly over the entire body, using both alpha- and beta-gamma detectors. The RADIAC should be moved slowly from head-to-toe and side to side, at a rate of about 2–3 cm/sec, and the number of counts/minute should be recorded frequently. At any site that has a high count (i.e., "hot"), a smear sample should be collected, or the area should be "swiped" using gauze or filter paper. These samples should be saved individually in suitable specimen containers for later laboratory analysis (see also the section on medical treatment later in this chapter).

Evaluation of wounds and orifices. Wounds and orifices should also be surveyed to see if decontamination of these sites is indicated. All wounds should be surveyed with RADIAC, because wounds are more likely to be contaminated than intact skin. A "swipe" sample should be collected from any wound with a high count. Wounds should be uncovered and exudates removed/collected before the survey, because dressings and exudates can block alpha particles and low-energy beta particles. Wounds should be dried by application of absorbent material, rather than by rubbing with gauze, which can force contaminants into the tissue.

Contaminants can be naturally cleared from the mouth and nose within about an hour. Therefore, nasal and oral swabs must be collected in the first hour. These should be collected at the accident scene if possible but certainly before the patient is washed or showered. Both nostrils should be swabbed and activity on the swab measured with the RADIAC. The swabs should be saved. Contamination of only one nostril means that the patient has touched his nose with contaminated hands, or that there is unilateral nasal obstruction. Samples of saliva, sputum, and vomitus should also be collected if available.

After each decontamination, all the above RADIAC surveys should be repeated, taking additional samples if there is residual radioactivity. Each sample should be labeled with the patient's identification, the sample site, and the date and time of sample collection.

Medical Diagnosis: Internal Radionuclide Contamination

Background

Worldwide, there have been hundreds of recorded accidents with significant radioactive contamination, but only the accidents that occurred at Chernobyl, Ukraine, and Goiania, Brazil, resulted in significant numbers of children who required internal and external decontamination (see historical overview earlier in this chapter). Radionuclides are widely used in research, medicine, nuclear power, and industry, so further incidents of external and internal contamination will certainly occur. Exposure to radionuclides can occur in peacetime and as a result of a terrorist event such as use of a radiological dispersal device (RDD). Both military and civilian populations are potential targets.

Medical management requires knowledge of the physical and chemical characteristics of radionuclides, as well as the indications and methods for their removal from the body. Patient management must be a team effort that includes physicians, other health care professionals, and health physicists. Patients can be contaminated by any one of the hundreds of radioisotopes in common use, although only a limited number are medically significant. The medical effects from these different exposures vary. Reference materials, such as the National Council on Radiation Protection and Measurements (NCRP) Report No. 65, health physics texts, and standard operating procedures, can be used to guide patient management.

Internal Contamination

Internal contamination occurs when radioactive material enters the bodies of unprotected people through inhalation, ingestion, or wound contamination. Health physicists have defined internal contamination as "unwanted radioactive material present in the body." Contamination and exposure are not the same. Patients can have external exposure to radiation, external contamination by radioactive material, internal contamination with radionuclides, or any combination thereof (Figure 6.7).

Medical effects. ARS has occurred after contamination with high-energy isotopes (e.g., Cs-137). However, internal contamination is usually asymptomatic initially, with no acute medical effects. The main medical concerns are chronic radiation injury of target organs, such as lung or bone, and long-term stochastic effects, such as malignancy.

Causes. The greatest public concern currently centers around terrorism involving radioactive materials. However, medical errors made during medical diagnosis and treatment historically have been the most common cause of significant internal contamination. Industrial accidents are the second most common cause. Institutional and military use of a great number and variety of isotopes has also led to contamination events.

The "dirty bomb." The most likely terrorist use of radioactive materials would be an RDD, variously referred to as a contaminated conventional explosive or "dirty bomb." Such a weapon can contain any liquid or particulate radioisotope. There is no nuclear detonation. Rather, the radioactive material is actually most hazardous before the

explosion, when it is concentrated in one place. The goal of terrorists is to add a radionuclide to an existing bomb (e.g., truck bomb) to create an RDD that can contaminate both victims and the surrounding terrain.

First responders can take several steps to avoid injury from a suspected RDD explosion. RADIAC devices can be used to detect radioactivity at a distance, confirming the RDD. First responders should don protective gear before entering the site and setting up a safe perimeter. A health physicist or a nuclear-biological-chemical team can then analyze samples using specialized laboratory equipment to determine the specific radionuclide.

Isotopes of interest. Internal contamination and casualties are most likely to occur from common radionuclides linked to improper disposal of radioactive sources or damage to a medical or commercial facility. Commonly used radioisotopes include high-energy gamma emitters, such as cesium (Cs)-137, cobalt (Co)-60, and iridium (Ir)-192. These materials are used in industrial and research applications, medical and commercial irradiation, tracer units, thickness gauges, and calibration devices. Standard nuclear fuels, such as uranium and plutonium (Pu) isotopes, are also relatively common. Radioactive iodines (e.g., I-131), Cs-137, and strontium (Sr)-90 are the most medically important fission products associated with rupture of a reactor core or radioactive fallout from nuclear weapon detonations.

Pathophysiology. Radionuclides, their non-radioactive (stable) counterparts, toxins, and chemicals are all governed by the same principles of toxicology. The same pharmacokinetics also apply, as all toxic agents must be absorbed, distributed, and expressed (through toxicological effects on target organs). Factors that determine the amount of internal hazard are the amount of radionuclide, the energy and type of radiation, the length of time in the body, the inherent chemical toxicity, and the critical organ(s) affected. The greatest potential for radiological injury is from large amounts of very energetic, long-lived radioisotopes that can affect certain critical (target) organs.

Biological half-time. Biological half-time (biological half-life) is defined as the time required for half of a substance to be removed from the body. This number comprises both the physical half-life and the metabolic clearance of a radioisotope. A treatment plan can be developed by combining information on half-life and the amount of nuclide exposure. This requires knowledge of the exposure and access to reference texts or experts, because every isotope and chemical formulation has a different half-time. For example, salts of Cs-137 have a half-time of 68–109 days, while tritium (H-3), Pu-239, and soluble uranium salts have biological half-times of 8–12 days, 100 years, and 15 days, respectively.

The critical organ. The critical organ is defined as the bodily location where an isotope exerts its primary effect. A radioisotope is chemically identical to a stable isotope of the same element. Both are metabolized according to their chemistry, so that the critical organ is determined by the chemical properties of the isotope.

Radionuclides that distribute to the whole-body include sodium (Na)-24, Cs-137 (which mimics potassium) and tritium (which is incorporated into water). Bone is the critical organ for radioactive minerals that the body uses as calcium. Similarly, radioactive iodides (e.g., I-131) are rapidly concentrated in the thyroid gland. Ultimately, these toxins will be eliminated through the body's normal excretory mechanisms.

Life Cycle of Internal Contamination

The life cycle of a radioisotope consists of its several stages of transit through the body:

- Intake.
- Uptake.
- Deposition.
- Elimination.

Intake. Radioactive materials can enter the body through inhalation, ingestion, and skin penetration.

Inhalation pathway. Inhalation is the most efficient route of absorption for most toxins, including radionuclides in insoluble particulate aerosols. Large particles are deposited only in the upper airways, from which the mucociliary system clears insoluble particles. The "respirable fraction" is 1–5 microns in size, which is the size associated with efficient deposition in the terminal bronchioles and alveoli. The most hazardous biowarfare agents (e.g., anthrax spores) and industrial dusts (e.g., silica) are in this size range.

Insoluble radioactive particles continue to irradiate surrounding tissues until cleared from the respiratory tract. Soluble isotopes are absorbed quickly and completely from the entire respiratory tract. Insoluble particles are cleared from the upper bronchi within 1 hour, before much injury occurs. The clearance time from the alveoli can be 100 to 1,500 days or more for some compounds. Radioisotopes deposited within alveoli are cleared to regional lymph nodes, where they may remain or be transported systemically. Alpha radiation is the most damaging to alveolar tissue, causing fibrosis and scarring to a spherical volume of tissue around the particle, in addition to any local inflammatory response to the foreign particles.

Ingestion pathway. Swallowed radioactive material enters the digestive tract and is handled like any other ingested material. This is true for material originating from contaminated food or water, as well as for material cleared from the respiratory tract. As with inhalation, absorption from the GI tract depends on the isotope chemistry, including solubility. Most radionuclides are insoluble, with GI absorption <10%. These materials stay in the GI tract without becoming a systemic hazard. The clearance time for the GI lumen is 1–5 days. As a rule, insoluble alpha-particle emitters do not cause significant injury, because the exposure time within the critical organ is relatively short.

However, soluble radionuclides and strong gamma emitters—which have occasionally been ingested—become a whole-body hazard and are capable of causing ARS. Soluble radionuclides are absorbed and metabolized according to their chemistry.

Skin pathway. Although intact skin is generally a barrier to most radionuclides, skin absorption can be important. Most skin absorption occurs through wounds or by passive diffusion. Examples of the latter include tritium-water and radioactive iodine, which can pass readily through skin. Skin permeability rates depend on relative solubility in both lipids and water. Infants are particularly at risk because of their thin epithelium and large surface area to mass ratio. Injuries such as trauma, burns, and chemical exposures also increase skin permeability. Abrasions and partial thickness burns create large denuded skin surfaces, which greatly increases absorption. Clinicians must evaluate all wounds for the presence of radioactivity, and thoroughly clean, debride, or excise all contaminants.

Uptake and deposition. Uptake can occur by simple deposition, diffusion, or metabolic processes. Many soluble nuclides are metabolic analogs of body chemicals, so the body incorporates them like normal building blocks. The critical organs for these soluble nuclides are identical to storage sites for their metabolic analogs (e.g., radium, calcium, and bone). Once a soluble radionuclide is absorbed, it may distribute to the whole body. The liver, kidney, adipose tissue, and bone have higher capacities for binding chemicals, including radionuclides, due to their high protein and lipid content.

Excretion. Insoluble radionuclides pass through the GI tract unchanged. At least some of the absorbed nuclide is eventually excreted, either in its original state or as metabolites. The main route of excretion is via urine, particularly for water-soluble compounds. Lipid-soluble compounds are excreted via the bile into the intestine. There is much individual variability in elimination.

Diagnosis

Clinicians may need to evaluate patients who are both contaminated and injured. Furthermore, patients with internal contamination will almost certainly be externally contaminated at the time of exposure. Initial management and diagnosis should be performed simultaneously if possible. Any life- or limb-threatening medical or surgical emergencies should be addressed first. Initial emergency care is the same as for cases unrelated to radiation injury, because contamination causes no acute medical effects. Assessment and treatment of internal contamination must wait until the patient is medically stable and external decontamination has been completed.

Initial evaluation. The initial evaluation begins at the same time as emergency treatment and consists of the following:

1. Address life-threatening conditions/injuries – the ABCs.
2. Evaluate and control initial contamination.
3. Assess potential for internal deposition and treat as indicated.
4. Patient history is crucial. Patient (or someone else) states what happened.
5. No sign or symptoms.
6. Perform initial survey (RADIAC) and collect nasal swabs (reflect lung deposition).

History. Patients exposed to radiation are usually asymptomatic at the time of presentation. However, history is still the biggest component of initial diagnosis of

contamination. Typically, the patient or someone else provides the important information that an exposure occurred. To quote the experts, "How does a physician become aware that his patient(s) may have an external exposure to radioactive material? Usually, someone tells her."

After a radiological attack, the history provides the best initial indicator of the likelihood of internal exposure. The history should include the exposure setting (e.g., enclosed space, open air in fallout field, etc.) and the protection status of the patient (e.g., wearing a mask). The patient or a fellow worker may know which isotope was used, or this may need to be determined by a health physicist in the laboratory.

Initial patient survey and nasal swabs. The initial external survey of the body can be performed using standard RADIACs with both beta-gamma and alpha probes. Nasal swabs collected before decontamination can help diagnose, but cannot exclude, a significant inhalation injury (because the nares are self-cleaning). These swabs should be collected early, in the first hour and before the patient is washed off or showered, but decontamination should not be delayed just to obtain nasal samples.

Each nostril should be swabbed separately, and the radioactivity on each swab measured using a RADIAC. The swabs should be saved. Activity measured by a RADIAC on a nasal swab reflects lung deposition. Health physicists can calculate the lung burden of the contaminant using standard equations and extrapolation curves. If both nostrils are "hot," the patient probably inhaled contamination. If only one nostril is hot, the patient either has touched his nose with a contaminated finger (suggesting no lung contamination), or there is unilateral nasal obstruction.

Measuring internal contamination. The laboratory tests most familiar to medical personnel (e.g., CBC and chemistry panels) are not helpful in diagnosing internal contamination. Instead, internal contamination is measured either directly with RADIACs or indirectly using samples of body fluids and excreta.

Direct measurement. Machines such as RADIACs and larger stationary units such as whole-body counters directly measure radioactivity of the body. The operator sweeps the RADIAC slowly over the body, maintaining a constant distance above the skin. A whole-body counter is a large fixed device, associated with a heavily shielded walk-in or lie-down chamber. This is the most reliable diagnostic instrument, although availability is limited.

Indirect measurement. Body fluids (obtained by swabs), tissue samples, and excreta can be directly examined for radioactivity and for the specific radioactive isotopes involved. A health physicist can then estimate the patient's "body burden" using extrapolation curves.

Nasal swabs should be obtained as soon as possible after contamination, as noted above. These are sent with the patient to the medical facility. Medical or health physics

personnel should also take a wipe sample from the surface of wounds or skin wherever hot spots are noted via RADIAC.

The level of radioactivity in urine and feces can also be used to estimate internal contamination. Initial radioactivity in urine and feces should be quantified with a baseline sample, followed by multiple postexposure urine and fecal samples. A thorough evaluation of serial samples is the only sure method of confirming contamination and quantifying body burden, which are key components of successful treatment.

Bioassay methods vary according to each nuclide. Published methods can be found in health physics texts and standard guidance documents (e.g., NCRP Report No. 65). Table 6.7 summarizes the sampling regimens for some important radionuclides. Biodosimetric approaches are useful for insoluble nuclides that are either ingested or inhaled, because inhaled particles will be swallowed and passed through the GI tract into feces.

Tritium exposure can be measured with a single voided urine sample. For uranium, a baseline 24-hour urine sample should be collected as soon as possible after the exposure. This establishes the pre-existing excretion of uranium commonly distributed in soil. This initial urine sample is then followed by another 24-hour sample to measure new contamination and an additional 24-hour sample 7–10 days later. Insoluble plutonium may not appear in the urine until 2–3 weeks after ingestion, so both urine and fecal bioassays are needed to identify this radionuclide.

Radiation Detection, Personal Protective Equipment, Personnel Monitoring, and Decontamination

Detection Using Radiation Survey Meters

Various instruments are available for detecting and measuring radiation, which cannot be detected by human senses. These instruments (Figure 6.8) use the energy of the radiation to create an electrical "pulse," which can then be measured and used to determine information about the radiation field. These various Radioactivity, Detection, Indication, and Computation meters are generally referred to by the acronym RADIAC**.**

Radiation detectors can be configured in several different ways. Some have an internal probe, while others have an external probe connected by a wire. The intended use of the meter is the most important factor in determining what configuration to use. If the meter will be used only for large area surveys, an internal probe works fine. If the meter will be used to determine the dose rate from a point source or to detect contamination, then an external probe will most likely be used.

Things to check before using a RADIAC:

- Check to see if the meter is in calibration. Most calibrations are good for 1 year, but always refer to the sticker on the meter, which indicates when calibration is due.
- Inspect for physical damage (broken meter face, frayed cables, etc.).

- Check the batteries (most RADIACs are battery powered). This is normally done by either pushing a button labeled BAT(T), or turning the switch to BAT and watching the meter. If it moves to within the designated area, it is satisfactory. On the newer digital meters, a self-diagnostic may run as soon as the meter is switched on, and the battery level will be displayed.
- In all cases, follow the procedures approved at your facility for use of these instruments.

There are two main classes of RADIAC instruments used to detect and survey for ionizing radiation: gas-filled and scintillation detectors.

Gas-filled detectors. The first and oldest type of RADIAC is the gas-filled detector. These are constructed by filling an electrically conductive chamber with an inert gas. The system contains two electrodes, one through the center of the chamber and the chamber itself, which also functions as an electrode. When a voltage is applied to the system, the center wire becomes positively charged (anode) and the outer (chamber) electrode takes on a negative charge (cathode). The fill gas, usually a noble gas such as argon, helium, or neon, is ionized by the radiation that strikes and penetrates into the chamber. This produces a pair of ions (a positive base ion and a free electron), which are attracted to the electrode of the opposite charge. The free electrons that are collected along the anode then create an electrical impulse. The electrical impulse (or current) is measured and converted to a meter reading, generally in counts/min or mR/hour.

As the amount of voltage applied to the tube increases, the attractive forces of the anode and cathode get stronger and the production of secondary ionizations increases. Secondary ionizations are those not caused by the incident radiation, but by multiplication within the inert gas (gas amplification). There are six distinct regions corresponding to the amount of voltage applied.

Detectors are described according to the radiation regions they detect. The regions most commonly used for detection are the Ion Chamber Region, the Proportional Region, and the Geiger-Mueller Region.

- Ion chambers operate at low voltages, which do not produce any gas multiplication. The applied voltage is just enough that the ions migrate to their opposite poles; without secondary ionization, they travel through the gas. This means that only the primary ions created by the initial radiation event are collected, so that one radiation interaction produces one count. The ion chamber is very good for exposure measurements but not for detection. It is therefore used mainly as a dosimeter (see below).
- Proportional counters rely on gas multiplication to amplify the charge created by the original ionization in the gas. The pulses they produce are therefore considerably larger than those from ion chambers used under the same conditions. This allows proportional counters to be used when the number of ion pairs is too low for effective detection by an ion chamber. Proportional counters are widely used in detection of low energy x-rays and neutrons. Because the current signal in a proportional counter is “proportional” to the energy of the radiation, these counters can also be used for spectroscopy of low energy x-rays.

- Geiger-Mueller or G-M counters operate at a significantly higher voltage than either ion chambers or proportional counters. They produce a large signal due to the "avalanche effect," in which one initial event can cause millions of subsequent secondary ionizations. Due to this effect, the signal is not proportional to energy, and the G-M detector cannot discriminate between different types and energies of radiation. Additionally, at high radiation levels, G-M counters will experience dead time, in which an ionization event is lost because it occurs too quickly after the preceding event. At high count rates, these dead time losses can become severe, requiring a correction to ensure accurate measurements. The G-M instrument is very good for detection but not so good for measurement of exposure. That is to say, it can accurately confirm contamination on a patient, but it cannot quantify it accurately. The probe is held approximately 0.5 inches off the surface and moved at a rate of 1–2 inches/second.

Scintillation detectors. The other common type of detector is the scintillation detector, which uses a liquid or a solid phosphor crystal. When radiation strikes the crystal, the energy of the incident radiation is expended to produce ionization and excitation. The ion that was formed eventually recombines, or the excitation decays, both of which result in the production of light. On average, the number of light photons emitted is proportional to the amount of energy originally deposited. A photomultiplier tube is then used to convert the scintillation photons (light) into an electrical pulse, which is counted or otherwise analyzed electronically. Some common scintillators use zinc sulfate, sodium iodide, or plastics. To detect alpha particles, scintillation detectors must be held $1/16^{th}$–$1/8^{th}$ of an inch from the surface being monitored because of the short range of alpha radiation.

The so-called multi-function RADIACs represent a modern innovation in radiation monitoring. These instruments use one readout unit, which can be connected to various probes so that most types of radiation and contamination can be detected. Most of these units use what is called a "smart box," which can detect the type of probe being used. These "smart" units automatically show the correct units for the monitor being used and calibrate themselves accordingly. Many of these units come with everything in one case, eliminating the need to carry many separate detectors.

Dosimeters

Dosimeters are a special type of instrument used to determine the total dose of radiation that a person receives. The simplest dosimeters are made of film (like camera or x-ray film), tiny wafers of plastic, or other specially formulated materials that respond to radiation in ways that can be assessed and matched to a particular radiation dose. Simple dosimeters require special processing to determine dose but are very reliable and accurate.

Slightly more complex are pocket ion chambers, which use the ionization caused by radiation to move a very tiny fiber that tracks the dose. Pocket chambers are inexpensive and can be read directly, but they are not very accurate and have to be protected from bumps that might cause the needle to move.

The standard dosimeter for most radiation workers, including medical personnel, is some form of thermoluminescent device (TLD). These use some type of crystal, such as lithium fluoride (LiF), calcium sulfate ($CaSO_4$), or calcium fluoride (CaF) as the detector element. These dosimeters are usually read by placing them in a machine that heats the crystal and reads its light output, which is proportional to the radiation dose received. An advantage of some TLDs is that one dosimeter can be used for beta, gamma, x-ray, and neutron radiation. Another advantage of the LiF dosimeters is that they are relatively resistant to "fade" (i.e., the loss of dose over time) and are sensitive down to 1 mrem (new LiF-Cu version).

Finally, electronic dosimeters have become popular. Most are about the size of a personal pager. These are more costly than pocket chambers but are much more reliable, accurate, and rugged. They also have a variety of features (e.g., alarms, digital readouts, dose rate readout, etc.) that can make them attractive to many first responders.

Personal Protective Equipment

This discussion describes many aspects of personal protective equipment (PPE), including the following:

- The need for PPE.
- The type of PPE needed for radiation contamination.
- How PPE for radioactive isotopes is different from PPE for chemical or biological exposure.
- Temporary PPE in an emergency.
- Disposal of PPE.
- Department of Energy Guidance on PPE.

Need for PPE. Radioactive isotopes in the form of dust particles can harm a victim via external or internal contamination. Radioactive contamination can be carried by air currents in the form of fallout, which may travel great distances or settle on surfaces locally, depending on climactic conditions. In war-torn countries, children can potentially be contaminated externally by radioactive particles on the skin or clothing or by playing on vehicles that have been destroyed by depleted uranium munitions. Terrorist attacks with RDD (dirty bombs) can also generate considerable risk of contamination. PPE can protect a health care provider from contamination when dealing with casualties from such events.

In internal contamination, radioactive material enters the body via inhalation, ingestion, or wound penetration. A small amount of radioactive material could also enter the body through the mucus membranes of the eyes.

Type of PPE needed for radiation contamination. Personal protective equipment refers to three types of equipment:

- Respiratory protection.

- Protective clothing— Disposable clothing, such as Tyvek® coveralls or something similar, is probably the least expensive option if an organization must purchase a large number.
- Monitoring equipment.

The goal of PPE is to prevent an individual from becoming contaminated and to prevent the spread of contamination from an already contaminated person. PPE does not shield the wearer from penetrating radiation, such as photons.

First responders. PPE should provide protection to the skin, respiratory and digestive orifices, and the eyes. Boots are part of the PPE needed to prevent contamination of footwear with radioactive material. The PPE should be sturdy enough to avoid tearing in emergency situations. If the PPE is not airtight, tape may be placed where the PPE suit contacts gloves and boots to avoid radioactive material from getting inside the suit. A variety of PPE is available commercially. The National Institute for Occupational Safety and Health (NIOSH) provides information and a statement of certification for respirators at http://www.cdc.gov/niosh/npptl/.

Hospital personnel. Radioactive fallout in the air should not be present inside a medical facility. Therefore, medical personnel working inside a facility do not need to wear as much PPE as first responders. Medical personnel who are decontaminating patients or providing emergency care to patients who have not yet been decontaminated should wear PPE similar to that worn in the operating room (e.g., head cover, face mask, scrubs or gown, and shoe covers). Wearing double gloves is recommended, in case the outer glove tears or breaks.

Differences in PPE for radioactive isotopes and chemical or biological exposure. First responders should wear PPE appropriate for "all hazards," because the type of hazard may not be initially known. However, radioactive material is easier to identify than chemical or biological material, because it can be readily identified with a RADIAC. If the hazard is identified as solely radiological, then first responders can reduce their level of protection to that necessary for radioactive material (e.g., self-contained breathing apparatus is not usually needed). Waterproof PPE is appropriate if the contaminant is wet. Otherwise, cotton or Tyvek® coveralls would be adequate.

Temporary PPE in an emergency. A person without PPE in a radioactive environment should take immediate emergency precautions, including the following:

- Cover nose and mouth with a handkerchief.
- Try to cover exposed skin.
- Move away from the event.
- Take shelter as soon as possible.

Adults should direct children to do the same and lead the children to a safe area.

Disposal of PPE. PPE may have radioactive contamination after an event. All such PPE should be collected in one area, placed in plastic bags (preferably red or yellow), and

labeled as radioactive material. A health physicist can later evaluate the material for the presence and amount of radioactivity and then advise if the PPE can be washed without radiation precautions, should be disposed of, or should be stored in a special manner.

DOE guidance. The Department of Energy Web site "Radiation Emergency Assistance Center/Training Site (REAC/TS)" at http://orise.orau.gov/reacts/rad-incident-response.htm provides guidance on proper use and disposal of PPE. Key information is summarized below.

- The purpose of PPE is to keep bare skin and personal clothing free of contaminants. Members of the radiological emergency response team should dress in surgical clothing (scrub suit, gown, mask, cap, eye protection, and gloves) and use waterproof shoe covers. A waterproof apron can also be worn by any member of the team using liquids for decontamination purposes. All open seams and cuffs should be taped using masking or adhesive tape. Fold-over tabs at the end of each taped area will aid in removal.
- Two pairs of surgical gloves should be worn. The first pair of gloves should be under the arm cuff and secured by tape. The second pair of gloves should be easily removable and replaced if they become contaminated.
- A radiation dosimeter should be assigned to each team member and attached to the outside of the surgical gown at the neck where it can be easily removed and read. If available, a film badge or other type of dosimeter can also be worn under the surgical gown.
- The above PPE effectively stops alpha and some beta particles but not gamma rays. Lead aprons, such as those used in x-ray departments, are not recommended, because they do not stop most gamma rays and give a false sense of security.

Monitoring of Personnel/Decontamination

The DOE Web site "Radiation Emergency Assistance Center/Training Site (REAC/TS)" at http://orise.orau.gov/reacts/rad-incident-response.htm also presents guidance on recommended procedures for monitoring of personnel (Figure 6.9).

The various aspects of decontamination include the following:

- Basic principles.
- How clean is clean?
- Techniques.
- Ambulatory versus non-ambulatory.
- Wet versus dry.

Basic Principles

The primary objective of skin decontamination is to prevent internal contamination through secondary ingestion or inhalation. The secondary objective is to minimize the radiation dose to the skin. Each potentially exposed person should be monitored. Contamination levels should be recorded in the appropriate location on a form containing an anatomical figure.

Decontamination priorities are as follows:

- Do not impede life-saving medical care.
- Decontaminate wounds first, then cover with waterproof bandage.
- Decontaminate areas where risk of internal contamination is high (i.e., the face).
- Decontaminate areas of greatest contamination.
- If multiple personnel are contaminated, decontaminate personnel first who may be most valuable in combating the casualty.

Simply removing clothing may eliminate up to 95% of contamination.

Treatment of life-threatening injuries always takes precedence over decontamination. No U.S. health care worker has ever suffered a radiation injury from caring for a contaminated patient.

How clean is clean? An individual is generally considered "clean" if all areas of the body are below 100 counts/minute above background.

Decontamination Techniques

For a conscious patient, the first step is an interview. Ask for a description of the contamination event (to determine likelihood of internal contamination), extent of skin contamination, and number of other people affected. The next step is decontamination of eyes, ears, nose, skin, and injury site, which should be performed (or supervised) by medical personnel familiar with decontaminating these body areas.

A number of techniques have proved quite effective for decontamination of radioactive material. The least invasive, or least potentially damaging, technique should be done first before moving to more aggressive techniques. The need for decontamination must be balanced with potential injury to the skin caused by aggressive decontamination methods. The individual should be monitored after each decontamination attempt.

Dry particulates. For a dry particulate or dust, the "tape-press method" has proven to be effective. In this procedure, an adhesive tape (e.g., masking tape) is pressed onto the contaminated area to lift off the contaminant. Strongly adhesive tape (e.g., duct tape) should not be used, because it may damage skin. The tape-press method should not be used on hairy areas or fragile tissue, such as eyelids.

Washing. Washing a contaminated area with soap and water for 1–3 minutes is very effective. The goal of this type of decontamination is to float the contaminants off the skin and rinse them away. This can be done in a basin for small areas of contamination, or in the shower if large portions of the body are contaminated. If plain soap and water are ineffective, then a mild abrasive soap (such as Lava® or Soft Scrub®) may be used. If only a small area of skin is contaminated, waterless hand cleaner may be used instead of soap and water.

During washing, care must be exercised not to splash contaminated wash solution around the area. A washcloth or soft surgical scrub brush may be used to help remove

contamination. The water should be lukewarm, and care must be taken not to damage the skin. If erythema becomes evident, decontamination should be stopped to prevent driving contaminants into the skin.

If washing is not effective, the site should be wrapped or covered with a bandage to allow removal through sweating and skin sloughing. Both the contamination site and the bandaging material should be monitored after 6–9 hours to determine effectiveness of this technique.

Wounds. Irrigation with sterile water or saline has proven to be effective for wound decontamination. If the wound is a mild burn or abrasion, gentle washing with soap and water may be effective. All contamination does not have to be removed, because the residual remaining on the surface will normally be incorporated into, and sloughed off with, the scab. For contamination remaining in a puncture wound, simple wet debridement may be used following standard surgical procedures. However, before any surgical procedure, careful consideration should be given to the risks/benefits of the procedure.

Medical Treatment: General Issues Unique to Pediatrics

Acute Susceptibility

The clinical manifestations of radiation injury in children are generally similar to those in adults. However, there are a number of characteristics that render the pediatric patient uniquely sensitive to the effects of radiation exposure (see also Chapter 1, Children Are Not Small Adults).

Children have a greater body surface area to weight ratio than adults and skin that is more permeable and less keratinized, which renders them more vulnerable to both thermal and radiation burns. The inability of young children to shield their eyes also makes them more susceptible to ocular injury from blast, radiation, and thermal effects. This latter problem is compounded by the increased sensitivity of a child's lens to radiation damage. Children have a higher baseline respiratory rate than adults and also exist in a lower breathing zone, which makes them more vulnerable to both generalized inhalation exposure and particulate exposure from radioactive fallout. Children also have a lower intravascular volume reserve, rendering them more susceptible to dehydration from the GI losses encountered in acute radiation syndrome (ARS).

Infants and young children are also more likely to engage in pica of radioactively contaminated materials in their environment. In addition, radioiodine, a common byproduct of nuclear reactor activity, is efficiently transmitted through both human breast milk and cow's milk, which are staples of the childhood diet.

Long-Term Susceptibility

The well-documented long-term effects of radiation exposure to the fetus and child are potentially of even greater concern for the more broadly exposed pediatric population.

These effects can occur anywhere from months to years after initial exposure. The collective experience from the events in Hiroshima and Nagasaki, the Marshall Islands, and most recently Chernobyl, clearly show that children are more susceptible to developing many more long-term consequences of radiation exposure than adults over the same latent period. The most well-studied of these outcomes is the increased incidence of thyroid cancer in children after the Chernobyl accident in Belarus, Ukraine, and the Russian Federation. These thyroid cancers began within 4 years of exposure and continue to the present. Thyroid cancer incidence was dramatically increased almost exclusively in those younger than 20 years of age at the time of exposure, underscoring the unique susceptibility of the pediatric thyroid gland to radiation-induced malignant transformation.

The incidence of leukemia from Hiroshima and Nagasaki was twice as high in children as in adult survivors. This increased incidence began within 2 years, peaked at 6 years, and then regressed to baseline after 25 years. There was a particularly high incidence of chronic myelocytic leukemia and acute myelocytic leukemia, which are cancers not typically seen in non-irradiated pediatric populations.

In addition to thyroid and hematologic malignancies, the incidence of breast cancer in female Japanese survivors of the atomic bomb was increased among those who were 10 to 19 years of age at the time of exposure compared with those older than age 20. The minimum period of latency appeared to be 10 years after exposure. There was also a surprisingly higher incidence of subsequent breast cancer in girls who were younger than age 10 at the time of exposure, indicating increased sensitivity even in prepubertal girls with little breast tissue.

A number of effects have also been documented from fetal exposure to radiation throughout gestation, such as children born to female survivors in Nagasaki and Hiroshima. These effects include a higher incidence of mental retardation, microcephaly, and postnatal growth retardation, particularly with exposures in the first trimester. Fetal exposure after the first trimester was associated with a significantly greater risk of development of leukemia and thyroid cancer.

Psychological Vulnerability

Children involved in a radiation-related incident will be particularly vulnerable to psychological trauma, as in any disaster or terrorist event. Depending on the child's stage of development, this increased vulnerability can manifest as generalized fear and anxiety, developmentally regressive behavior, sleep and appetite problems, altered play, school problems, or greater dependence on caregivers. This latter problem may be exacerbated by physical separation from parents in the chaos of the event. Repetitive television and news broadcasts relating to the event may even traumatize children in areas remote from the actual disaster, convincing them that they are also at risk.

Children also experience stress by witnessing the reactions of their parents. In one survey of adults exposed to the Chernobyl accident, the group displaying the highest level of psychological distress were mothers with children younger than 18 years of age. This

finding has relevance to children, because a direct correlation exists between a parent's response to a disaster and the response of the child.

Any treatment plan regarding children exposed to radiation should take these unique vulnerabilities and parental reactions into consideration. Although many of the psychological reactions of children involved in a radiation-related incident are to be widely expected as an initial response, severe reactions or those persisting beyond several weeks may prompt the need for involvement by pediatric mental health professionals. See also Chapter 8, Mental Health.

Immediate Care

The first priority during the care of anyone exposed to radiation is to treat life-threatening injuries before addressing radiation exposure and contamination. In general, evolving injuries such as burns, lacerations, and fractures need to be stabilized before decontamination and subsequent transport to facilities where radiation-specific injuries are managed. In most instances, radiation levels will not be known, and survey instruments may not be available. Contamination risks to medical responders will be minimal in most cases, unlike situations involving biological and chemical exposures. However, simple precautions such as wearing gloves and wrapping victims in sheets or blankets to reduce the spread of contamination should be done before transport.

Medical Treatment: Acute Radiation Syndrome

Treatment of acute radiation syndrome (ARS) includes both general supportive care and specific actions and medications. No one is known to have survived whole body doses of radiation exceeding about 8 Gy. Therefore, those receiving doses of greater than 8 Gy can be considered expectant, although they may survive for a few months with extensive medical care. ARS is usually caused by direct radiation exposure, not internal contamination.

Supportive Care

Supportive therapy is a key factor in minimizing morbidity and mortality with significant whole body exposure, regardless of the type of radiation. A baseline history should be obtained before or shortly after supportive therapy is initiated. The history should include information regarding the source of radiation, the duration of exposure, the interval between exposure and presentation, and the physical property of the radioactive compounds (e.g., solid, liquid, particulate). The review of systems and physical examination should be as complete as possible, particularly focused on those organ systems that show early signs of damage, such as the skin and the hematopoietic, GI, and neurovascular systems.

The first step is to make sure the victim has been medically stabilized and decontaminated, and that appropriate samples have been obtained for biological dosimetry. Next, the first symptoms likely to occur during the prodromal phase, such as nausea, vomiting, and diarrhea, should be addressed. Treatment of these early

manifestations of ARS may range from minimal intervention to the use of parenteral fluids and antiemetic agents. Antiemetic agents include ondansetron or granisetron at dosages commonly used to prevent nausea and vomiting during chemotherapy. For children 4–18 years of age, ondansetron is usually given at 0.15 mg/kg tid, and granisetron at 10 μg/kg/day. However, antiemetics may be contraindicated initially, particularly if catharsis is deemed necessary for internal decontamination.

During the first day, the focus should be not only on treating acute symptoms, but also on systematically recording them as a way of estimating the dose involved. When combined with other biodosimetric indices, the time to onset of clinical symptoms is particularly useful in determining prognosis and the extent of supportive care that may be needed much later in the clinical course.

Nutrition. Maintenance of adequate nutrition is important to counter the catabolic effects of radiation and to allow healing and recovery. Oral feeding is preferred if possible to maintain functioning of the intestinal mucosa and reduce risk of infection from parenteral feeding. Parenteral feeding may be necessary if the patient is not able to tolerate oral feedings or if fluid loss is profound due to diarrhea.

Hematopoietic syndrome. The main focus of supportive therapy in patients with survivable exposures between 1 and 8 Gy centers around the hematopoietic system. Hematopoietic syndrome may not become clinically apparent until after a latent period of 2–4 weeks. A key feature of this syndrome is neutropenia.

Established or suspected infection after exposure to radiation is usually characterized by neutropenia and fever. In general, such patients can be managed the same as other febrile neutropenic patients. However, important differences between the two conditions exist. Most individuals exposed to irradiation are otherwise healthy. They also generally are exposed to total body irradiation without the shielding that is used in therapeutic exposure to irradiation. Patients with neutropenia after radiation are also susceptible to irradiation damage to other tissues (e.g., lungs, CNS, etc.), which may require therapeutic interventions not needed in other types of neutropenic patients. Furthermore, irradiated individuals may respond to antimicrobial therapy in unpredictable ways, as has been shown in experimental studies in which metronidazole and pefloxacin therapies were detrimental to irradiated laboratory animals.

Neutropenia: Cytokine Therapy

Hematopoietic growth factors, such as granulocyte colony stimulating factor (G-CSF) and granulocyte macrophage colony stimulating factor (GM-CSF), are not FDA approved for the management of radiation-induced marrow aplasia. However, these growth factors have been used in the aftermath of a number of radiation accidents (e.g., Chernobyl and Goiania) in an effort to lessen the severity and duration of neutropenia and thereby reduce the risk of infection. The experience with these few clinical cases, along with even more convincing data from controlled studies in animals, suggest that early cytokine therapy can significantly enhance survival. Benefit is maximal if treatment begins within the first 24–72 hours after radiation exposure. In the absence of significant complicating injuries,

cytokine therapy is recommended for radiation doses ≥3Gy in adults, and >2Gy in children. Practical limitations, such as limited availability and supply of cytokines, need to be considered when determining the level of exposure that indicates this treatment.

None of these cytokines is approved for the specific indication of radiation-induced illness. The long-acting form of G-CSF (pegfilgrastim) is FDA approved as a one-dose therapy for the management of chemotherapy-induced neutropenia in adults and adolescents weighing more than 45 kg. It is not approved for younger children and infants. The other two FDA-approved cytokines are G-CSF (filgrastim) and GM-CSF (sargramostim). These are administered daily until the absolute neutrophil count reaches >1,000. Dosages for adults and children are 5 μg/kg/day for G-CSF and 250 $\mu g/m^2$/day for GM-CSF.

Neutropenia: Antibiotic Therapy

Each institution or agency should follow established guidelines to develop a standardized plan for the management of febrile, neutropenic patients. Antimicrobials should be used mainly in radiation victims who develop fever and neutropenia. An empirical regimen of antibiotics should be selected, based on the degree of neutropenia and on the pattern of bacterial susceptibility and nosocomial infections in the particular area and institution (Table 6.8). Broad-spectrum empirical therapy (see below for choices) with high doses of one or more antibiotics should be initiated at the onset of fever. The antimicrobial spectrum should include efficacy against gram-negative aerobic organisms, which account for more than 75% of the isolates causing sepsis. Aerobic and facultative gram-positive bacteria (mostly alpha-hemolytic streptococci) cause sepsis in about 25% of victims, so coverage for these organisms may also be necessary, especially in institutions where infection from these organisms is prevalent. Antimicrobials that decrease the number of strict anaerobes in the gut (e.g., metronidazole) generally should not be given, because they may promote systemic infection and death from aerobic or facultative anaerobic bacteria.

If infection is confirmed by cultures, the empirical regimen may require adjustment to provide appropriate coverage for the specific isolate(s). After the patient becomes afebrile, the initial regimen should be continued for a minimum of an additional 7 days. Therapy may need to be continued for at least 21–28 days or until the risk of infection has declined because of recovery of the immune system. A mass casualty situation may mandate the use of oral antimicrobials.

The initial antibiotic regimen should be modified when microbiological culture shows specific bacteria that are resistant to the initial antimicrobials. Modification, if needed, should also be considered after a thorough evaluation of the history, physical examination findings, laboratory data, chest radiographs, and epidemiologic information. If resistant gram-positive infection is evident, vancomycin should be added. If diarrhea is present, fecal cultures should be examined for enteropathogens (e.g., *Salmonella, Shigella*, *Campylobacter*, and *Yersinia*).

Neutropenia: Viral and Fungal Infections

Antifungal coverage with amphotericin B is indicated for certain situations, including:

- Clinical evidence of infection in patients who remain persistently febrile for 7 days or more on antimicrobial therapy.
- A new instance of fever on or after day 7 of antimicrobial therapy.

Oral and pharyngeal mucositis and esophagitis suggest *Herpes simplex* infection or candidiasis. Either empirical antiviral therapy, antifungal therapy, or both, should be considered in this situation.

Neutropenia: Prophylactic Antimicrobials

Prophylactic antimicrobials should also be considered in any individuals exposed to doses >2 Gy, when the absolute neutrophil count is $<0.5 \times 10^9$ cells/L. Whenever possible, exposure dose should be estimated by biological dosimetry and detailed history of exposure.

Thrombocytopenia and Anemia: Blood Products

Platelets are the blood products most commonly needed for treatment of radiation-induced marrow aplasia, because they have a shorter lifespan than red blood cells and will likely regenerate later (i.e., marrow transplant is not necessary). Prophylactic transfusions have traditionally been recommended when platelet counts are $<20 \times 10^9$ cells/L, although a level of 10×10^9 cells/L in the absence of overt bleeding may be adequate unless surgery is planned. A platelet count $>75 \times 10^9$ cells/L is desirable before surgery.

Red blood cell transfusions are used to maintain a hemoglobin of 80–100 g/L, which provides a sufficient reserve in case of severe bleeding. Limited experience with granulocyte transfusions has not revealed any significant clinical benefit, so it cannot be recommended at this time. All blood products should be leukoreduced and irradiated to 25 Gy to prevent the risk of cytomegalovirus infection and transfusion-associated graft versus host disease, respectively.

Bone Marrow Transplantation

The use of bone marrow transplantation in victims of radiation exposure dates back to the 1950s and on up to the 1999 radiation accident in Tokaimura, Japan. Despite the theoretical benefit of stem cell transplantation, the cumulative experience in these clinical situations (e.g., Chernobyl and Goiania) have not demonstrated any clear lifesaving benefit. However, it is difficult to interpret the results from these events because of the lack of precision with which the radiation exposure was known in most patients and the heterogeneity of comorbid conditions.

From a theoretical standpoint, comprehensive supportive therapy alone can save many patients with exposures <8 Gy, while death from complications of gastrointestinal syndrome is virtually inevitable in those with exposures >10 Gy. Therefore, the decision

to pursue stem cell transplantation will need to be carefully considered for a narrow subset of patients with exposures between 8 and 10 Gy and no other significant comorbidity. The limitations of stem cell transplantation include potential complications (e.g., graft-versus-host disease), potential for morbidity associated with other organ toxicity, and a limited pool of suitable donors. In the rare instance in which a syngeneic donor (i.e., a twin sibling) or previously harvested stem cells are available, stem cell transplantation may be considered for those with exposures of 4–10 Gy. This recommendation applies to both adults and children.

Medical Treatment: Internal Contamination

External decontamination should be performed before treatment of internal contamination, which depends on the chemical nature of the radioisotope, its physical quantity, and the nature of radioactivity. A risk/benefit decision must be made. For small amounts of radioactive material or those with a short half-life, treatment may be unnecessary. Medical providers need to consult health physicists or medical physicists for information about the nature and amount of internal contamination. The National Council on Radiation Protection and Measurements Report No. 65 *Management of Persons Accidentally Contaminated with Radionuclides* is a valuable reference.

Treatment of internal contamination reduces the absorbed radiation dose and the risk of future biological effects. Administration of diluting and blocking agents enhances elimination rates of radionuclides. Treatment with mobilizing or chelating agents should be initiated as soon as practical when the probable exposure is judged to be significant. Gastric lavage and emetics can be used to empty the stomach promptly and completely after the ingestion of poisonous materials. Purgatives, laxatives, and enemas can reduce the time that radioactive materials are present in the colon.

Radioactive Iodine: Potassium Iodide

Thyroid uptake of radioactive iodine can be reduced if potassium iodide (KI) is taken before or shortly after exposure. The main exposure routes for radioiodine are through inhalation and through ingestion of contaminated food, milk, or water. In 1986 during the Chernobyl nuclear reactor disaster, large populations in the proximity of Chernobyl—most notably in Belarus, northern Ukraine, and the Russian Federation—were not initially aware of the danger and were not evacuated, sheltered, or treated with KI. Since 1990, the incidence of thyroid cancer in children has increased significantly. To date, the largest public health action involving the use of KI involved the distribution of 18 million doses to children and adults in Poland after the Chernobyl accident. KI was distributed to 90% of the children younger than 16 years and to about 25% of the adults in Poland from the 4^{th} day to the 7^{th} day after the beginning of the release of radioactivity from the Chernobyl power plant. Although no significant increase in thyroid cancer has been noted in KI-treated populations in Poland, there are several factors that may have contributed to this difference. The dose of radioactive iodine to the Polish population may have been less than that to the population closer to Chernobyl. Also, the children in the Chernobyl area may have been more iodine-deficient than the children in Poland and therefore more susceptible to uptake of the radioactive iodine from Chernobyl.

The main reason for targeting pregnant women and children specifically for KI prophylaxis is that the smaller thyroid gland of the fetus and child concentrates proportionately more radioactive iodine than that of an adult, resulting in the well-documented increased risk of thyroid cancer in this age group. Despite the fact that the fetal thyroid is not functional until approximately 12 weeks gestation, KI administration is still recommended during the first trimester for maternal protection.

Although the potential side effects of stable iodine administration are well-described, during the Chernobyl experience, virtually no major side effects were seen in the 10 million children to whom it was administered. Moderate effects observed in Poland included transient hypothyroidism in 12 neonates and otherwise occasional gastrointestinal symptoms and rash in others. In the adult population of 7 million who received KI, only two severe reactions were noted, both in individuals with an already known allergy to iodine. The incidence and severity of these side effects would be expected to be greater with repeated dosing, but there are minimal data available regarding the true risk associated with repeated KI administration.

The indication for using KI to block uptake of radioactive iodine by the thyroid depends on the predicted thyroid dose and the age of the patient (Table 6.9). KI dosages are also age-dependent. If KI is administered, timing of the dose is of major importance. If given before or within 1 hour of the exposure, >90% blockage of uptake can be expected. After 4–5 hours, there is 50% blockage. After 12 hours, the effect is minimal. Because of the short time period for effective administration, supplies of KI need to be already in place in areas at highest risk of exposure, such as around nuclear power plants from which high levels of radioiodine might be released.

Because of the expected increased risk of side effects with repeated dosing, particularly in pregnant women and neonates, the decision to repeat KI administration because of ongoing unavoidable exposure needs to be made at a broader public health level, carefully considering the risk-benefit ratio. Regardless of how many doses are given, the thyroid function of the newborn should be monitored through a TSH level 2–4 weeks after administration, and thyroid hormone should be given to those found to be hypothyroid. Because of the risk for transmission to the child from breast milk, nursing should be interrupted until public health authorities recommend resumption.

KI is stockpiled as tablets for logistical reasons, but it may be difficult for very young children to take the medication in this form. When dissolved in water, KI is very salty; however, a variety of liquids such as raspberry syrup, low-fat chocolate milk, orange juice, or flat soda such as cola make the taste more acceptable. Although KI pills are available over-the-counter, KI is also available in prescription form as potassium iodide oral solution, USP (saturated) (SSKI) containing 1,000 mg/mL. SSKI has such a high concentration of KI that accurate dose titration for children would be complicated by dilution calculations. For detailed instructions for home preparation, see http://www.fda.gov/cder/drugprepare/kiprep.htm.

Radioactive Cesium and Thallium: Prussian Blue

Ferric hexacyanoferrate, also known as Prussian blue, is a prescription medication approved by the FDA for the treatment of internal contamination with cesium or thallium. Prussian blue is taken orally, is not absorbed by the gastrointestinal tract, and serves as an ion exchanger during the enterohepatic cycling of these isotopes. During the Goiania incident, 249 individuals were exposed to cesium-137 from an abandoned medical radiotherapy device. Forty-six of these individuals were treated with Prussian blue, including 13 children who received daily doses up to 3 g/day in 3 divided doses. Prussian blue treatment reduced the whole-body effective half-life of Cs-137 by 46% in adolescents and by 43% in children 4–12 years of age.

Dosage and administration of Prussian blue:

- Adults and adolescents: 3 g, PO, tid.
- Children (2–12 yr): 1 g, PO, tid.
- Dosing in infants and neonates has not been established.

Treatment should start as soon as possible after contamination is suspected and continue for a minimum of 30 days.

Prussian blue is manufactured as a 500-mg gelatin capsule and is available directly from the manufacturer, Heyl, in Germany. Supplies are also maintained by the U.S. Strategic National Stockpile and are available from the Radiation Emergency Action Center/Training Site (REAC/TS), Oak Ridge, TN. Significant side effects are rare and consist mainly of mild to moderate constipation.

Radioactive Plutonium, Americium, and Curium: DTPA

Uranium: Bicarbonate

Diethylenetriaminepentaacetate (DTPA) is a powerful chelating agent effective in removing some heavy metal isotopes. In 2004, the FDA approved calcium-DTPA and zinc-DTPA for treatment of individuals with known or suspected internal contamination with plutonium, americium, or curium to increase the rates of elimination. These chelators, however, should not be used to decorporate for uranium because of potential renal toxicity. Instead, for uranium, urine alkalinization with bicarbonate should be done in an effort to promote excretion (Table 6.10).

Dosage and administration of DTPA. There are two salts of DTPA, Ca-DTPA and Zn-DTPA. In general, treatment should be started with Ca-DTPA on the first day and then changed to Zn-DTPA on the second day. This is because Ca-DTPA is more effective than Zn-DTPA on the first day, while being no more effective than Zn-DTPA later on. It does, however, have increased risks of causing metabolic abnormalities. It is supplied as 1 g in 5 mL of diluent, and is currently available from the U.S. Strategic National Stockpile and from the Radiation Emergency Action Center/Training Site (REAC/TS), Oak Ridge, TN, and directly from the manufacturer in Germany.

No significant side effects from its usage have been reported, although cases of nausea, vomiting, diarrhea, chills, fever, pruritus, and muscle cramps have been noted in the first 24 hours after administration when given repeatedly and with short intervals allowed for recovery.

Adults and adolescents. The initial dose of Ca-DTPA is 1 g IV. On day 2, start Zn-DTPA at 1 g as daily maintenance dose. Pregnant women should begin and continue with Zn-DTPA. Nursing mothers should not breastfeed if they are suspected of having internal radionuclide contamination.

Children younger than 12 years of age. The initial dose of Ca-DTPA is 14 mg/kg IV. On day 2, start Zn-DTPA at 14 mg/kg as daily maintenance dose.

Other Radioactive Isotopes

Because every element has one or more radioactive isotopes, there are hundreds of different radioactive isotopes. Each radioactive isotope behaves chemically the same as the non-radioactive isotope of the same element. Treatment of internal radioisotope contamination depends on the specific chemical properties of each isotope. The main radioactive isotopes that have caused internal contamination in people are described above. Detailed information about the diagnosis and treatment of these and other radioactive isotopes is available in the National Council on Radiation Protection Report No.65 and also by calling REAC/TS or the Armed Forces Radiobiology Research Institute (AFRRI).

Surgical Issues

Local Radiation Injury: Cutaneous Radiation Syndrome (CRS)

A complication of external contamination or of close contact with a radioactive source is the effect of local radiation on tissues and organ systems (see Figure 6.10). This scenario along with partial body exposure has occurred more often than incidents involving whole body irradiation in past radiation mishaps. Local radiation injury will also alter the clinical course of patients suffering from acute radiation syndrome (ARS). In fact, significant morbidity and mortality in these cases may result from local injury rather than from ARS itself. The damage to tissues results in the release of endogenous toxins into the circulatory system, resulting in high body temperatures, metabolic disorders and neurological complications.

The basic pathophysiology involves the response of the various cell types of the skin and organs that are exposed to radiation. That response is a function of the relative radioresponsiveness of the tissues involved. Individual cell types respond differently based on the amount of radiation delivered. The individual cellular response to radiation is also a function of its lineage, its level of development or maturity, and the stage during cell division when it is irradiated. Cell lineage is important because some cells are more radiosensitive (e.g., erythroblasts) than others (e.g., muscle cells). The developmental stage during which a cell is irradiated also is an important determinant of cellular

response. Precursor/stem cells are more sensitive than fully developed, mature cells. Similarly, cells that are actively replicating (going through mitosis) are also more responsive.

After receiving a radiation dose, a progressive, chronic, and complex inflammatory process begins. The clinical course is a function of the following:

- Type of radiation.
- Inherent energy of the source.
- Dose and the dose rate.
- Length of exposure.
- Tissue involved.
- How the energy is distributed within that tissue.
- Size and area of the body involved in the exposure.

Although only a small superficial area may initially appear to be affected, because of the amount of energy involved, deeper tissues and organ systems may also be affected. Depending on the individual cells involved, onset of clinical symptoms will be variable. Perhaps the most pertinent rule of thumb in these types of injuries is that there is no pathognomonic sign or symptom of radiation injury. There is not always a specific linear relationship between the dose of radiation that a tissue receives and the subsequent somatic manifestations that result. Skin damage evolves over time according to the local dose with the tissue furthest from the direct local injury being the slowest to display damage. The extent of tissue damage or involvement is inversely proportional to the square of the distance from the source of radiation. There is no definite correlation between specific symptoms and cell types.

CRS has been divided into five time-related stages: prodromal erythematous, manifestational, subacute, chronic, and late.

Prodromal erythematous stage. This stage may last minutes to hours after exposure to doses >5–6 Gy. The time to onset, intensity, and duration are used to predict prognosis. The early erythema is likely due to release of vasoactive amines and secondary vasodilation. A clinically asymptomatic latent period may follow. At this stage, high-dose and low-dose casualties cannot be distinguished. If additional symptoms (e.g., nausea, vomiting, CNS changes) develop along with relevant patient history, then further assumptions or conclusions will be possible.

Manifestational stage. After a latency period of 7–21 days, clinical signs develop that range from bright erythemas with a burning sensation to painful blisters and ulcers. These changes are due to injury to blood vessels and underlying connective tissue and death of skin stem cells. From 8 to 12 Gy, there is dry desquamation, and from 15 to 20 Gy, moist desquamation ensues. Moist desquamation occurs less commonly in children undergoing radiation therapy than in adults. This is probably because of the ability of the epithelium to recover more quickly in children than in adults. More changes are also observed in fair-skinned individuals and at the more radiosensitive areas of the body such as the

axilla, groin, and skin folds. Radiation sensitivity in other areas of the body decreases in the following order:

- Inner neck.
- Antecubital, popliteal, flexor surface of extremities.
- Chest.
- Abdomen.,
- Face.
- Back.

Least sensitive areas are the nape of the neck, scalp, palms, and soles. Dry desquamation usually leads to complete recovery. However, recovery from moist desquamation depends on the extent of injury.

Subacute stage. The subacute stage is characterized by initiation of progressive dermal and subcutaneous fibrosis leading to a second ulcerative phase and cutaneous ischemia in affected areas.

Chronic stage. Onset is usually from 16 weeks to 2 years after initial irradiation with epidermal atrophy and erosions associated with dermal and subcutaneous fibrosis being the main clinical manifestations. Concomitant inflammation tends to progress indefinitely with no endpoint. As such, long-term evaluation and management may be required.

Late stage. This occurs 10–30 years after irradiation in the exposed field with development of "spontaneous" angiomas, keratoses, ulcers, and squamous and basal cell carcinomas.

Treatment and Management Issues

There are no specific protocols published to guide treatment. However, treatment of past victims provides a valuable point of reference for treating future victims. The clinical situation helps guide whether the treatment approach should be conservative or surgical, based on the following guidelines:

- Radiation ulcer and localized necrosis without signs of regeneration is the primary and most often encountered indication for surgical intervention, as well as severe pain. The more rapid the progression of the pathologic process, the more severe the injury is, and the earlier the intervention should be performed.
- Resection of injured and dead tissue must be accomplished to permit an effective engraftment. Erythematous tissue surrounding frank ulcers is likely to ulcerate if traumatized.
- Antihistamines and topical antipruritic agents may be used for the relief of symptoms. Antihistamines may also prevent or weaken the subsequent inflammatory process.
- Use of high-dose systemic glucocorticoids, combined with topical class III or IV steroids, should be considered.
- Potential opportunistic pathogens (i.e., bacteria, fungi, and viruses) should be treated.

- Necrotic areas should be carefully debrided.
- Treatment of radiation fibrosis with pentoxifylline (to improve blood flow) and vitamin E should be considered.
- There is no standard treatment for radiation fibrosis. Consensus has been to attempt to treat patients who have a subacute onset of radiation injury with corticosteroids if they have symptoms. Other agents include pentoxifylline, vitamin E, colchicines, penicillamine, interferon-gamma, and pirfenidone.

Trauma and Radiation (Timing of Surgery)

The overall morbidity and mortality of trauma patients is exacerbated when there has been acute exposure to ionizing radiation or contamination with radioactive materials. Trauma can be in the form of lacerations, puncture wounds, abrasions, gunshot wounds, blunt force injuries, crush injuries, and burn injuries (see also Chapter 7, Blast Terrorism).

The initial step in the management of victims with combined injuries, i.e., radiation and trauma, should be the immediate stabilization of the most life-threatening injury as well as addressing airway, breathing, and circulation problems. After stabilization, radiation injury can be assessed and further managed. The fundamental concept to appreciate is that radiation injuries are not acutely life threatening.

Thermal burn injuries may be complicated by the fact that the wounds may become contaminated with radioactive particles that need to be removed. Tissue that is irradiated may not respond in the normal physiological manner afterward. This may affect surgical success. Animal studies have indicated that performing initial surgery within 36–48 hours is optimal. Surgery beyond that time puts the patient at risk of life-threatening sepsis due to profound neutropenia with the acute radiation syndrome.

Management of the Patient with Embedded Radioactive Material and Depleted Uranium

Radioactive material embedded in wounds should be removed if possible. Otherwise, a victim is at risk of both infection and local radiation injury. Surgeons should never touch the radioactive particles (even with a gloved hand) due to the high probability of direct permanent damage to the fingers. Particles should be touched only with forceps.

Depleted uranium (DU) munitions may be encountered because they are a critical component of U.S. weaponry. DU has <50% of the radioactivity found in natural uranium. DU is not a radiation risk, but it is a heavy metal risk. Prime concern is its effect on the kidneys as a heavy metal if it is absorbed. However, DU has also been demonstrated to have potential tumorigenic effects in animal studies. Therefore, DU fragments should be removed from wounds if possible, the victim should be kept hydrated, and renal function should be monitored. Sodium bicarbonate helps bind DU and reduce renal toxicity. DU may also be absorbed via inhalation from a DU fire or ingestion.

Followup Care, Including Risk of Carcinogenesis

Children are particularly susceptible to the transforming effects of ionizing radiation. This is true for children exposed to radioactive fallout, release of radioactive materials from nuclear power plants, and external beam radiation therapy for medical conditions. External beam radiation therapy, used for example in the treatment of Hodgkin's disease or CNS tumors, is associated with an increased risk of second malignancies, particularly solid tumors arising in the radiation field. In contrast, children exposed to high levels of radioactive fallout from nuclear weapons at Hiroshima, Nagasaki, or the Marshall Islands testing site have increased risks of leukemia, benign and malignant thyroid neoplasms, and breast cancer (as young women). Those exposed to lower levels of radiation or possibly a more limited number of isotopes, such as from the Chernobyl nuclear accident, have an increased risk of thyroid neoplasms.

Thyroid nodules and cancers are one of the most frequent late complications of ionizing radiation. Although generally uncommon in children, they are very frequent 10–20 years after radiation exposure. Four of the 39 children (10%) exposed after the Bravo nuclear test in the Marshall Islands developed thyroid cancer, and the incidence of thyroid cancers increased by 193-fold in areas contaminated by the Chernobyl nuclear accident. In contrast, only 1 of 39 children (2.5%) developed leukemia after the Marshall Islands nuclear test (compared with 10% incidence of thyroid cancer), and the incidence of leukemia did not increase after the Chernobyl nuclear accident (compared with 193-fold increase in the incidence of thyroid cancer). From these data, it is clear that children are very susceptible to radiation-induced thyroid neoplasia. Indeed, children were found to be 10-fold more likely than adults to develop thyroid neoplasms after exposure to similar doses of ionizing radiation. It is also clear that thyroid neoplasms are a more common late effect from radiation exposure than are leukemias.

Although there may be quantitative differences in the risk of thyroid neoplasia after both of these accidents (Marshall Islands Bravo test and the Chernobyl accident), this could relate to differences in the types of radionuclides released or the doses absorbed. Exact quantification of the doses to which these children were exposed has been difficult to determine because many ingested contaminated milk, which would be a leading source. Quantification of the level of contamination and the quantity of milk consumed has proved difficult. Current studies are underway to better define the individual levels of exposure.

These data underscore the particular susceptibility of the thyroid that arises from unique genetic changes known as recombinant *ret* proto-oncogene in papillary thyroid cancer (*ret*/PTC) rearrangements. These rearrangements can be induced by ionizing radiation and are sufficient to cause thyroid cancer in experimental animals. Once induced, *ret*/PTC gene rearrangements generate a chimeric gene that places the tyrosine kinase portion of the *ret* proto-oncogene under the control of different gene promoters, leading to increased and uncontrolled *ret* tyrosine kinase activity.

Stable iodine prophylaxis can reduce the risks of thyroid cancer after nuclear disasters or accidents but has no effect against external beam radiation therapy because the latter does

not involve radioactive iodine. The World Health Organization and the U.S. Food and Drug Administration recommend stable iodine prophylaxis for exposed populations stratified according to age. Thyroid function tests should be monitored in infants to allow early recognition and treatment of hypothyroidism.

In general, radiation-induced thyroid cancers appear to be more aggressive than "spontaneous" thyroid cancers and are frequently multifocal. Exposed children should be monitored by serial ultrasound examinations, and suspicious lesions should be removed by total thyroidectomy to eliminate other microscopic foci of disease.

Most children who develop thyroid cancer present with a thyroid nodule. Malignancy must be considered in any child with a thyroid nodule because the risk of malignant disease is much higher in children (30–50%) than in adults (10–14%), and it is even higher after radiation exposure. Routine serum thyroid function tests are not helpful in making a diagnosis of malignant disease because they are usually normal even in the presence of malignancy.

Suspicious lesions can be evaluated by ultrasound, which may reveal features highly suggestive of malignancy (such as microcalcifications, heterogeneous echo-density, and central blood flow), in addition to identifying other unsuspected lesions that may also require surgical removal. Radionuclide scans generally cannot distinguish benign from malignant disease and should not be routinely ordered for this purpose. Determination of serum calcitonin levels should be reserved for patients with a family history suggestive of multiple endocrine neoplasia (MEN) because medullary thyroid carcinoma, which is the form associated with MEN and elevated serum calcitonin, is not induced by radiation exposure. Also, there is some risk of false-positive calcitonin levels in patients with any thyroid nodule. Fine needle aspiration cytology is the best single test to distinguish benign from malignant disease but may have a higher false-negative rate in children than in adults unless combined with clinical risk assessment. Due to the high risk of malignant disease, some have recommended removing all thyroid nodules from children, especially those with a history of radiation exposure. Thyroid hormone has been prescribed to reduce the size of benign nodules. Only a few respond, but those that increase in size must be removed.

A logical sequence to evaluate a thyroid nodule would involve ultrasound to define the architectural features and the presence of other unsuspected disease, fine needle aspiration to determine benign or malignant cytology, and then removal if the overall clinical picture is suspicious of malignant disease. Most centers do not rely entirely on the results of a single test such as a fine needle aspirate but would remove the entire thyroid gland due to the increased risk of developing another malignant lesion in any remaining thyroid tissue that was exposed to radiation.

Thyroid cancer is most frequently treated in children with total thyroidectomy, cervical lymph node dissection, and radioactive iodine ablation (RIA). This approach has induced remission in 70% of children but with a significant risk of disease recurrence (19%) and complications (5–25%). Disease-specific mortality is low (1–2%), but this could be

artificial because followup in almost all studies has been brief. After surgery and RIA, thyroid hormone is prescribed to suppress thyrotropin (thyroid stimulating hormone [TSH]) without inducing hyperthyroidism. Routine surveillance for recurrence has generally included ^{131}iodine whole body scans and serum thyroglobulin (Tg) levels performed after thyroid hormone withdrawal. After total thyroidectomy and RIA, undetectable serum Tg when the patient is off thyroid hormone is predictive of complete remission, whereas Tg levels >10 ng/mL off thyroid hormone suppression or TSH-stimulated Tg levels >2 ng/mL indicate residual disease. Despite favorable survival, recurrence is three times as likely in children as in adults.

Due to the increased incidence of leukemia in children exposed to high doses of radiation, close followup with regular physical examinations and complete blood counts is warranted. The incidence of breast cancer is also increased in young women who had been either pubertal or lactating at the time of exposure to the Hiroshima device. Cancers began to appear as early as ages 25 to 30 years, which was earlier than in sibling controls. For that reason, regular breast examinations should be emphasized, and abnormalities should be evaluated with a high level of suspicion. Enrollment in a high-risk breast clinic may be indicated. The latency period (i.e., the time interval between irradiation and appearance of a malignancy) is shortest for leukemia (5–7 years) but can extend to 45 years or more for solid tumors.

Environmental Issues Affecting Children After a Terrorist Incident Involving Radioactive Materials

The environmental damage from a terrorist incident involving radiation has many potential consequences for children. These effects can be minimized by a better understanding of the types of incidents that involve radioactive material and the environmental exposure pathways involved. Response planning involves actions that may be taken to minimize exposure both immediately after and during recovery from a terrorist incident.

Type of Incident

The environmental damage and effects on children differ significantly depending on the type of terrorist incident. More specifically, whether the incident involves a/an:

- Radiological dispersal device (RDD, or “dirty bomb”).
- Improvised nuclear device (IND).
- Attack on a nuclear power plant.

Radiation dispersal device. An RDD is any device that causes the spread of radioactive material across an area, typically to contaminate people and buildings in an urban environment. RDDs can also be used to contaminate water, livestock, fish, and food crops. The radioactive material acts as a toxic chemical that is harmful or fatal. RDDs use an explosive device to scatter the radioactive material over a general but fairly confined area. Simple RDDs spread radioactive material without the use of explosives, typically by the covert placement of radioactive material in a high traffic area. The radioactive

material is then spread when it is disturbed, e.g., by people walking through material spread on the ground or spread on the wheels of vehicles that drive over the material.

Sources[1] of radioactive material for construction of an RDD include medical facilities, military, industrial and waste storage facilities, the black market, and orphaned sources. The vast majority of sources contain only one radioactive element, such as cesium-137, strontium-90, cobalt-60, or iridium-192.[2] Cesium chloride is of particular concern because of the fine powder form in which it is normally used. However, any nuclear material, including nuclear power plant fuel rods (uranium-235) or spent fuel rods (contains a mixture of radioactive components), could be used for dispersal.

The health effects of RDD weapons can vary sharply. The time required to accumulate significant doses of radiation can vary. Harmful effects often require either inhalation or ingestion. Although exposure does not require actual contact with radioactive material, health effects are further reduced as the radioactive material spreads. In most cases, it is extremely unlikely that an RDD can spread enough radioactive material to the air and ground to pose an immediate health hazard to people near the event.

Improvised nuclear device. INDs (improvised nuclear devices) use fissile material, either uranium or plutonium, and produce a nuclear yield. The fission process produces tremendous, potentially catastrophic damage from an initial intense radiation, intense heat, and blast effects over a large area. A plume of radioactive fallout is usually produced, composed of large quantities of a variety of radioactive products. Although fallout is carried by atmospheric conditions and potentially may circle the earth, the heaviest dispersal pattern—and the area of greatest concern for health effects—is the area close to the blast zone. Health effects from an IND include injuries from the blast and heat effects, as well as acute radiation syndrome symptoms from the high doses of radiation released from the nuclear weapons explosion. Depending on the population density, an IND is capable of killing tens to hundreds of thousands of people.

Attack on a nuclear power plant. A terrorist attack at a nuclear power plant could release radioactive material into the environment. Nuclear power plants produce energy through the controlled release of energy from a critical mass of nuclear material by the fission process. Many safety and security features have been designed into U.S. reactors. For example, U.S. reactors use containment structures designed to contain release of

[1] Medical facilities contain high activity irradiators, teletherapy machines and brachytherapy sources, as well as radionuclides which are vulnerable to malevolent use. Military and industrial sources include: radioisotopic thermoelectric generators (RTGs), high activity irradiators, gamma radiography equipment, high activity gauges and well logging equipment, used in drilling or oil, gas and water exploration. Vulnerability to theft or malevolent intent, including sale on the black market, of these sources is increased by factors such as bankruptcy, abandonment, long-term storage, transport, or change in expert personnel. An orphan source is defined by the International Atomic Energy Agency (IAEA) as a radioactive source that poses sufficient radiological hazard to warrant regulatory control but which is not under regulatory control because it has never been so or because it has been abandoned, lost, misplaced, stolen, or otherwise transferred without proper authorization. See International Atomic Energy Agency, IAEA-TECDOC-1388, dated February 2004.

[2] Each radioactive element has its own decay scheme by which it becomes a stable element. Most sources have short decay schemes, with few radioactive intermediates.

radioactive material in an accident. The most serious U.S. nuclear power plant accident occurred at Three Mile Island Unit 2 (TMI) in 1979. The accident at TMI was the worst case scenario of a nuclear power plant accident, namely, a loss of coolant, resulting in the partial meltdown of the reactor core and release of radioactive material into the atmosphere. Although the reactor core at TMI was damaged by excessive heat, the mishap resulted in only very small releases of radioactivity into the surrounding environment. Environmental samples of air, water, milk, vegetation, soil, and foodstuffs at TMI showed that most of the radiation was contained, and that those isotopes released had no physical or health effects on individuals or the environment.

More extensive releases at TMI did not occur primarily because of the containment structure. Containment structures have been included in U.S. reactor designs since the early stages of commercial power plants. They are meant to prevent the release of radioactive material to the area surrounding the power plant if an accident occurs. In contrast, in the reactor accident at Chernobyl in 1986, which was the costliest industrial and environmental accident ever, there was no containment structure. The reactor core at Chernobyl continued to release radioactive material—mainly xenon-133, iodine-131, and, to a lesser extent, cesium-137, strontium-90, and plutonium-239—into the atmosphere for 2 weeks after the accident occurred due to graphite fires that could not be extinguished.

Environmental Exposure Pathways

There are three main routes of exposure by which the health of people are affected by radioactive material:

- Inhalation of radioactive material (e.g., breathing the plume).
- Ingestion of radioactive material (e.g., through food contamination).
- External exposure (e.g., from ground contamination).

Figure 6.2 illustrates one way in which children could be harmed by radiation in the aftermath of a terrorist attack involving radioactive material. In this example, I-131 is used because it is the isotope for which there is the most evidence of its environmental exposure pathway—namely, the grass-cow-milk pathway—and because it is one of the few isotopes for which there is a specific radioprotective agent. However, the likelihood of an RDD using I-131 is extremely low because of its short half-life (a few weeks). I-131 will be released if there is a nuclear yield after an attack using an IND, and it may be released after a terrorist attack on a nuclear power plant if the attack results in an environmental release of radioactive material.

In contrast, strontium-90, which also follows the grass-cow-milk pathway, is of lesser concern because it does not become airborne as easily as the iodines and thus is less likely to travel as far as the radioiodines. However, strontium-90 has a much longer half-life (30 years), and although there is less concern, it will last for decades.

In contrast to radioiodine, most radioisotopes do not have a stable isotopic form that is biologically important and for which the radioactive form can cause biological damage via normal metabolic pathways. The element of each isotope has different biological,

chemical, and physical properties that would result in different health effects. Most of the effects of the radiation from radioiodine are on the thyroid. Radioiodine is also one of the few radioisotopes for which there is a targeted organ and specific method of prevention and treatment, namely, administration of stable potassium iodide.

The Chernobyl nuclear power plant accident in 1986 provides the best documented example of a massive radionuclide release in which large numbers of people across a broad geographical area were exposed to radionuclides released into the atmosphere. Chernobyl also most closely mimics the worst-case scenario of a successful terrorist attack on a nuclear power plant. At Chernobyl, radioiodine was released from a graphite and nuclear fuel fire that burned for 2 weeks after the initial core meltdown.

The medical effects on children from an environmental radioiodine release have been extensively studied. The U.S. Food and Drug Administration did a comprehensive review of the data relating radioiodine exposure to thyroid cancer risk after the Chernobyl reactor accident in 1998. Hundreds of thousands of measurements were made from some of the millions of people exposed in the most heavily contaminated regions of the former Soviet Union: Belarus, Ukraine, and the Russian Federation.

Thyroid radiation exposures after Chernobyl were virtually all internal, either through ingestion or inhalation of radioiodines. Starting 4 years after the accident, thyroid cancer increases of 30- to 100-fold were observed (compared with pre-Chernobyl rates), with estimated doses of <30 cGy (30 rads) to the thyroid. Consistent with the short half-life of iodine-131, children born more than 9 months after the accident have not shown an incidence of thyroid cancer above the levels seen prior to the Chernobyl accident. The FDA concluded from the Chernobyl data that there is a significant increase in the risk of childhood thyroid cancer at exposures of ≥5 cGy (5 rads) or greater. In contrast, there was no perceived increase in thyroid cancer in adults from the exposure to radioiodine during the accident.

Stable KI protects the thyroid by preventing the uptake of radioactive iodine. Stable KI was distributed throughout Poland after the Chernobyl accident to more than 10.5 million children younger than 16 years of age. Although there is no evidence on the degree of protection provided by the KI distribution throughout Poland compared with other regions around Chernobyl, the widespread distribution does provide valuable data on the safety and tolerability of KI in the general population. Twelve of 3,214 neonates treated with KI in Poland after Chernobyl showed a transient hypothyroidism. The FDA concluded that the benefits of KI treatment are especially important to children and neonates to reduce the risk of thyroid cancer and outweigh the risks of transient hypothyroidism. In contrast, the recommendation for adults older than 40 years is to administer stable KI only in the case of a projected dose of radiation to the thyroid greater than 500 cGy (rads), 100 times the exposure level for children.

Other measures may be taken to reduce exposures. For example, bans on the sale of milk products from contaminated areas prevent internal contamination during the first few weeks after an emergency where radioiodines are released. Due to the short half-life of I-

131, canned milk or other milk products that can be stored for a few months will not have any residual radioactive iodine and may be consumed.

Short-Term Evacuation Versus Sheltering

Decisions and actions to prevent radiation exposure must "do more good than harm." In other words, the benefit to health effects from dose reduction by using protective actions should more than offset the socioeconomic disadvantages of the protective actions.

Evacuation is an effective countermeasure to the presence of radiation and may prevent exposure to children. The decision to evacuate should take into account the potential disruption and actual risk. Area evacuations can result in increased risk of exposure if a plume already exists or if evacuation is to a location with a higher risk of exposure. Also, casualties can result from the evacuation process, and negative psychological effects can occur. Ideally, the evacuation would begin before the passage of any radioactive material carried in a dispersal cloud. Evacuation is almost always indicated if the projected average effective dose is likely to be >0.5 Sv (50 rem) within a day.

Variables affecting the pattern of radioactive material distribution include the time of day and year, the elapsed time since the accident, the size of the accident, and meteorological data to include wind patterns. Again, when deciding whether to evacuate or shelter in place, it is not recommended that predetermined levels alone be used for the decision. Basing the decision on these levels alone might lead to socioeconomic, physical, and psychological hardships that outweigh the benefits of lower exposure. For example, during the TMI accident, the Governor of Pennsylvania recommended that those members of society most vulnerable to radiation—namely, pregnant women and pre-school-age children within a 5-mile radius of the plant—leave the area. Studies done more than 10 years after the TMI accident found that many residents still showed psychological symptoms of stress. Most vulnerable to stress were the mothers of young children and those who had been evacuated. Symptoms included somatic complaints, anxiety, and depression, posttraumatic stress disorder symptoms and physiological symptoms, hypertension, and higher levels of norepinephrine, epinephrine, and cortisol. Persistent fears and anxiety were found that centered on the fact that the residents had been living close (within 5 miles) to the source of such potentially catastrophic danger years earlier.

The evacuation after the Chernobyl accident was poorly planned and chaotic. The 45,000 residents of Pripyat, the city closest to the power plant, were evacuated during a 3-hour period 36 hours after the accident occurred and were not allowed to return to their homes. Ninety thousand more people were evacuated over the next few days, clearing a 30-km zone around the power plant. Thousands of farm animals were slaughtered because there was no longer anyone to tend to them. The evacuees often were relocated to areas that were openly unreceptive and even hostile to them. Preliminary reports suggest that women pregnant at the time of the accident were more likely to have stress symptoms than others evacuated during the accident. Children evacuated to Kiev were more likely to report frequent headaches, chronic illness, and poorer overall health.

Additional factors must be considered when deciding whether to advise evacuation or sheltering in place in response to a radiological terrorist incident. Earlier recommendations by the EPA for evacuation were written for a nuclear power plant accident in which the release of radioactive material would occur hours after the initial accident, allowing the population to be evacuated in a plume-free environment. However, a terrorist attack is more likely to release a plume within minutes rather than hours of the initiation of the event. Therefore, sheltering is likely to be more protective in response to a radiological terrorist event. Sheltering should be performed whenever it is more protective than evacuation.

Sheltering in place for protection from radioactive fallout is also an effective countermeasure with little negative impact for short periods of time (hours) and may be done in a fallout shelter, an underground area, or in the middle of a large building. Any roofed structure would be of some benefit and would protect people from alpha and beta radiation, because the structure would keep radioactive particles from falling on the skin and further reduce the chance of inhalation. The degree of protection from gamma radiation increases with the type of material used in constructing the shelter, from wood, to masonry, to metal, metal being the most effective. Glass is not very effective in preventing the penetration of gamma radiation, and windows should be avoided because of the risk of lacerations from broken glass due to pressure changes or other consequences of the accident. Sheltering generally reduce exposures to external and internal contamination by 5 to 10 times. Sheltering is almost always justified if it will prevent exposures of 0.050 Sv (5 rem). At exposures <0.005 Sv (500 mrem), sheltering is not warranted. Sheltering provides protection from the falling radioactive material by absorbing the radiation (shielding) and keeping the radiation outside of the shelter (distance) while the radioactive material decays (time).

Table 6.11 shows the shielding factors from gamma radiation in a radioactive cloud plume. After the plume has passed and local authorities have announced that it is permissible or advisable to leave indoor protection, there will still be some radioactive material spread by the plume that is contaminating tree leaves, automobiles, and outdoor playground areas. Care should be taken to ensure that when children go outside they are in an area that has not been contaminated. Also, after leaving the shelter, it may still be necessary to relocate the population surrounding the region if the levels of contamination cannot be reduced quickly. It is also important to remember that family pets are not allowed in government shelters. Families should make plans and arrangements for the family pets ahead of time to keep them safe during the crisis.

Water and food may be scarce. They should be used prudently, but severe rationing of water should not be imposed, especially for children because they are more susceptible to dehydration than adults.

Parents should check with school officials to determine the school's plans for an emergency and discuss with their children what procedures will be followed to reunite children with parents and caregivers. It may not be possible to reach children away from

home immediately after the incident. A prearranged plan of where the children should go, if not in school, should be discussed and agreed on ahead of time.

Long-Term Habitation Versus Abandonment

The decision whether to remain long-term in an area or to abandon the area will depend on the levels of radiation remaining, the decay properties of the material, and the ability to physically remove the contaminated material. The decision should be reached in coordination with qualified health physicists because of complicating factors.

Rehabilitation/Abatement

Response organizations (local, State, and Federal) will need to prepare a site remediation plan. The clean-up process is lengthy and depends on the type of contamination and the site contaminated. There are temporary measures to fix radioactive material in place that will stop the spread of contamination. For example, flour and water mixtures, road oil, and water can be used to wet ground surfaces and prevent resuspension of the radioactive material.

Rehabilitation of contaminated areas, equipment, and facilities depends on the physical removal of contaminated particles. Usually, this will be done by trained personnel. It is important to determine whether or not water sources have been contaminated.

Hard surfaces are easier to rehabilitate than porous ones. Walls will generally be less contaminated than top and ground surfaces. Washing clothes and blankets in home washing machines can remove most contamination. It may not be possible to remove all contaminated material, however. Some articles can no longer be safely used (e.g., toys that have been left outside).

Contamination of Crops, Water, Food Animals, and Milk Sources

RDDs can also be used to contaminate livestock, fish, and food crops. However, huge quantities of radioactive material would be required to effectively contaminate food or water, and most radioactive material is not soluble in water. The U.S. Food and Drug Administration has guidance for accidental radioactive contamination of human food and animal feed.

Water may also be contaminated as an aftereffect of an explosion involving an RDD or IND. The safety of water sources should be evaluated after the attack. Water filtration in the home can reduce levels of radiation by removing many of the radioactive particles, but boiling or chlorination is not beneficial. Until the water supply has been determined to be safe, only bottled water should be used for drinking and cooking.

Water of questionable quality can be used for cleaning contaminated skin. This is because radiation exposure follows the principles of time, distance, and shielding. Radioactive material left on the skin will cause a higher level of exposure to an individual than exposure to water or the exposure from washing with contaminated water. Cleaning and

washing the hands and face are especially important to prevent internal contamination when eating and drinking.

Women who are breastfeeding should be especially concerned about the products they are ingesting. Fruit may be eaten if thoroughly washed or after the skin has been removed. Many types of fallout can become more concentrated in breast milk. For example, commercial milk can become contaminated with radioactive fallout if the milk was produced by cows grazing in the contaminated area. Contamination is further concentrated in human breast milk. Commercial formula reconstituted with bottled water is a safe alternative to breastfeeding until the mother's milk is demonstrated to be safe.

Bottled water and canned food products or foodstuffs imported from noncontaminated areas or produced before the radiological incident occurred should be free of radioactive contamination.

Pets may bring radioactive material into the house on the bottoms of their paws and should be kept indoors. Wiping or washing the pet's paws can be helpful to reduce the spread of contamination into the house.

Mortuary Affairs

The body retains radioactive materials after death. The body itself provides adequate shielding for alpha and beta-emitters. For gamma emitters (e.g., cesium-137), which are more penetrating radiation sources, radiation levels will be emitted by a contaminated corpse. Cremation should be avoided to prevent the radioactive material from vaporizing and becoming airborne. Guidance should be sought from local health physicists or city or State radiation safety experts or health departments. Concerns about burying the victims of radiation accidents have, in the past, raised fears in a community; specifically, the radiation accident in Goiania, Brazil, in 1987, and this possibility will need to be considered.

Bibliography

Radiological Threats

- American Academy of Pediatrics Committee on Environmental Health. Policy statement: radiation disasters and children. *Pediatrics.* 2003; 111(6):1455-1466.
- Glasstone S, Dolan P, eds. *The Effects of Nuclear Weapons*, 3rd ed. Washington, DC: U.S. Department of Defense and the Energy Research and Development Administration; 1977.
- National Council on Radiation Protection and Measurements. NCRP Report No. 65, *Management of Persons Accidentally Contaminated with Radionuclides.* Bethesda, MD: National Council on Radiation Protection and Measurements; 1979.

- National Council on Radiation Protection and Measurements. NCRP Report No. 138, *Management of Terrorist Events Involving Radioactive Material.* Bethesda, MD: National Council on Radiation Protection and Measurements; 2001.
- Sullivan JP, Bunker RJ, Lorelli, EJ, et al, eds. *Jane's Unconventional Weapons Response Handbook*, 1st ed. Alexandria, VA: Jane's Information Group; 2002.
- United States Army Center for Health Promotion and Preventive Medicine. *The Medical NBC Battlebook,* USACHPPM Tech Guide 244. Aberdeen Proving Ground, MD: United States Army Center for Health Promotion and Preventive Medicine; August 2002.

Historical Overview

- Adams RE, Bromet EJ, Panina N, Golovakha D, Goldgaber D, Gluzman S. Stress and well-being in mothers of young children 11 years after the Chornobyl nuclear power plant accident. *Psychol Med*. 2002; 32:143-156.
- American Academy of Pediatrics Committee on Environmental Health. Policy statement: radiation disasters and children. *Pediatrics.* (6)111;1455-1466.*
- Armed Forces Radiobiology Research Institute. *Medical Management of Radiological Casualties, Handbook,* 2nd ed. Bethesda, MD: Armed Forces Radiobiology Research Institute. April 2003. Available at: http://www.ntis.gov/products/mmrc.asp. Accessed August 18, 2006.
- Centers for Disease Control and Prevention. Emergency Preparedness and Response. *Frequently Asked Questions (FAQs) About Dirty Bombs*. Available at: http://www.bt.cdc.gov/radiation/dirtybombs.asp. Accessed August 1, 2006.
- Collins DL, de Carvalho A. Chronic stress from the Goiania 137Cs radiation accident. *Behav Med*. 1993;18:149–157.
- Radiation Effects Research Foundation. Acute death due to radiation. Hiroshima and Nagasaki, Japan: Radiation Effects Research Foundation. Available at: http://www.rerf.or.jp/eigo/radefx/early/death.htm. Accessed August 1, 2006.
- Hall, Eric. *Radiobiology for the Radiologist,* 5th ed. Philadelphia: Lippincott Williams and Wilkins; 2000.
- International Atomic Energy Agency. The Radiological Accident in Goiania. Vienna, Austria: International Atomic Energy Agency. 1988. Available at http://www-pub.iaea.org/MTCD/publications/PDF/Pub815_web.pdf. Accessed August 2, 2006.
- Larin V. Mayak's walking wounded. *Bull Atomic Scientists.* 1999;55(5):20–27. Available at: http://www.thebulletin.org/article.php?art_ofn=so99larin. Accessed August 18, 2006.
- Likhtarev IA, Kovgan LN, Jacob P, Anspaugh LR. Chernobyl accident: retrospective and prospective estimates of external dose of the population of Ukraine. *Health Phys*. 2002;82:290–303.
- Medical Research Council. *The Hazards to Man of Nuclear and Allied Radiations*. London: His Majesty's Stationery Office; 1956.
- Merke DP, Miller RW. Age differences in the effects of ionizing radiation. In: Guzelian PS, Henry CJ, Olin SS, eds. *Similarities and Differences Between*

Children and Adults: Implications for Risk Assessment. Washington, DC: International Life Sciences Institute; 1992:139–149.

- Miller RW. Delayed effects of external radiation exposure: A brief history. *Radiation Res.* 1995;144:160–195.
- Muirhead CR. Cancer after nuclear incidents. *Occup Environ Med*. 2001;58:482–487.
- National Academy of Sciences. *The Biological Effects of Atomic Radiation, Summary Reports*. Washington, DC: National Academy of Sciences-National Research Council;1956.
- National Council on Radiation Protection and Measurements. *Management of Terrorist Events Involving Radioactive Material.* NCRP Report No. 138. Bethesda, MD: National Council on Radiation Protection and Measurements; 2001.
- Niefert A. Case Study: *Accidental Leakage of Cesium-137 in Goiania, Brazil, in 1987.* Huntsville, AL: Camber Corporation; 1999. Available at http://www.nbc-med.org/SiteContent/MedRef/OnlineRef/CaseStudies/csgoiania.html. Accessed August 18, 2006.
- NOVA. *Dirty Bomb, Chronology of Events.* Public Broadcasting Service; February, 2003.
- O'Neill K. *The Nuclear Terrorist Threat*. Washington, DC: Institute for Science and International Security;1997. Available at http://www.isis-online.org/publications/terrorism/threat.pdf. Accessed August 18, 2006.
- *Public Health Strategies for Protecting the Thyroid with Potassium Iodide in the Event of a Nuclear Incident*. A symposium of the American Thyroid Association, Feb 28, 2003, Washington, DC, sponsored by the Johns Hopkins University School of Medicine in conjunction with the ATA.
- Talbott EO, Youk AO, McHugh KP, et al. Mortality among the residents of the Three Mile Island accident area: 1979-1992. *Environ Health Perspect*. 2000;108:545–552.
- Manhattan Engineer District of the United States Army. Chapter 10, Total Casualties. In: *The Atomic Bombings of Hiroshima and Nagasaki by the Manhatten Engineer District of the United States Army June 29, 1946*. New Haven, CT: The Avalon Project at Yale Law School: Documents in Law, History, and Diplomacy; 2003.
- U.S. Department of Energy. Advisory Committee on Human Radiation Experiments - Final Report. Washington, DC: U.S. Department of Energy; October, 1995. Available at: http://www.eh.doe.gov/ohre/roadmap/achre/report.html. Accessed August 18, 2006.
- U.S. Food and Drug Administration, Center for Drug evaluation and Research. *Guidance Document: Potassium Iodide as a Thyroid Blocking Agent in Radiation Emergencies.* Available at http://www.fda.gov/cder/guidance/4825fnl.htm. Accessed Nov 9, 2003.
- U.S. Nuclear Regulatory Commission. *Fact Sheet on Dirty Bombs,* March 2003. Available at http://www.nrc.gov/reading-rm/doc-collections/fact-sheets/dirty-bombs.html. Accessed Dec 9, 2003.

- U.S. Nuclear Regulatory Commission. *Fact Sheet on the Accident at Three Mile Island*. Available at http://www.nrc.gov/reading-rm/doc-collections/fact-sheets/3mile-isle.html. Accessed Nov 9, 2003.
- Weinberg AD, Kripalani S, McCarthy PL, Schull WJ. Caring for survivors of the Chernobyl disaster. What the clinician should know. *JAMA*. 1995;274:408–412.

*This document was the source for a large volume of material cited in this chapter.

Physical Principles of Ionizing Radiation

- Hall, Eric J. *Radiobiology for the Radiologist*, 5th ed. Lippincott-Raven, Philadelphia, 2000.
- Turner, James E. *Atoms, Radiation, and Radiation Protection*, 2nd ed. Wiley-Interscience, New York, 1995.

Radiation Biology and Dosimetry

- Anno GH, Baum SJ, Withers HR, Young RW. Symptomatology of acute radiation effects in humans after exposure to doses of 0.5-30 Gy. *Health Physics.* 1989;56(6):821–838.
- Armed Forces Radiobiology Research Institute. *Medical Management of Radiological Casualties – Handbook*, 2nd ed. AFRRI, 2003.
- Armed Forces Radiobiology Research Institute. Biodosimetry Assessment Tool (BAT). Bethesda, MD.
- Baranov AE, Densow D, Fliedner TM, et al. *Clinical Pre-Computer: Performa for the International Computer Database for Radiation Exposure Case Histories*. Berlin: Springer-Verlag, 1994.
- Goans RE, Holloway EC, Berger ME, Ricks RC. Early dose assessment following severe radiation accidents. *Health Physics*. 1997;72:513–518.
- Goans RE, Holloway EC, Berger ME, Ricks RC. Early dose assessment in criticality accidents. *Health Physics*. 2001;81(4) 446–449.
- Goans RE. Clinical care of the radiation-accident patient: patient presentation, assessment, and initial diagnosis. *The Medical Basis for Radiation-Accident Preparedness*. Ricks RC, Berger ME, O'Hara FM, Jr, eds. Washington, DC: The Parthenon Publishing Group. 2002;11-22.
- International Atomic Energy Agency. *Cytogenetic Analysis for Radiation Dose Assessment – A Manual*. Technical Report Series No. 405, IAEA, Vienna, 2001.
- International Atomic Energy Agency. *Diagnosis and Treatment of Radiation Injuries, Safety Reports Series No. 2.* Vienna, Austria: International Atomic Energy Agency; 1998.
- International Atomic Energy Agency. Generic Procedures for Medical Response During Nuclear and Radiological Emergency. Vienna, Austria: International Atomic Energy Agency; April 2005. Available at: http://www-pub.iaea.org/MTCD/publications/PDF/EPR-MEDICAL-2005_web.pdf. Accessed August 2, 2006.

- Lloyd DC, Edwards AA, Moquet JE, Guerrero-Carbajal YC. The role of cytogenetics in early triage of radiation casualties. *Appl Radiat Isotopes.* 2000;52:1107–1112.
- Lniecki J, Bajerska A, Adryszek C. Chromosomal aberrations in human lymphocytes irradiated in vitro from donors (males and females) of varying ages. *Int J Radiat Biol* 1971; 19:349–360.
- Prasanna PGS, Loats H, Gerstenberg HM, et al. AFRRI's Gamma-ray, X-ray, and Fission-neutron Calibration Curves for the Lymphocyte Dicentric Assay: Application of a Metaphase Finder System. Armed Forces Radiobiology Research Institute Special Publication, 2002.
- Prasanna PGS, Subramanian U, Greenhill RG, et al. Cytogenetic Biodosimetry Strategy for Potential Radiation Mass Casualties. In *Radiation Safety Aspects and Emergency Response, Proceedings of the Health Physics Society Midyear Topical Meeting on Homeland Defense and Emergency Response*, 36th Health Physics Society Topical Meeting, 2003. 218–223.
- Sine RC, Levine IH, Jackson WE, et al. Biodosimetry assessment tool: A postexposure software application for management of radiation accidents. *Mil Med*. 2001;166(12):85–87.
- Voisin P, Barquinero F, Blakely B, Lindholm C, et al. Towards a standardization of biological dosimetry by cytogenetics. *Cell Mol Biol.* 2002;48: 501–504.

Medical Diagnosis

- Armed Forces Radiobiology Research Institute. *Medical Management of Radiological Casualties Handbook*. Bethesda, MD: Military Medical Operations, Armed Forces Radiobiology Research Institute; April 2003.

External Contamination

- Armed Forces Radiobiology Research Institute. *Medical Management of Radiological Casualties Handbook* 2nd ed. Armed Forces Radiobiology Research Institute, Bethesda, MD. April 2003.
- Radiation Emergency Assistance Center/Training Site (REAC/TS). Oak Ridge, TN: Oak Ridge Institute for Science and Education. Available at http://orise.orau.gov/reacts. Accessed August 1, 2006.

Internal Radionuclide

- Armed Forces Radiobiology Research Institute. *Medical Management of Radiological Casualties Handbook* 2nd ed. Armed Forces Radiobiology Research Institute, Bethesda, MD. April 2003.
- Gusev IA, Guskova AK, Mettler FA, eds. *Medical Management of Radiation Accidents*, 2nd ed. Boca Raton, FL: CRC Press; 2001.
- National Council on Radiation Protection and Measurements. Report No. 65, *Management of Persons Accidentally Contaminated With Radionuclides*. Bethesda, MD: National Council on Radiation Protection and Measurements;1979.

Radiation Detection, PPE, Monitoring, and Decontamination

- National Institute for Occupational Safety and Health. Respirators. Available at: http://www.cdc.gov/niosh/npptl/. Accessed August 18, 2006.
- Radiation Emergency Assistance Center/Training Site. *Guidance for Radiation Accident Management, Detection.* Available at: http://orise.orau.gov/reacts/guide/detect.htm. Accessed August 1, 2006.
- Radiation Emergency Assistance Center/Training Site. *Guide for Radiation Accident Management.* Available at: http://orise.orau.gov/reacts/rad-incident-response.htm. Accessed August 1, 2006.
- U.S. Department of the Navy. Bureau of Medicine and Surgery Instruction 6470,10A. *Initial Management of Irradiated or Radioactively Contaminated Personnel*. Washington, DC: U.S. Department of the Navy; December 1998.

Medical Treatment: Issues Unique to Pediatrics

- American Academy of Pediatrics Work Group on Disasters. *Psychosocial Issues for Children and Families in Disasters: A Guide for the Primary Care Physician.* Rockville, MD: U.S. Department of Health and Human Services, Substance Abuse and Mental Health Services Administration, National Mental Health Information Center; 1995.
- American Academy of Pediatrics Committee on Environmental Health. Risk of ionizing radiation exposure to children: a subject review. *Pediatrics.* 1998;101:717–719.
- American Academy of Pediatrics Committee on Environmental Health. Policy statement: radiation disasters and children. *Pediatrics.* 2003; 111(6):1455-1466.
- Cronkite EP, Conrad RA, Bond VP. Historical events associated with fallout from Bravo Shot—Operation Castle and 25 Y of medical findings, *Health Phys*. 1997:73(1):176–186.
- Fliedner T, Friesecke M, Beyrer K (eds). *Medical Management of Radiation Accidents: Manual on the Acute Radiation Syndrome.* Oxford: British Institute of Radiology; 2001.
- Miller RW. Special susceptibility of the child to certain radiation-induced cancers. *Environ Health Perspect.* 1995;131(6):41–44.
- Moysich KB, Menezes RJ, Michalek AM. Chernobyl-related ionizing radiation exposure and cancer risk: an epidemiological review. *Lancet Oncol*. 2002;3(5):269–279.
- National Council on Radiation Protection and Measurements. *Management of Terrorist Events Involving Radioactive Material, NCRP Report No. 138.* Bethesda, MD: National Council on Radiation Protection and Measurements: 2001.
- National Council on Radiation Protection and Measurements. *Radiation Protection in Pediatric Radiology, NCRP Report No. 68.* Washington, DC: National Council on Radiation Protection and Measurements; 1981.

- Pacini F, Vorontsova T, Molinaro E, *et al.* Thyroid consequences of the Chernobyl nuclear accident. *Acta Paediatr Suppl* 1999;433:23–27.
- Rutherford MW, Seward JP. Radiation injuries and illnesses in pediatric emergency medicine. *Clin Ped Emerg Med*. 2001;2:141–151.
- Sugar M. Children in a disaster: an overview. *Child Psychiatry Hum Dev.* 1989;19:163–179.
- Sutow WW, Conard RA. Effects of ionizing radiation in children. *J Peds*. 1965;67(4):658–672.

Medical Treatment: Acute Radiation Syndrome

- Brook I, Elliott TB, Ledney GD, Knudson GB. Management of postirradiation sepsis. *Mil Med*. 2002;167(2 Suppl):105–106.
- Brook I, Walker RI, MacVittie TJ. Effect of antimicrobial therapy on bowel flora and bacterial infection in irradiated mice. *Int J Radiat Biol Stud Phys Chem Med*. 1988;53:709–716.
- Dainiak N, Waselenko JK, Armitage JD, MacVittie TJ, Farese AM. The hematologist and radiation casualties. *Hematology* 2003:473–496.
- Farese AM, Hunt P, Grab LB, MacVittie TJ. Combined administration of recombinant human megakaryocyte growth and development factor and granulocyte colony-stimulating factor enhances multilineage hematopoietic reconstitution in nonhuman primates after radiation-induced marrow aplasia. *J Clin Invest.* 1996; 97:2145–2151.
- Fliedner T, Friesecke M, Beyrer K, eds. *Medical Management of Radiation Accidents: Manual on the Acute Radiation Syndrome.* Oxford: British Institute of Radiology; 2001.
- Gunn V, Nechyba C, eds. *The Harriet Lane Handbook: A Manual for House Officers* Mosby; 2002.
- Hall EJ. Acute effects of total-body irradiation. In *Radiobiology for the Radiologist.* Philadelphia: Lippincott Williams and Wilkins; 2000.
- Hughes WT, Armstrong D, Bodey GP, et al. 2002 guidelines for the use of antimicrobial agents in neutropenic patients with cancer. *Clin Infect Dis*. 2002:15(34):730–751.
- Maekawa K. Overview of medical care of highly exposed victims in the Tokaimura accident. In *The Medical Basis for Radiation-Accident Preparedness: The Clinical Care of Victims.* New York: Parthenon; 2002.
- Medical Economics, ed. *Physicians' Desk Reference* 59th ed. Montvale, NJ: Thomson PDR, 2005.
- Medical Preparedness and Response Sub-Group, Department of Homeland Security Working Group on Radiological Dispersal Device Preparedness. December 9, 2003. Available at: http://www1.va.gov/emshg/docs/Radiological_Medical_Countermeasures_Indexed-Final.pdf. Accessed August 18, 2006.
- Nash RA, Schuening FG, Seidel K, et al. Effect of recombinant canine granulocyte-macrophage colony-stimulating factor on hematopoietic recovery after otherwise lethal total body irradiation. *Blood*. 1994;83:1963–1970.

- National Council on Radiation Protection and Measurements. *Management of Terrorist Events Involving Radioactive Material, NCRP Report No. 138.* Bethesda, MD: National Council on Radiation Protection and Measurements: 2001.
- Patchen ML, Brook I, Elliott TB, Jackson WE. Adverse effects of pefloxacin in irradiated C3H/HeN mice: correction with glucan therapy. *Antimicrob Agents Chemother.* 1993;37:1882–1889.
- Wade JC, Rubenstein EB. NCCN Fever and Neutropenia Practice Guidelines Panel: Fever and neutropenia. *Cancer Cntl*. 2001;8(6 Suppl 2):16–21.
- Waselenko JK, MacVittie TJ, Blakely WF, et al. Medical management of the acute radiation syndrome: recommendations of the Strategic National Stockpile Radiation Working Group. *Ann Intern Med*. 2004;140(12):1037–1051 and W-64 to W-67.

Medical Treatment: Internal Contamination

- American Academy of Pediatrics Committee on Environmental Health. Policy statement: radiation disasters and children. *Pediatrics* 2003;111(6):1455–1466.
- Armed Forces Radiobiology Research Institute. *Medical Management of Radiological Casualties*, 2nd ed. Bethesda, MD: Armed Forces Radiobiology Research Institute; April 2003.
- Guskova AK, Gusev IA. Medical aspects of the accident at Chernobyl. In: Gusev IA, Guskova AK, Mettler FA. *Medical Management of Radiation Accidents*. Washington, DC: CRC Press; 2001.
- International Atomic Energy Agency. The Radiological Accident in Goiania. Vienna, Austria: International Atomic Energy Agency; 1988. Available at: http://www-pub.iaea.org/MTCD/publications/PDF/Pub0815_web.pdf. Accessed May 31, 2004.
- Medical Economics, ed. *Physicians' Desk Reference*. Montvale, NJ: Thomson PDR; 2003.
- National Council on Radiation Protection and Measurements. *NCRP Report No. 65, Management of Persons Accidentally Contaminated with Radionuclides*, Bethesday, MD: National Council on Radiation Protection and Measurements;1979.
- National Research Council. *Distribution and Administration of Potassium Iodide in the Event of a Nuclear Incident.* Washington, DC: National Academy Press; 2004.
- Radiation Emergency Assistance Center/ Training Site (REAC/TS). *Managing Radiation Emergencies: Guidance for Hospital Medical Management*. Oak Ridge, TN: Oak Ridge Institute for Science and Education. Available at: http://orise.orau.gov/reacts/guide/manage.htm. Accessed August 2, 2006.
- Radiation Emergency Assistance Center/ Training Site (REAC/TS). *Zn-DTPA Informational Material*. Oak Ridge, TN: Oak Ridge Institute for Science and Education; 2002. Available at: http://orise.orau.gov/reacts/zinc-dtpa.htm. Accessed August 2, 2006.

- U.S. Food and Drug Administration, Center for Drug Evaluation and Research. Calcium-DTPA and Zinc-DTPA. Available at: http://www.fda.gov/cder/drug/infopage/DTPA/default.htm. Accessed August 22, 2006.
- U.S. Food and Drug Administration, Center for Drug Evaluation and Research. *Home Preparation Procedure for Emergency Administration of Potassium Iodide Tablets to Infants and Small Children*. Last updated March 6, 2006. Available at http://www.fda.gov/cder/drugprepare/kiprep.htm. Accessed August 2, 2006.
- U.S. Food and Drug Administration, Center for Drug Evaluation and Research. *Prussian Blue for Treatment of Internal Contamination with Thallium or Radioactive Cesium*, Oct 2003. Available at http://www.fda.gov/cder/drug/infopage/prussian_blue/default.htm. Accessed August 22, 2006.
- U.S. Food and Drug Administration, Department of Health and Human Services. Guidance for industry on prussian blue for treatment of internal contamination with thallium or radioactive cesium. Federal Register. 2003; 68(23):5645–5648.
- U.S. Government, Public Law 107–188, June 12, 2002. Public Health Security and Bioterrorism Preparedness and Response Act of 2002, Section 127. Potassium Iodide.
- Verger P, Aurengo A, Geoggroy B, LeGuen B. Iodine kinetics and stable iodine prophylaxis after intake of radioactive iodine: a review. *Thyroid* 2001;11(a):353–360.
- World Health Organization. Guidelines for Iodine Prophylaxis Following Nuclear Accidents: Update 1999. Geneva, Switzerland: WHO; 1999.

Surgical Issues

- Barabanova AV. Local Radiation Injury. In: Gusev D, Guskova AK, Mettler FA, eds. *Medical Management of Radiation Accidents*, 2nd ed. 2001; CRC Press, Washington DC: p 221–232.
- Bushberg JT. Health Physics Society, *Emergency Department Management of Radiation Casualties*. 5 October 2004. Available at: http://hps.org/hsc/documents/emergency.ppt Accessed August 22, 2006.
- Delanian S, Porcher R, Balla-Mekias A, Lefaix J. Randomized, placebo-controlled trial of combined pentoxifylline and tocopherol for regression of superficial radiation-induced fibrosis. *J Clin Oncol.* 2003;21(13):2545–2550.
- Gusev IA, Guskova AK, Mettler FA. *Medical Management of Radiation Accidents*, 2nd ed. 2001; CRC Press, Washington DC; 76.
- Hall EJ. *Radiobiology for the Radiologist*. New York, New York: Lippincott Williams & Wilkins, 2000; 343, 536
- McClain DE, Benson KA, Ejnik J, et al. Health effects of embedded depleted uranium. *Military Medicine* 2002;167(2 Suppl):117–119.
- *Medical Management of Radiological Casualties Handbook*, 2nd ed. Armed Forces Radiobiology Research Institute, Bethesda, MD; April 2003;36–37.

- Peter RU, Gottlober P. Management of cutaneous radiation injuries: diagnostic and therapeutic principles of the cutaneous radiation syndrome, *Military Medicine* 2002:167(2 Suppl):110–112.
- Peter RU. The cutaneous radiation syndrome. In: MacVittie TJ, Weiss JF, Browne D. *Advances in the Treatment of Radiation Injuries. Advances in the Biosciences.* Tarrytown, NY: Pergamon;1996;94:237–240.

Followup Care

- American Academy of Pediatrics Committee on Environmental Health Radiation disasters and children. Policy statement: radiation disasters and children. *Pediatrics.* 2003:111:1455–1466.
- Bauer AJ, Tuttle RM, Francis G. Thyroid nodules and thyroid carcinoma in children. In: Pescovitz O, Eugster E (eds) *Pediatric Endocrinology: Mechanisms, Manifestations, and Management*. Philadelphia, PA: Lippincott Inc; 2004; 522–547.
- Drozd VM, Astachova LN, Polyanskaya ON, et al. Characteristics of thyroid ultrasound pictures in children with nodular thyroid changes effected by radionuclides. *Bildgebung* 1995;62:236–241.
- Gharib H. Changing concepts in the diagnosis and management of thyroid nodules. *Endocrinol Metab Clin North Am.* 1997;26:777–800.
- Hall E. *Radiobiology for the Radiologist*, 5th ed. Philadelphia, PA: Lippincott Williams and Wilkins. 2000; 162.
- Howard JE, Vaswani A, Heotis P. Thyroid disease among the Rongelap and Utirik population--an update. *Health Phys.* 1997;73:190–198.
- Hung W. Solitary thyroid nodules in 93 children and adolescents. a 35-year experience. *Horm Res*. 1999;52:15–18.
- Nikiforov YE, Koshoffer A, Nikiforova M, Stringer J, Fagin JA. Chromosomal breakpoint positions suggest a direct role for radiation in inducing illegitimate recombination between the ELE1 and RET genes in radiation-induced thyroid carcinomas. *Oncogene.* 1999;18:6330–6334.
- Poth M. Thyroid cancer in children and adolescents. In: Wartofsky L (ed). *Thyroid Cancer: A Comprehensive Guide to Clinical Management.* Towota, NJ: Humana Press; 2000; 121–128.
- Preston DL, Shimizu Y, Pierce DA, Suyama A, Mabuchi. K. Studies of mortality of atomic bomb survivors. Report 13: solid cancer and noncancer disease mortality: 1950-1997. *Radiat Res*. 2003;160:381–407.
- Robbins RJ, Chon JT, Fleisher M, Larson SM, Tuttle RM. Is the serum thyroglobulin response to recombinant human thyrotropin sufficient, by itself, to monitor for residual thyroid carcinoma? *J Clin Endocrinol Metab.* 2002;87:3242–3247.
- Schlumberger MJ. Diagnostic follow-up of well-differentiated thyroid carcinoma: historical perspective and current status. *J Endocrinol Invest* 1999;22:3–7.
- Shore RE. Issues and epidemiological evidence regarding radiation-induced thyroid cancer. *Radiat Res.* 1992;131:98–111.

- Sklar CA, Mertens AC, Mitby P, et al. Risk of disease recurrence and second neoplasms in survivors of childhood cancer treated with growth hormone: a report from the Childhood Cancer Survivor Study. *J Clin Endocrinol Metab* 2002;87:3136–3141.
- Tuttle RM, Becker DV. The Chernobyl accident and its consequences: update at the millennium. *Semin Nucl Med.* 2000;30:133–140.
- U.S. Food and Drug Administration, Center for Drug Evaluation and Research 2002. Guidance for Industry: KI in Radiation Emergencies - Questions and Answers. Available at: http://www.fda.gov/cder/guidance/5386fnl.htm. Accessed August 24, 2006.
- Welch Dinauer CA, Tuttle RM, Robie DK, et al. Clinical features associated with metastasis and recurrence of differentiated thyroid cancer in children, adolescents and young adults. *Clin Endocrinol* (Oxf) 1998;49:619–628.

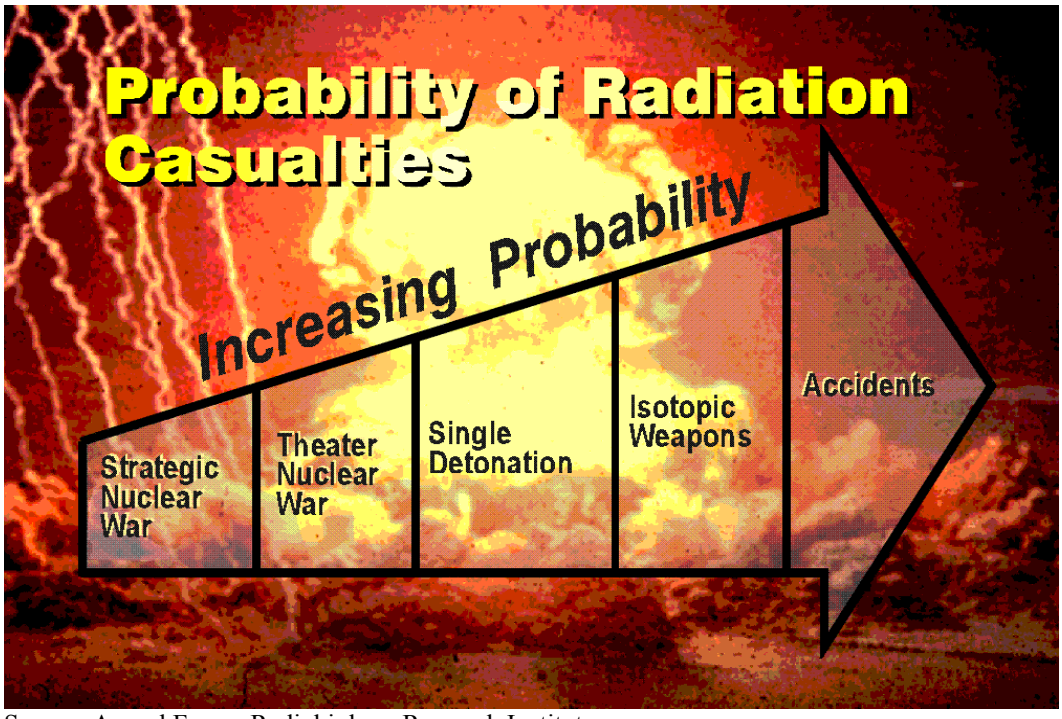
Environmental Issues

- Bromet EJ. Psychological Effects of Radiation Casualties. In: *Effects of Ionizing Radiation: Atomic Bomb Survivors and Their Children (1945-95)*. Washington, DC: John Henry Press, 1998; 288–289. Available at: http://books.nap.edu/catalog/5805.html. Accessed August 24, 2006.
- Cordesman AH. *Terrorism, Asymmetric Warfare, and Weapons of Mass Destruction, Defending the U.S. Homeland*. Westport, CN: Praeger Publishers; 2002.
- Eisenbud M, Gesell T. *Environmental Radioactivity, from Natural, Industrial, and Military Sources,* 4th ed. San Diego: Academic Press;1997;108, 415, 418, 530–531.
- Health Physics Society. *Guidance for Protective Actions Following a Radiological Terrorist Event, Position Statement of the Health Physics Society*. Adopted Jan 2004. Available at: http://www.hps.org/documents/RDDPAGs.pdf. Accessed August 2, 2006.
- International Atomic Energy Agency. The Radiological Accident in Goiania. Vienna, Austria: International Atomic Energy Agency. 1988. Available at http://www-pub.iaea.org/MTCD/publications/PDF/Pub815_web.pdf. Accessed August 2, 2006.
- International Atomic Energy Agency, IAEA-TECDOC-1388, Feb 2004.
- National Council on Radiation Protection and Measurements. *Management of Terrorist Events Involving Radioactive Material, NCRP Report No. 138*, Bethesda, MD: National Council on Radiation Protection and Measurements; 2001.
- Nuclear Regulatory Commission Web site, Fact Sheet on the Accident at Three Mile Island. Available at http://www.nrc.gov/reading-rm/doc-collections/fact-sheets/3mile-isle.html. Accessed August 2, 2006.
- U.S. Army Center for Health Promotion and Preventive Medicine. *Medical NBC Battlebook Section 3-4 to 3-6*. Aug 2002.
- U.S. Dept of Health and Human Services, Food and Drug Administration, 13 Aug 1998. *Accidental Radioactive Contamination of Human Food and Animal Feeds:*

Recommendations for State and Local Agencies. August 13, 1998. Available at: http://www.fda.gov/cdrh. Accessed August 24, 2006.

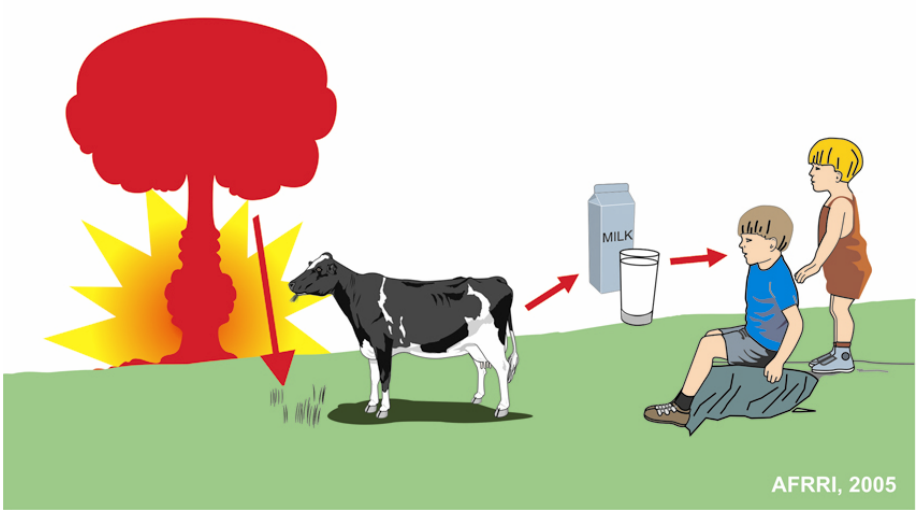
- U.S. Dept of Health and Human Services, Food and Drug Administration, Center for Drug Evaluation and Research. *Guidance, Potassium Iodide as a Thyroid Blocking Agent in Radiation Emergency*. Dec 2001. Available at www.fda.gov/cder/guidance/4825fnl.htm. Accessed August 2, 2006.
- U.S. Environmental Protection Agency. *Manual of Protective Action Guides and Protective Actions for NuclearIincidents.* Washington, DC: U.S. Environmental Protection Agency; EPA 400-R-92-001; May 1992. Available at: http://www.epa.gov/radiation/docs/er/400-r-92-001.pdf. Accessed August 2, 2006.

Figure 6.1. Probability of radiation casualties



Source: Armed Forces Radiobiology Research Institute.

Figure 6.2. Environmental exposure pathway

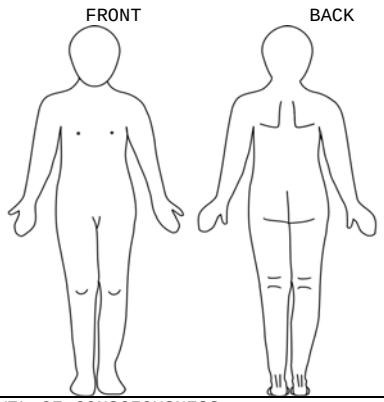


Note: Radioactive particles produced from a terrorist event travel to pastures as fallout. If the fallout contains radioactive iodine, some of it eaten by the cow would be passed into the cow's milk. Consumption of contaminated fresh milk must be controlled during the first few weeks of an emergency. However, products made with contaminated milk, e.g., cheeses and canned milk, which can be stored for several months, may then be consumed safely.

Source: Armed Forces Radiobiology Research Institute.

Figure 6.3. Generic pediatric medical field card

1. LAST NAME, FIRST NAME	AGE	☐ MALE ☐ FEMALE
PATIENT ID	ESTIMATED WEIGHT	RELIGION
2. ADDRESS		
PARENT/GUARDIAN'S NAME	NATIONALITY	
☐ NON-COMBATANT INJURY	☐ DISEASE	☐ PSYCH
3. INJURY		☐ AIRWAY
		☐ HEAD
		☐ WOUND
		☐ NECK/BACK INJURY
		☐ BURN
		☐ AMPUTATION
		☐ STRESS
		☐ OTHER (SPECIFY)

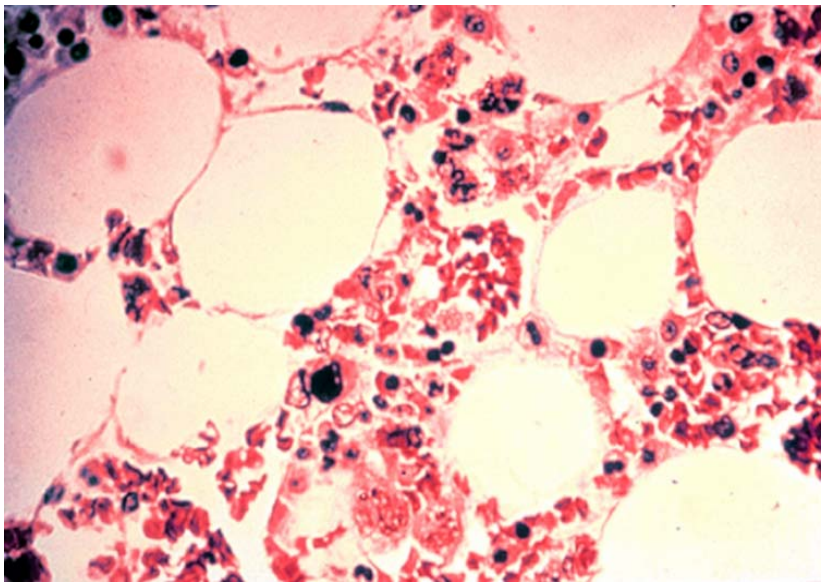


4. LEVEL OF CONSCIOUSNESS					
☐ ALERT			☐ PAIN RESPONSE		
☐ VERBAL RESPONSE			☐ UNRESPONSIVE		
5. PULSE	TIME	6. TOURNIQUET ☐ NO ☐ YES		TIME	
7. MORPHINE ☐ NO ☐ YES	DOSE	TIME	8. IV	TIME	
9. TREATMENT/OBSERVATIONS/CURRENT MEDICATION/ALLERGIES/NBC (ANTIDOTE)					
10. DISPOSITION	☐ RELEASED ☐ EVACUATED ☐ DECEASED		TIME		
11. PROVIDER/UNIT			DATE/DATE (YYMMDD)		

12. REASSESSMENT				
DATE (YYMMDD)		TIME OF ARRIVAL		
TIME				
BP/PS				
PULSE/POULS				
RESP/RESP				
DATE/TIME	13. CLINICAL COMMENTS/DIAGNOSIS			
	14. ORDERS/ANTIBIOTICS (SPECIFY)/TETANUS/IV FLUIDS			
15. PROVIDER			DATE (YYMMDD)	
16. DISPOSITION	☐ RELEASED TO PARENT/GUARDIAN ☐ EVACUATED ☐ DECEASED		TIME	
17. RELIGIOUS SERVICES	☐ BAPTISM ☐ ANOINTING ☐ CONFESSION		☐ PRAYER ☐ COMMUNION ☐ OTHER	
CUSTODIAL ADULT AT DISPOSITION				

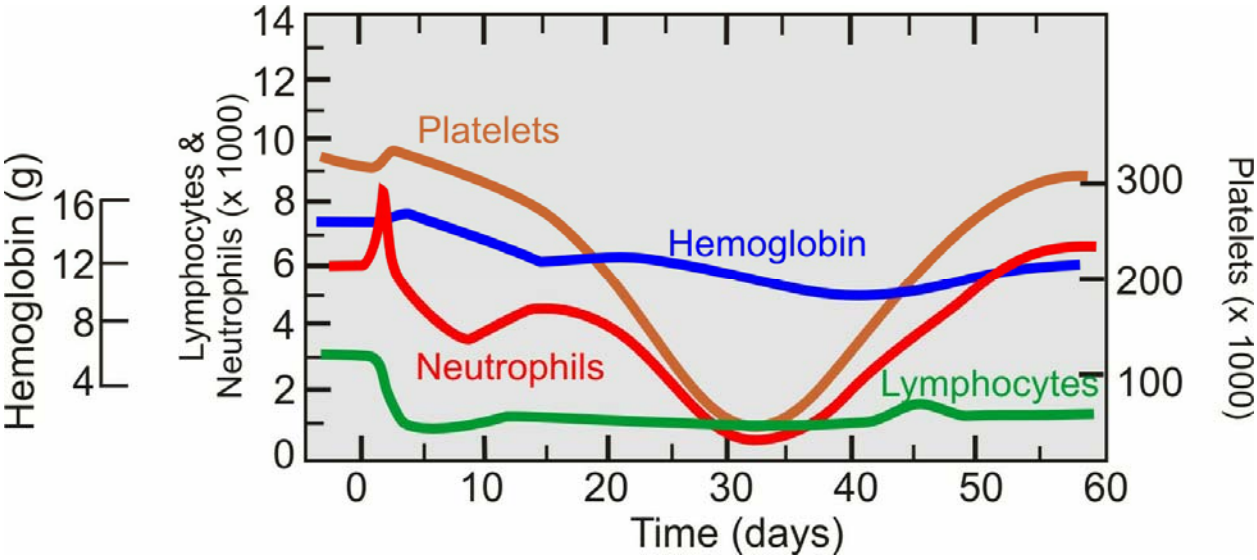
Source: Armed Forces Radiobiology Research Institute.

Figure 6.4. Bone marrow irradiated to 3 Gy



Source: Armed Forces Radiobiology Research Institute.

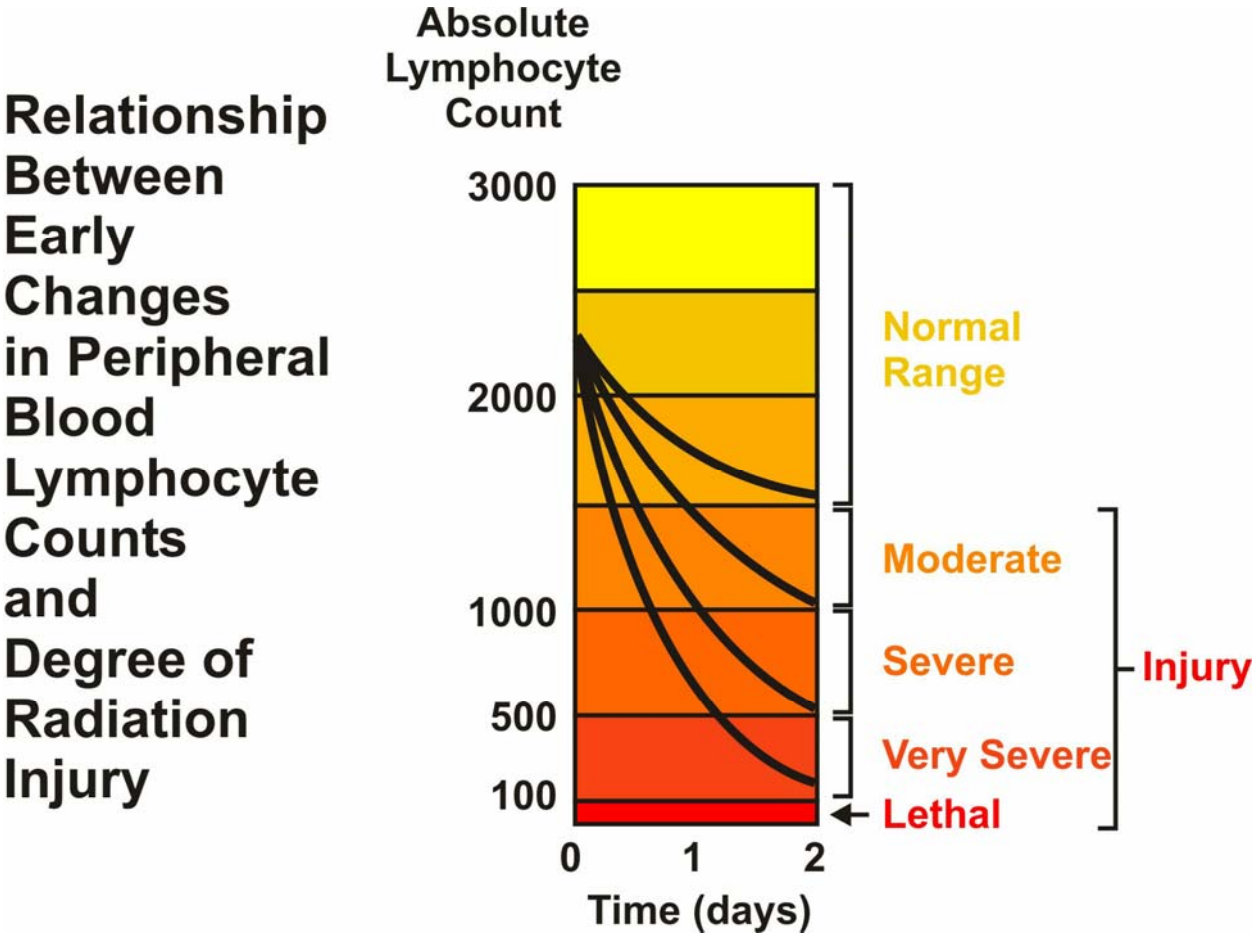
Figure 6.5. Pattern of a series of blood counts graphed over time after 3 Gy of whole-body exposure



Note: Typical changes in the peripheral blood profile will occur as early as 24 hours after irradiation, with lymphocytes falling immediately on day 1. At about 4 weeks, cell lines bottom out. Without white cells, overwhelming infection occurs, and without platelets, hemorrhage is expected.

Source: Armed Forces Radiobiology Research Institute.

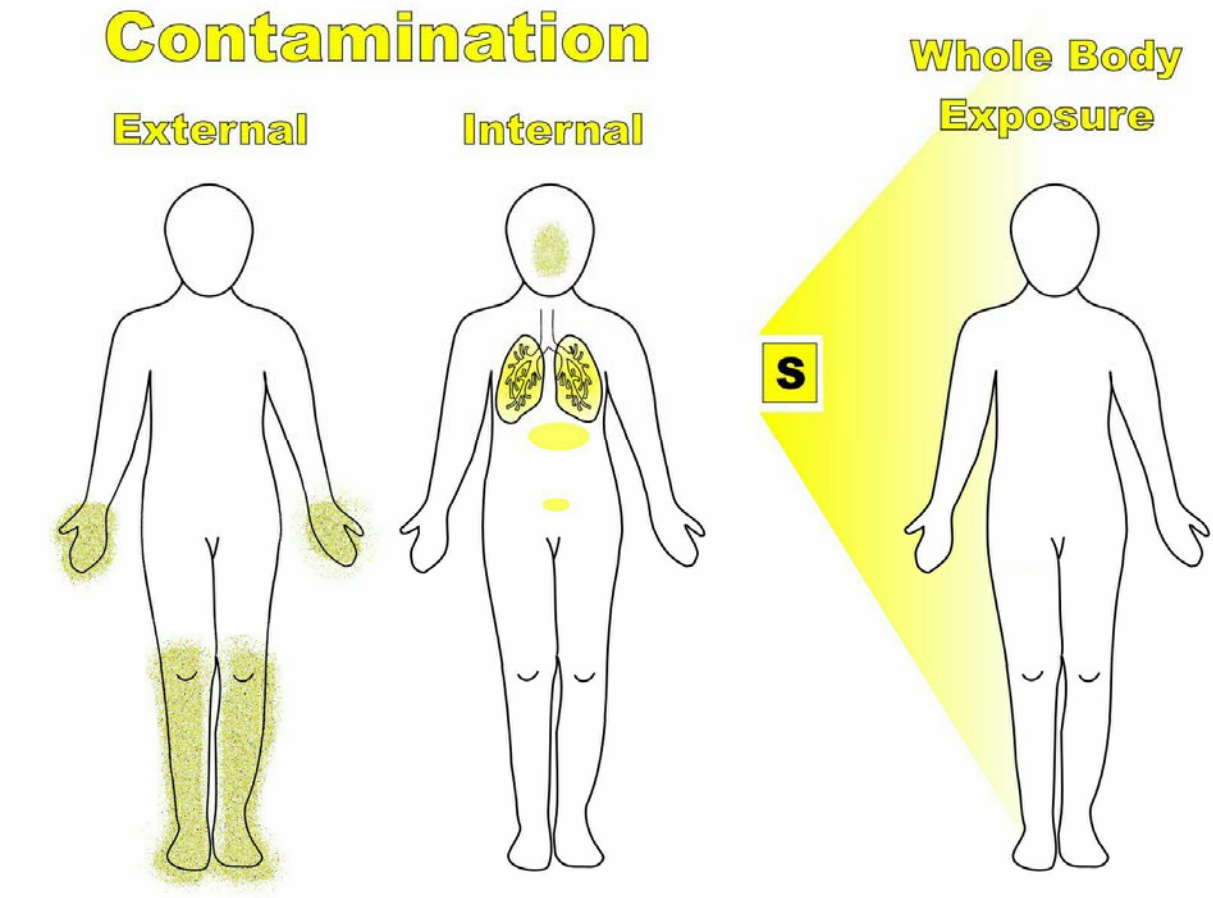
Figure 6.6. Classic Andrews diagram (1965)



Note: Lymphocytes drop quickly with high radiation doses; a drop of 50% or more in 24 hours indicates a severe radiation injury.

Source: Armed Forces Radiobiology Research Institute.

Figure 6.7. Contamination versus exposure



Note: Contamination and exposure are not the same. Patients can have external exposure to radiation, external contamination by radioactive material, internal contamination with radionuclides, or any combination thereof.

S = Source of radiation.

Source: Armed Forces Radiobiology Research Institute.

Figure 6.8. Examples of radiation survey meters

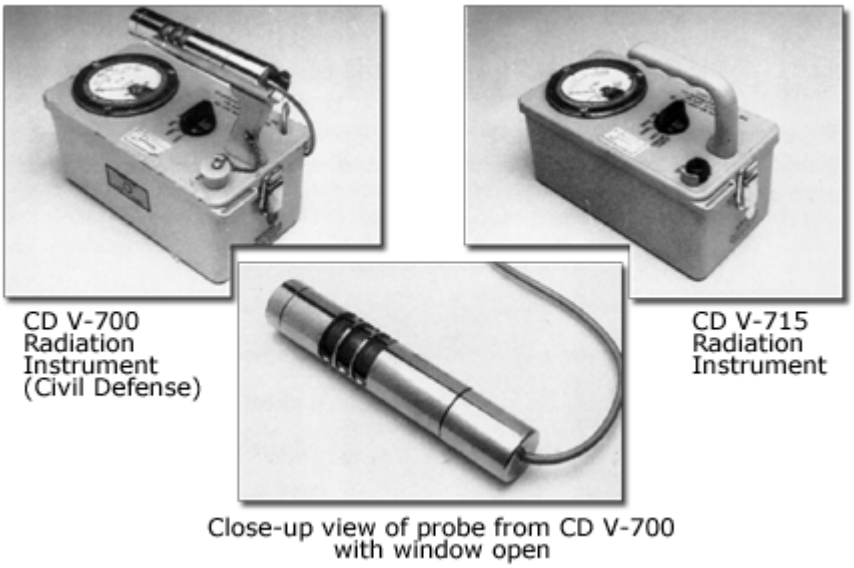
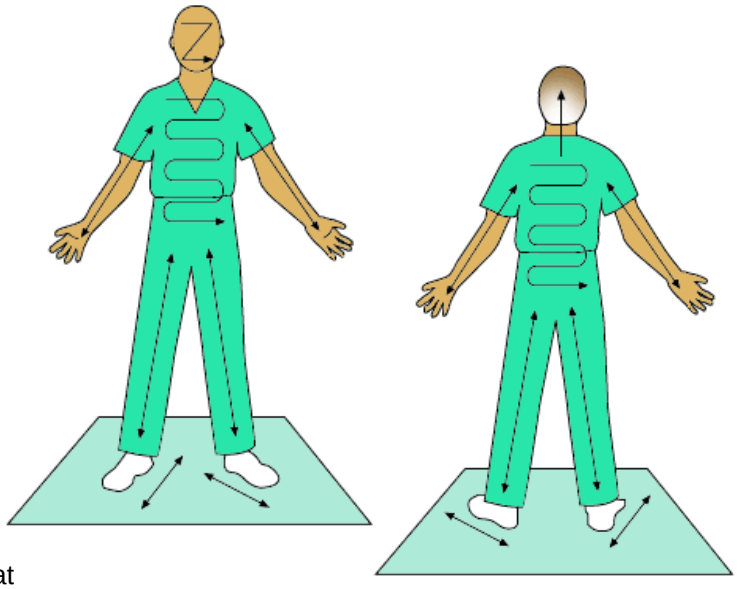


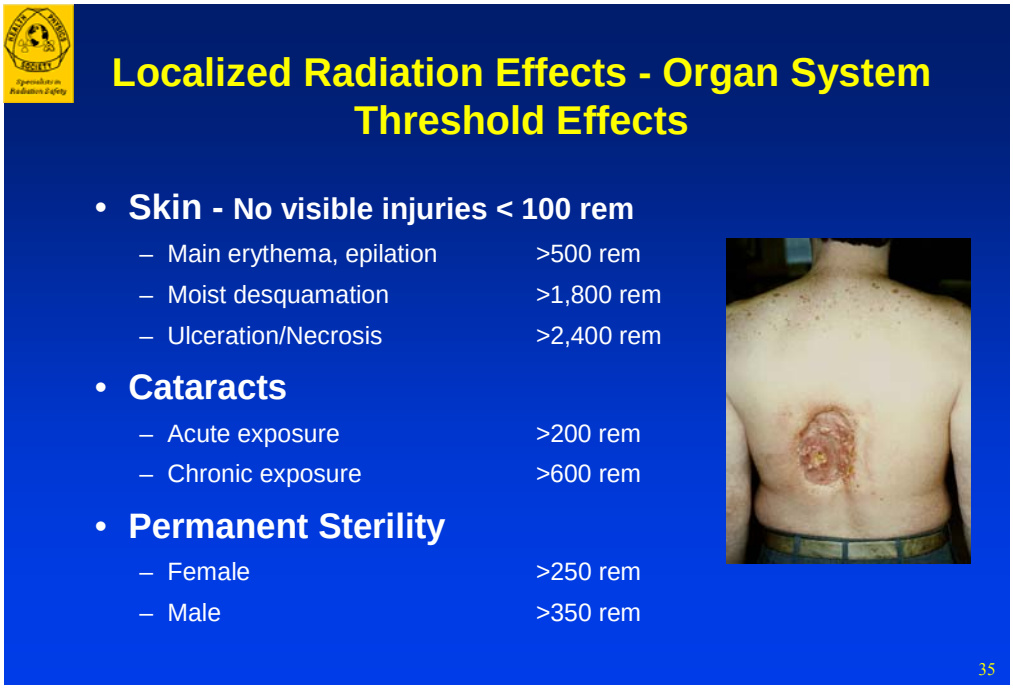
Figure 6.9. Recommended procedures for monitoring personnel



1. Have the person stand on a clean pad.
2. Instruct the person to stand straight, feet
spread slightly, arms extended with palms up
and fingers straight out.
3. Monitor both hands and arms; then repeat with hands and arms turned over.
4.Starting at the top of the head, cover the entire body, monitoring carefully the forehead,
nose, mouth, neckline, torso, knees, and ankles.
ankles.
5. Have the person turn around, and repeat the survey on the back of the body.
6. Monitor the soles of the feet.

Source: Courtesy Oak Ridge Associated Universities' REAC/T Center.

Figure 6.10. Localized radiation effects



Source: Health Physics Society. From "Emergency Department Management of Radiation Casualties," a presentation for hospital staff trainees. Available for download at http://hps.org/hsc/documents/emergency.ppt.

Table 6.1. Biological dosimetry assays and operational parameters

Class of biomarker	Assay	Time window	Operating dose window (Gy, acute photon-equivalent)
Prodromal signs and symptoms	Onset of vomiting	<12 hr	2–20
Hematological	Depletion of peripheral blood lymphocytes	<1.5 wk	2–8
Cytological	Lymphocyte-metaphase spread dicentric Lympho cyte-premature chromosome condensation	<several months* <several months*	0.2–5 0.2–20

* Generally, blood is sampled 24 hours after radiation exposure for cytological chromosome aberration analysis of dose.

Table 6.2. Biodosimetry based on acute photon-equivalent exposures

Dose estimate	Time to onset of vomiting		Absolute lymphocyte count ($\times 10^9$/L)[b] (day)						Lymphocyte depletion rate[c]	Number of dicentrics[d]	
Gy	%[a]	Time (hr)	0.5	1	2	4	6	8	Rate constant	Per 50 cells	Per 1000 cells
0	—	—	2.45	2.45	2.45	2.45	2.45	2.45	—	0.05-0.1	1-2
1	19		2.30	2.16	1.90	1.48	1.15	0.89	0.126	4	88
2	35	4.63	2.16	1.90	1.48	0.89	0.54	0.33	0.252	12	234
3	54	2.62	2.03	1.68	1.15	0.54	0.25	0.12	0.378	22	439
4	72	1.74	1.90	1.48	0.89	0.33	0.12	0.044	0.504	35	703
5	86	1.27	1.79	1.31	0.69	0.20	0.06	0.020	0.63	51	1024
6	94	0.99	1.68	1.15	0.54	0.12	0.03	0.006	0.756		
7	98	0.79	1.58	1.01	0.42	0.072	0.012	0.002	0.881		
8	99	0.66	1.48	0.89	0.33	0.044	0.006	<0.001	1.01		
9	100	0.56	1.39	0.79	0.25	0.030	0.003	<0.001	1.13		
10	100	0.48	1.31	0.70	0.20	0.020	0.001	<0.001	1.26		

Note: Depicted above are the three most useful elements of biodosimetry. The first column illustrates the percent of people who vomit, based on dose received and time to onset. The middle section reveals the time frame in which a patient develops lymphopenia. Two lymphocyte samples drawn at different times can then be used to predict a rate constant, which is then used to estimate exposure dose. The final column represents the current "gold standard," which requires several days before the result is known. We recommend initiating colony-stimulating factor if either onset of vomiting or lymphocyte depletion kinetics suggests an exposure for which treatment is recommended, which can then be discontinued if necessary when data from chromosome dicentrics are known.

[a] Cumulative percentage of victims with vomiting.

[b] Normal range 1.4–3.5 $\times 10^9$/L.

[c] The lymphocyte depletion rate is based on the model $Lt = 2.45 \times 10^9/L \times e\text{-}k(D)t$, in which Lt equals the lymphocyte count ($\times 10^9$/L), 2.45×10^9/L equals a constant representing the consensus mean lymphocyte count in the general population, k equals the lymphocyte depletion rate constant for a specific acute photon dose, and t equals the time after exposure (days).

[d] Number of dicentric chromosomes in human peripheral blood lymphocytes.

Acknowledgments: The authors thank W.E. Jackson for assistance in preparation of this table. Special thanks go to Mark Behme for expert graphical assistance. This work was supported in part by the Armed Forces Radiobiology Research Institute, work number AFRRI-04-3.

Table 6.3. Initial or prodromal phase, whole body irradiation from external radiation or internal absorption (gamma radiation)

	Subclinical range (cGy [rad])		Sublethal range(cGy [rad])		Lethal range(cGy [rad])	
	0–100	100–200	200–600	600–800	800–3000	>3000
Vomiting (onset and percentage)	None	3–6 hr 5%–50%	2–4 hr 50%–100%	1–2 hr 75%–100%	<1 hr 90%–100%	<1 hr 100%
Nausea duration		<24 hr	<24 hr	<48 hr	<48 hr	<48 hr
Diarrhea (severity, onset, and percentage)			Moderate 3–8 hr <10%	Severe 1–3 hr >10%	Severe <3 hr >10%	Very severe <1 hr 100%
Lymphocyte count ($\times 10^6$/L)*	Unaffected	Minimally decreased within normal range	2000–1000 at 24 hr	1000–800 at 24 hr	<1000 at 24 hr	Decreases with hours
Headache, (severity, onset, and percentage)		Slight	Mild to moderate 4–24 hr <50%	Severe 3–4 hr <80%	Severe <4 hr >80%	
CNS function	No impairment	No impairment	Routine task performance; cognitive impairment for 6–20 hr	Simple and routine task performance; cognitive impairment for >24 hr	Progressive incapacitation	
Body temperature (severity, onset, and percentage)		Normal	Increased and fever <3 hr 10%–100%	High fever <1 hr 100%		

Source: Adapted from other resources (Armed Forces Radiobiology Research Institute, 2003; International Atomic Energy Agency. *Diagnosis and Treatment of Radiation Injuries*. Safety Reports, Series 2. IAEA, Vienna, Austria; 1998); used with permission.

*Normal range 1400–3500 $\times 10^6$/L

Table 6.4. Diagnostic steps in acute radiation sickness

Rapid diagnosis
Prodrome: nausea, vomiting
Lymphocyte counts – *best*
Later
Physical dosimetry
Biodosimetry
Skin changes

Table 6.5. Timing to onset of vomiting (prodrome)

Dose (Gy)	Onset (hr)	Duration (hr)	Latency
0.5–2	Absent or 6–24	<24	Absent to 3+ wk
2–3	2–6	12 to <24	2–3 wk
3–5	1–2	24	1–2.5 wk
>5	Minutes to 1	48	2–4 days

Table 6.6. Dose assessment summary

Signs and symptoms	Radiation dose (Gy)
Chromosome abnormalities	0.20
Lymphocytes reduced	1
Nausea/vomiting at 3 hr	2–3
Skin: erythema (1 day), epilation (14 days)	3–5

Table 6.7. Guidelines for bioassay sampling

Suspected radioactive material	Feces: begin sample after exposure	Urine: begin sample after exposure	Sample quantity
Plutonium	24 hr	24 hr then 2 wk	24 hr total
Uranium	24 hr	24 hr then 7–10 days	24 hr total
Tritium	N/A	12 hr	1 voiding

Table 6.8. Antibiotics for treatment of post-radiation neutropenia

Antimicrobial	Dosage and route	Comments and spectrum of activity
First-choice antimicrobials (listed in order of potential efficacy)		
Cefepime	150 mg/kg divided q 8 h, IV or IM	Not available in oral form. Activity against gram-positive organisms, except *Enterococcus* spp. Activity against gram-negative organisms, including *Pseudomonas aeruginosa* and certain *Enterobacteriaceae* that generally are resistant to most third-generation cephalosporins.
Ceftazidime	100–300 mg/kg/day divided q 8 h	May need to add amoxicillin* or vancomycin† Not available in oral form. Activity against gram-negative organisms, including *Pseudomonas aeruginosa* and certain *Enterobacteriaceae* that generally are resistant to most third-generation cephalosporins.
Ceftriaxone	50–75 mg/kg/day, IV or IM, once a day	Not available in oral form. Activity against gram-positive organisms, except *Enterococcus* spp.
Second-choice		
Gentamicin	3–7.5 mg/kg/day divided q 8–24 h, IV or IM	May need to add amoxicillin* or vancomycin† Not available in oral form.
Amikacin	15–22.5 mg/kg/day divided q 8 h, IV or IM	May need to add amoxicillin* or vancomycin† Not available in oral form.
Third-choice		
Ciprofloxacin	20-30 mg/kg/day divided q 12 h, PO, IV, or IM	May need to add amoxicillin* or vancomycin† Effective against gram-negative organisms and atypical bacteria. Also effective against *Pseudomonas* species but poor coverage against gram-positive organisms (including *Staphylococcus aureus* and nonhemolytic streptococci).
Levofloxacin	10 mg /kg/day, PO, IV, or IM	May need to add amoxicillin* or vancomycin† Effective against gram-negative organisms and atypical bacteria. More gram-positive coverage than ciprofloxacin.
Gatifloxacin	10 mg/kg/day, PO or IV	Expanded spectrum against both gram-negative and gram-positive organisms. Can be administered as single agent.

* Dosage: 25–50 mg/kg/day divided q 8 h, PO

† Dosage: 40–60 mg/kg/day divided q 6–8 h, IV

Table 6.9. Threshold radioactive exposures and recommended prophylactic single doses of KI

	Predicted thyroid exposure (Gy)	KI dose (mg)	No. of 130-mg tablets	No. of 65-mg tablets
Adults >40 yr old	≥5	130	1	2
Adults 18–40 yr	≥0.1	130	1	2
Pregnant or lactating women	≥0.05	130	1	2
Adolescents 12–18 yr old	≥0.05	130	1	2
Children 3–12 yr old	≥0.05	>65	½	1
Children 1 mo to 3 yr old	≥0.05	32	¼	½
Neonates birth to 1 mo old	≥0.05	16	1/8	¼

Adapted from U.S. Department of Health and Human Services, FDA, Center for Drug Evaluation and Research. Potassium Iodide as a Thyroid Blocking Agent in Radiation Emergencies. Available at http://www.fda.gov/cder/guidance/4825fnl.htm.

Table 6.10. Radionuclide specific therapies

Radionuclide	Therapy	Mechanism of action
Iodine	KI	Blocks thyroid deposition
Cesium/Rubidium/Thallium	Prussian blue	Blocks GI absorption and prevents recycling
Plutonium and transuranics (Americium, Californium, Lanthanum, Curium)	DTPA	Acts as chelating agent
Uranium	Bicarbonate	Alkalinizes urine, decreases chances of acute tubular necrosis
Tritium	Forced fluids, diuretics	Dilutes isotope
Strontium	Oral aluminum phosphate or barium sulfate	Blocks GI absorption
Radium	Magnesium sulfate lavage	Prevents absorption
Phosphorus	Stomach lavage, aluminum hydroxide, oral phosphates	Prevents absorption, blocks cellular deposition
Cobalt	Gastric lavage, purgatives; penicillamine if severe	Prevents absorption, acts as chelating agent

Source: National Council on Radiation Protection and Measurements. *Management of Persons Accidentally Contaminated with Radionuclides*. Report Number 65, 1979. Bethesda, MD. Reprinted with permission.

Table 6.11. Reduction in exposure by sheltering

Material – structure or location	Relative shielding strength
Outside	No reduction in exposure
Vehicle	No reduction in exposure
Wooden frame house	Slight reduction in exposure
Basement of wooden house	Almost 50% reduction in exposure
Masonry home	Almost 50% reduction in exposure
Basement of masonry house	>50% reduction in exposure
Large office or industrial building (inner areas, away from windows)	80% reduction in exposure

Source: Adapted from *Management of Terrorist Events Involving Radioactive Material*, Report 138, Table 8.5, p. 109. Bethesda, MD: National Council on Radiation Protection and Measurements; October 2001. Used with permission.

Chapter 7. Blast Terrorism

Introduction

In the short time between January and September 2003, explosive devices were involved in 73 of 189 terrorist events that occurred worldwide. A 1997 report by the Department of Justice found an abundance of evidence suggesting that given intent, the knowledge required to build bombs is readily available in print and on the Internet. The report cited at least 50 publications in the Library of Congress; several texts intended for military training, agricultural, and engineering use; 48 different underground pamphlets and publications; and countless sources on the World Wide Web. Bomb data from the FBI indicate that from 1987 to 1997, bombing incidents increased approximately 2.5 times, peaking in 1994 with 3,163 domestic bombing incidents. Of those incidents, 66% involved explosive devices, and the remaining 24% involved incendiary devices. More recent bomb data indicate that the number of domestic bombing incidents has decreased since 1994, with 1,797 incidents in 1999. In most incidents, low-explosive fillers were used. High-explosive ammonium nitrate mixtures, however, were used during the first World Trade Center bombing and the Oklahoma City bombing, highlighting the tremendous destructive power of a significant amount of a high explosive.

The raw materials for explosive devices are regularly found in areas of farming or mining activities. Due to the public accessibility of explosives materials and bomb-building knowledge, a domestic terrorist attack would probably take the form of a conventional explosive munitions attack. This chapter introduces the spectrum of injuries caused during an explosion and the differences between blast trauma and conventional trauma. Both blast trauma and conventional trauma have aspects of blunt, penetrating, burn, crush, and inhalational injuries. However, victims of a blast may suffer all of these injuries simultaneously, with additional injury caused by the blast wave itself, i.e., primary blast injury. Primary blast injuries are lethal, unique, and often subtle. Although the vast majority of blast injury victims suffer from conventional injuries, lack of knowledge about primary blast injuries and failure to recognize a blast's effect on certain organs can result in additional morbidity and mortality.

Explosives

Explosives are solid, liquid, or gaseous substances that, when detonated, transform rapidly into more stable products in the form of heat, gas, and energy. Explosives are frequently used in military, demolition, and industrial applications and are categorized as low explosives or high explosives. Low explosives are considered propellants and are used chiefly in small arms and munitions. Two examples are smokeless powder or black powder. High explosives have a greater potential for destruction due to their higher burning rate and therefore higher shattering effect. Notable examples of high explosives are TNT (trinitrotoluene), dynamite, RDX, C-4, ammonium nitrate, ammonium-nitrate fuel oil, HMX, and PETN. Due to their relative stability, these compounds require another explosive (e.g., a primer or detonator) to initiate a charge.

Blast Fundamentals

Understanding how an explosion causes tissue damage and injures victims requires some knowledge of the physics occurring during an explosion. Detonation of an explosive device causes rapid chemical conversion of an explosive material with the release of high-pressure gases and energy. These high-pressure gases expand supersonically, moving air particles, called the blast wind, and propagating in all directions in the form of a shock wave. The blast wind lasts milliseconds, and its strength varies with the strength of the explosive device detonated. Wind velocity can vary from 40 mph from the generation of 1 psi (pound per square inch) to 1,500 mph from a 100 psi detonation. As it expands, this shock wave causes compression of the ambient atmosphere and an instantaneous rise in atmospheric pressure above baseline, known as overpressure. The leading edge of the expanding blast wave is termed the blast front. As the wave of overpressure passes through, it subjects tissues in its path to enormous forces called blast-loading forces (measured in psi). The amount of force capable of perforating a tympanic membrane is 5 psi, and that considered to be lethal in 50% of exposures is 80 psi.

Pressure measurements of the blast wave recorded in one place reveal the pressure-time relationship of the waveform. Key observations include the following:

- Atmospheric pressure rises almost instantaneously to a peak overpressure as the wave passes through.
- Atmospheric pressure remains supra-atmospheric for some period of time and then decays to dip below the atmospheric pressure baseline, heralding the negative pressure phase.

Over time, atmospheric pressure returns to baseline. Due to the expansion of gases, a state of relative vacuum is created at the detonation site. This causes the gas flow to reverse during the negative phase—in contrast to the outward air flow occurring during the positive phase. All of the tissue injuries described by the blast wave have been due to overpressure. However, the pathophysiologic effects of underpressure are still under investigation, there has been some suggestion that underpressure itself can cause injury.

Many believe that the harmful effects on the body caused by a blast result from the pressure differentials exerted on tissues by the expanding wave. However, because the peak overpressure decays exponentially, a victim must be relatively close to the detonation for the blast wave itself to induce tissue injury. Several factors, including the following, affect the degree of blast pressure loaded to objects:

- The distance between the object and the detonation.
- The orientation of the object to the incident wave.
- The degree of reflected waves to which the object is subjected.

This latter point is the reason that, given equal peak overpressures, victims found in corners or in underwater blasts suffer greater injury. In both situations, the victim is subject to the incident wave in addition to multiple reflected waves.

The three physical properties that cause tissue damage during a blast are the spalling effect, implosion, and inertia.

Spalling effect. When a blast wave travels through tissue of homogenous density, it causes the tissue to vibrate. However, when a wave passes through air-filled tissue such as the lungs, intestinal lumen, or the middle ear, it travels from areas of higher density to the air-filled areas of lower density. The result is tension at the surface interface, and particles are thrown or spalled into the less dense medium. This causes micro- and macro-tears in the tissue wall, resulting in hemorrhage, edema, and the loss of structural integrity.

Implosion. In implosion, the blast wave is thought to cause compression of tissues (or organs), resulting in recoil and expansion of the tissue (or organ) as the wave exits. This causes structural damage to solid tissues principally at the areas of attachment, e.g., at the hila for solid organs.

Inertia. Blasts result in a wind with the ability to accelerate people and large stationary objects. Acceleration and deceleration causes multiple types of injuries, in particular blunt injuries. Experimental data have suggested that inertia may act at the level of tissues themselves. In the lungs, due to differing densities, bronchovascular elements would be expected to accelerate at different rates than delicate alveolar tissue, resulting in shearing at these sites.

Blast Trauma

Many mechanisms of injury are involved in blast injuries.

- Primary blast injury refers to tissue damage by the blast wave itself, specifically in areas with tissue-gas interfaces such as the lungs, the intestines, and the tympanic membrane.
- Secondary injury refers to penetrating or blunt injury that results from the acceleration of shrapnel or debris. Terrorists often add metallic fragments such as nails to devices to maximize the potential for penetrating injuries. Secondary injury is the most common type of injury seen, because it does not require the victim to be near the point of detonation.
- Tertiary injuries result from acceleration-deceleration forces imposed as the blast wind propels the victim. As the body is tumbled on a rigid surface, it suffers from blunt injury, in particular closed head injury, as well as penetrating injuries as it is accelerated over sharp debris.
- A fourth mechanism includes flash and flame burns, inhalational injury, and crush injuries incurred from fires and structural collapse.

Secondary and tertiary injury overlap significantly, and both are more common than primary blast injury. However, primary blast injuries are the most severe.

The effects of the blast wave on structural elements and on human tissues combine to cause complex combinations of injuries in blast victims; injuries are variable within one event. The principal factor that determines severity of injury is the distance of the victim from the site of detonation (Table 7.1). Injuries also vary due to the victim's position with

respect to incident waves and the degree of reflected shock waves to which the victim is exposed.

Primary blast injury. Primary blast injuries (PBI) are injuries caused specifically by exposure of the body to the blast wave. Pulmonary barotrauma, air embolization, and intestinal perforation are the unique principal causes of death after a blast. Although most injuries in an explosion are secondary, tertiary, and miscellaneous (crush, burn, inhalational), a person close enough to a detonation would be subjected to the effects of the blast on a microscopic level.

Urban bomb blasts tend to have the following characteristics:

- Most victims sustain minor injuries.
- Most injuries affect the head, neck, and extremities.
- Torso injuries are uncommon yet lethal.
- Primary blast injuries are uncommon because victims tend to die before arriving at the hospital.

However, because all bomb blast incidents are different, the types of injuries seen are variable. A blast that occurs in an enclosed space, such as a bus, is associated with more severe injuries and a higher incidence of primary blast injuries. The number of casualties would be expected to be less than in an equipotent detonation in open space. Mortality is also higher when a blast occurs in an enclosed space, because the shock wave is contained and reaches a higher overpressure and a longer positive phase. However, containment of the wave does not affect the generation of propelled debris. Therefore, secondary and tertiary injuries, including amputations from large objects, are the same whether the blast occurs in an enclosed space or open air.

The spectrum of PBI reflects involvement of the gas-containing organs and the pathophysiologic effects of these organs on other systems (Table 7.2). As in conventional trauma, all victims should be managed with careful attention to the airway, breathing, and circulation; however, in certain patients, complications may arise with respect to positive-pressure ventilation and fluid resuscitation management.

Blast lung injury and air embolization. The anatomic structure of the lung makes it susceptible to the effects of blast barotrauma. Alveolar spaces are engulfed by delicate capillaries in a way that maximizes the surface area available for gas exchange.

Pathophysiology. The pathophysiology induced by the blast involves the spalling of particles across the tissue-gas interface (alveolus) with the generation of micro-tears. This fills the air space with blood, edema, and tissue particles, impairing gas exchange. The most common lesion of the airway is the stripped-epithelium lesion, in which the bronchial epithelium and mucociliary apparatus are stripped from the basal lamina, resulting in ulcerations of the submucosa and impaired clearing of secretions. Structural tears occur through interfaces of blood vessels and air spaces, creating direct openings where air bubbles could escape into the circulation.

Clinical findings and diagnosis. Clinically, blast lung injury is evidenced by various degrees of the following:

- Hemoptysis.
- Hypoxia.
- Hemothorax.
- Dyspnea.
- Tachypnea.
- Chest pain.
- Cough.
- Wheezing.
- Rales/crackles.
- Decreased breath sounds.
- Pneumothorax.
- Hypopharyngeal hemorrhage.
- Subcutaneous crepitus.
- Tracheal deviation.

Clinical findings range from contusion and ecchymosis to massive hemoptysis, severe ventilation/perfusion mismatch, and air leak, leading rapidly to death. Most blast lung injury develops early in the course of treatment, within 1–2 hours. Signs and symptoms may progress within 24–48 hours to respiratory failure or acute respiratory distress syndrome (ARDS), or both. Respiratory failure is often due to secondary additive effects such as shock, organ failure, or inhalation of smoke and toxic substances.

The most important diagnostic test for blast lung injury is a chest radiograph. However, in stable patients, CT scans provide important additional information. Pulmonary hemorrhage is the most consistent microscopic finding in blast lung injury, and most survivors of a blast will have infiltrates on a chest radiograph.

Treatment and complications. Blast lung injury is not universally fatal, given aggressive and timely management. Initial management involves maximizing oxygenation and minimizing additional barotrauma. Most important is maintaining a patent airway, free of blood and secretions. Victims should be placed on oxygen to prevent hypoxia. Control of massive hemoptysis involves tracheal intubation and, whenever possible, selective ventilation of the contralateral lung. The source of bleeding in massive hemoptysis may be from one or both lungs and is often difficult to determine. Having a high index of suspicion for pneumothorax or tension pneumothorax cannot be overstated. The risk is so great that prophylactic tube thoracostomy has been suggested.

The development of systemic air embolization from injured lung tissue is a grave complication. The greater the degree of lung injury, the higher the risk of emboli formation. Although the actual incidence is unknown and is probably underrecognized, air embolization in blast injury is speculated to be the main cause of death within the first hour after a blast. Air emboli in the vascular system carry a high mortality rate because the air bubbles can potentially cause occlusion of the coronary arteries (myocardial ischemia), cerebral vessels (stroke), or cardiac outflow tracts (shock). They cause

additional morbidity in the nature of blindness (occlusion of retinal arteries) and ischemia of end organs. The ultimate clinical result depends on the site of embolization. Signs and symptoms that suggest arterial air embolization include the following:

- Air bubbles in retinal vessels.
- Blindness.
- Chest pain.
- Arrhythmia.
- Myocardial ischemia.
- Focal neurologic signs.
- Seizures.
- Loss of consciousness.
- Vertigo.
- Livedo reticularis.
- Tongue blanching.

Air emboli pose a challenge in emergency management of blast victims. Air emboli are not only difficult to diagnose, but also have a clinical presentation similar to that of other more familiar clinical entities. For example, myocardial ischemia, which is usually easily recognized, is most likely to be secondary to coronary vessel embolization (versus the traditional mechanisms of ischemia) in victims with blast lung injury. Management of these patients should focus on halting the passage of air. However, in patients exhibiting a change in their mental status, more common traumatic causes (e.g., intracranial hemorrhage from blunt head injury) should be addressed first, before focusing on embolization.

Air emboli can be confirmed by direct visualization of air bubbles or disrupted air passages via echocardiography, transcranial Doppler, CT scan, or bronchoscopy. Unfortunately, there are no data on the sensitivity of these techniques in detecting emboli in blast victims. Transesophageal echocardiography can detect gas bubbles as small as 2 μm, but its availability is limited. Sudden circulatory or neurologic collapse, especially if PPV has been started, combined with a high index of suspicion, is enough to make the diagnosis of air embolization until proved otherwise. Other suggestive clinical findings include possible evidence of bubbles in retinal vessels, aspiration of air from arterial lines, or marbling of the skin or tongue.

In conventional penetrating and blunt lung injuries, management of massive air embolization involves thoracotomy on the affected side to stop the passage of air. Based on this experience, management of air embolization in blast lung injury has also been primarily surgical. However, in blast lung injury, identifying the source of emboli may be difficult, since both lungs or multiple sites may be involved. Temporarily placing patients in specific positions to trap air bubbles anatomically (to prevent them from entering the circulation) has been suggested. However, there is no single maneuver that prevents air from entering both the arterial and venous circulation simultaneously. Despite the lack of data, placing the patient in a modified left decubitus position (more toward prone) or prone position is thought to be the most anatomically logical alternative. These positions place the coronary ostia in the lowest position in the body and the left atrium in the

highest position. Because of the practical limitations of having patients in these positions, placing the patient with the injured lung down, or in the dependent position, to minimize embolization by increasing venous pressures on that side, has also been suggested.

Hyperbaric oxygen therapy has been successfully used to treat cerebral air emboli from diving decompression injuries by actually causing bubble volume to decrease. Again, recommendations for management of blast-induced air embolization are largely based on conventional trauma experience. Stopping the passage of air bubbles into the circulation is paramount; however, doing so by surgically clamping the hilum when the injury is not clearly unilateral may not be necessary. Knowledge of lung isolation techniques is important for patient management.

Positive-pressure ventilation (PPV) is a last resort for blast victims; it is reserved for cases of severe respiratory failure or massive hemoptysis, or for patients requiring emergency surgery for other reasons. Cardiovascular, respiratory, or neurologic collapse within minutes of PPV being instituted has been reported. In addition, PPV is thought to contribute to the generation of air emboli due to the high airway pressures it causes, and it has been implicated in the later reopening of fistulas.

In the spontaneously breathing patient, pulmonary venous pressures are higher than airway pressure, which prevents the passage of emboli into the venous system. During PPV or when pulmonary vascular pressures are low (e.g., with hypovolemia), airway pressures are higher, and the gradient is reversed, facilitating the passage of air and debris into the vascular system. Techniques based on experience in ventilating patients with pulmonary contusion and ARDS have been proposed for ventilating victims of blast lung injury who must be intubated. These techniques include low peak inspiratory pressures (<35–40 cm H_2O), low tidal volumes, high peak end-expiratory pressures, permissive hypercapnea, high-frequency jet ventilation, pressure-controlled inverse-ratio ventilation, and nitric oxide inhalation. As a last heroic attempt, extracorporeal membrane oxygenation has been suggested.

Gastrointestinal blast injury. After lung injury, GI injury is the second most lethal injury after a blast. Abdominal injuries secondary to open-air blasts are less common than blast lung injury; however, they are much more common in underwater blasts.

Pathophysiology. The pathophysiology of GI injury is similar to that of blast lung injury, and abdominal injuries are a significant cause of delayed mortality. As the wave impacts the abdominal cavity, it compresses and distorts the internal tissues, resulting in hemorrhage and/or rupture of solid organs. As the blast wave passes through tissues with a gas interface, it causes spalling of particles into the intestinal lumen. The terminal ileum and colon are predisposed to injury because they contain the greatest amount of air, while the small intestine is relatively spared. The resultant wall tears, intramural hematomas, and hemorrhage may predispose the intestine to perforation. In severe blasts, the direct force of the wave itself may also cause perforation, although it is unknown whether the perforation is immediate or delayed.

Clinical findings and diagnosis. The signs and symptoms of GI injury may be nonspecific and change over time. They include the following:

- Lack of bowel sounds.
- Hematochezia.
- Hypotension.
- Involuntary guarding.
- Rebound tenderness.
- Abdominal pain.
- Nausea and vomiting.
- Orthostasis or syncope.
- Testicular pain.
- Tenesmus.

Evaluation of the abdomen begins with a physical examination, standard trauma screening laboratory tests, and a high index of suspicion for injury. Making the diagnosis of perforation in an area of trauma is challenging for many reasons. First, the findings can be subtle and masked by other, more critical injuries. Second, the patient may be unconscious, making the value of serial examinations limited. Third, diagnostic examinations, although useful for detecting hemorrhage, may be misleading or insensitive in the early stages of perforation.

Management. The goals of management are to identify and control internal bleeding and to identify and repair any perforated viscus. In stable patients in whom injury is suspected, the abdominal radiograph has largely been replaced by CT scan, ultrasonography, and diagnostic peritoneal lavage. CT scan provides useful information regarding intra-abdominal hemorrhage, organ injury, free intraperitoneal air, and intramural hematoma; however, it has a low sensitivity for identifying a hollow viscus perforation. In hemodynamically stable patients with blast lung injury too severe to be surgical candidates, exploratory abdominal procedures may be delayed. In these patients, broad-spectrum antibiotics are recommended pending confirmation of an intact bowel. Exploratory laparotomy may be necessary in hemodynamically unstable patients in whom internal bleeding is suspected. Because surgical outcomes in blast victims are poor, surgery, like intubation, is a last resort and should be weighed against the risk associated with missing a perforation.

Blast auditory injury. The auditory system is the system most frequently injured during a blast. Auditory injury is more common than lung or GI injury because the overpressure necessary to perforate tympanic membranes (5 psi) is well below that expected to cause lung or GI injury. Hearing loss either with or without a ruptured tympanic membrane is quite common. It can be debilitating and make communication with the victim difficult. Although some sensorineural hearing deficits improve over the first few hours, deficits are permanent in approximately 30% of victims.

Pathophysiology. Normally, sound pressure waves are transmitted from the tympanic membrane through the ossicular bones in the middle ear, where they are converted into mechanical vibrations. These mechanical vibrations, through the involvement of

perilymph in the membranous labyrinth, are converted again into nerve impulses at the organ of Corti in the cochlea. The blast wave overwhelms this intricate and delicate system and is then amplified down the conductive and sensory neural pathways. The result can be injury to any of part of the auditory system, including perforation of the tympanic membrane, disruption of the ossicular chain, or damage to the organ of Corti.

In addition to the damage caused by amplification of the shock wave within the auditory system, the instantaneous rise in overpressure inflicts additional damage. The ability to equilibrate middle ear pressure via the eustachian tube is overwhelmed. The resulting expansion of the cavity and distortion of tissue causes additional mechanical injury.

Tympanic membrane rupture. Tympanic membrane rupture from most causes heals spontaneously, with 10% of the eardrum expected to heal per month. Spontaneous healing has been observed in perforations involving <80% of the membrane. Complications include failure to heal (10-20%), infection, and cholesteatoma formation.

Despite the long-held belief that tympanic membrane perforation is a marker for delayed onset of pulmonary and GI blast injuries, this does not appear to be the case based on outcomes of 142 survivors of suicide bombings. Of the survivors who had perforated tympanic membranes, none developed delayed presentations of other primary blast injuries. Those with pulmonary blast injury were acutely ill early in their course, and none had a delay in presentation of respiratory symptoms. Notably, 18% of those with blast lung injury did not have perforated tympanic membranes. Therefore, isolated tympanic membrane rupture does not seem to be a marker for delayed onset of other primary blast injuries. Regardless, due to the paucity of data, and in particular the lack of pediatric data, it would seem prudent to delay the implementation of official clinical guidelines regarding the management of pediatric victims of auditory blast injury. Evaluation and management should be done on a case-by-case basis.

Ossicular chain and cochlear injury. Ossicular bones attach to the tympanic membrane and transmit its vibrations to the cochlea, where the vibrations are converted into neural impulses via tiny hair cells. The overpressure can cause distortion and fracture of the ossicular bones, but this is rare, and blasts causing inner-ear damage, yet sparing the ossicular bones have been reported. The relative resilience of the ossicular bones is not shared with the cochlea at the organ of Corti. This delicate system is overwhelmed by the amplification of waves, causing loss of structural integrity with damage of the inner and outer hair cells. The result is the development of conductive and/or sensorineural hearing loss.

Clinical findings. Victims may be initially unaware of acoustic injury or may complain of tinnitus, hearing loss, or ear pain. The spectrum of clinical findings in auditory blast injury includes the following:

- Tinnitus.
- Otalgia.
- Tympanic perforation.
- Ossicular chain disruption.

- Ossicular chain fracture.
- Labyrinthine fistula.
- Perilymphatic fistula.
- Loss of hair cell integrity.
- Conductive hearing loss.
- Sensory hearing loss.
- Basilar membrane rupture.

Management. In general, emergency management of auditory injuries involves clearing the ear canal of debris, minimizing exposure to loud noises or water, and taking precautions against infection. Otolaryngology followup for a complete evaluation and to watch for future complications (e.g., cholesteatoma formation) is recommended. Prophylactic use of antibiotics is not recommended.

Cardiovascular effects. The heart and blood vessels can be directly or indirectly injured by a blast wave. Cardiac involvement during a blast usually manifests as coronary vessel embolization and ischemia. Blood vessels within certain organs have a propensity for injury and may contribute to the generation of microthrombi, which results in disseminated intravascular coagulation. Cardiac blast injury that manifests as hemorrhage in the epicardium, myocardium, or papillary muscles is quite rare.

Hypotension in blast victims often has multiple causes. It can involve blood loss from major musculoskeletal or abdominal injury or from a blast-related, vagally mediated reflex. This reflex, which is seen immediately after a significant blast exposure, causes hypotension without vasoconstriction and bradycardia. It is the most common effect on the cardiovascular system by the blast wave itself. The hypotension can be profound and self-limiting, and there may be no external signs of injury.

Traditionally, aggressive volume replacement to support circulation is required in trauma victims with cardiovascular collapse. However, excessive volume replacement is detrimental to patients with lung injury. Research in animals has suggested that fluid replacement actually impairs cardiovascular performance in the setting of a blast. However, inadequate pulmonary vascular pressure has been suggested to promote the passage of air into the pulmonary venous system. Therefore, administration of fluids in increments of 5 mL/kg, titrated to clinical response, has been recommended. Either too much or too little fluid can be harmful, and the judicious use of fluids to maintain euvolemia is probably the best approach. As in all trauma patients, colloids should be used to replace massive hemorrhage, improve oxygenation, and minimize oncotic fluid shifts. Monitoring of central venous pressure and/or pulmonary pressures is a useful adjunct to fluid management.

Sentinel injuries. Sentinel injuries are subtle injuries that can increase the risk of having or developing serious blast injury. These patients should be monitored closely, no matter how clinically well they appear. Sentinel injuries include the following:

- Traumatic amputations.
- Hypopharyngeal contusion.

- Hemoptysis.
- Subcutaneous emphysema.
- Hearing loss.
- Ruptured tympanic membrane.

Traumatic amputations frequently result from a blast. However, they are rarely found among survivors because the overpressure needed to cause such injury more often than not causes death at the scene. The mechanism for traumatic amputations has been hypothesized to be a combination of the blast wave itself, the effect of propelled fragments on tissues, and the added force of the blast wind.

Incendiary Weapons

Incendiary devices are fire-bombs used to cause maximal fire damage to flammable objects including people, buildings, and equipment. The massive fire caused by these devices effectively and easily causes mass panic, and the relative ease of obtaining materials to fabricate some devices makes them a potential vehicle for terrorist attacks. Incendiary devices range from simple Molotov cocktails, which are gasoline-filled bottles ignited with a rag, to military bombs that contain flammable materials such as napalm. Modern incendiary devices are complex bombs with flammable substances (e.g., kerosene) that can be ignited by either a fuse or a primary explosive.

Napalm was originally made by mixing an aluminum soap of naphthalene and palmitate with gasoline. The result was a sticky flammable substance that, in contrast to liquid flammables, would stick to the intended target and burn longer. Napalm was later modified to contain mixtures of benzene, gasoline, and polystyrene. Napalm stores were eventually destroyed; however, incendiary devices containing napalm have allegedly been used in the 1990s in northern Iraq and Croatia. Incendiary devices containing mixtures of kerosene still have military applications.

The principal incendiary agents are thermite, magnesium, white phosphorus, and hydrocarbons. Based on FBI data for 1999, most of the devices involved in incendiary bombing incidents (223 actual bombings and 114 attempts) in the United States used gasoline as the flammable agent.

Emergency management of victims of incendiary devices involves identifying and treating the following:

- Severe burns (second- and third-degree).
- Respiratory compromise.
- Carbon monoxide poisoning.
- Dehydration.

These burn victims should be managed as any other burn victim, with special attention to identifying blast injuries and removing the incendiary agent from the skin. Carbon monoxide poisoning is of particular concern in napalm exposure, because carbon

monoxide is a byproduct of napalm combustion. Due to the radiant heat emitted from the combustion of these materials, prolonged exposure may lead to severe dehydration.

Aviation Terrorism

The Aviation and Transportation Security Act, signed into law on November 19, 2002, instituted several measures with the goal of making flying safer, including the following:

- Creating the Transportation Security Administration (TSA).
- Screening of all baggage for explosive materials.
- Fortifying of cockpit doors.
- Increasing the number of sky marshals aboard planes.
- Training flight crews on management of hijacking incidents.

According to TSA data, 100% of baggage is now screened (compared with 5% prior to September 11, 2001). By August 2003, the TSA had intercepted 2.4 million knives, nearly 1,500 firearms, and more than 51,000 box cutters.

Trauma Systems

The medical response to blast terrorism is built on the foundation of the regional trauma system. About 98% of all terrorist events worldwide involve physical trauma, and approximately 75% of all terrorist events are due to blast trauma. Therefore, regional emergency management, public safety, and public health agencies should include not only regional child health care experts, but also regional pediatric trauma professionals in planning for mass casualty events that could affect children. Blast terrorism, like all other mass casualty events, needs to be directed with an Incident Command Structure (see Chapter 2, Systems Issues).

Trauma Hospitals

Most trauma hospitals are full-service general hospitals that provide the highest level of health care service in their communities. However, modern trauma system design does not rely solely on such hospitals but integrates all health facilities within the region to the level of their resources and capabilities. Thus, the complete trauma system should consist of an integrated network of health care facilities within a region, designed for safe and rapid transport of injured patients to the health care facilities that best meet their medical needs. As of April 2002, 35 States had formally designated or certified trauma centers, while the remaining States had at least one verified trauma center, which is the key element recognized as essential to trauma systems. Many stand-alone pediatric hospitals also serve as "pediatric trauma centers."

Trauma Centers

Trauma centers are general hospitals that are committed, both institutionally and financially, to priority care of injured patients. Emergency medicine physicians and emergency trauma surgeons are the primary care providers within the context of the trauma center, and they provide appropriate information and followup to each patient's usual primary health care provider. Emergency medicine physicians begin evaluation and

management and immediately involve emergency trauma surgeons whenever injuries meet any of the following criteria:

- Are multiple or severe.
- Require support of a full trauma team, based on previously established trauma triage criteria or scores.
- Would benefit from trauma consultation with an emergency trauma surgeon

Trauma centers should have the following attributes:

- Designated as such by emergency medical and public health authorities within the region, based on self categorization according to established standards.
- Followed by on-site peer verification by impartial trauma experts.
- Subject to ongoing review of performance and participation in the regional trauma system.

All trauma centers have key organizational characteristics in common:

- All trauma services should be led by a properly qualified and credentialed emergency trauma surgeon who has education, expertise, and experience in trauma care.
- This emergency trauma surgeon, together with a trauma nurse program manager and trauma registrar, should maintain active programs of continuing education and performance improvement for all members of the trauma service.
- Trauma care should be provided by properly qualified and credentialed physician specialists in general or pediatric emergency medicine, general or pediatric trauma surgery, anesthesiology, radiology, pathology, and the three core surgical subspecialties (critical care, neurologic surgery, and orthopedic surgery).
- This physician team should work in collaboration with properly qualified and credentialed nursing personnel.
- Appropriate physical resources should include properly equipped emergency departments, operating suites, intensive and acute-care units, imaging capabilities, laboratory facilities, and blood bank.
- The in-house trauma team should be available immediately, 24 hours per day, 7 days per week.
- Appropriate and culturally competent mental health, social work, pastoral care, injury prevention programs, and ideally, professional education and trauma research programs should be in place to serve both patients and the community.

Level One Trauma Centers. Level One Trauma Centers offer comprehensive care of seriously injured patients that includes specialists and services for resuscitation, recovery, and rehabilitation. They usually are located in full-service general or university hospitals or, in the case of children, in full-service children's hospitals in which comprehensive care of the trauma patient is part of the institutional mission. The key issue is comprehensive, readily available, and consistent care of injured patients by all needed specialists and services.

Level Two Trauma Centers. Level Two Trauma Centers provide most specialists and services that are available in Level One Trauma Centers, but typically they are located in

full-service general hospitals that do not support medical or nursing educational programs (e.g., residency training) or trauma research. Patient care remains exemplary, and community outreach activities are a key part of the hospital's mission. Most Level Two Trauma Centers are located in large urban areas that are served by an academic medical center but with a sufficiently large population to require a second full-service trauma center, or they are in mid-sized urban areas that are not served by an academic medical center. In the latter situation, the Level Two Trauma Center acts as the regional trauma center, serving as the tertiary referral center for Level Three and Four Trauma Centers, as well as for non-trauma centers and other facilities within the region.

Level Three Trauma Centers. Level Three Trauma Centers provide most trauma care in the United States. They typically are located in community hospitals that serve small urban or large suburban areas. Key specialists and services are available, suitable for managing patients with injuries of a single system and few comorbidities. However, medical and surgical subspecialist coverage may be limited, and patients with multiple or severe injuries, with complex comorbidities, or who are very young or very old are usually transferred to a nearby Level One or Level Two Trauma Center after initial stabilization. Most Level Three Trauma Centers play an integral role in the regional trauma system and collaborate with a Level One or Level Two Trauma Center within the region. Again, patient care is exemplary, within the resources of the hospital and the community. Community outreach is essential, particularly in terms of support for the typically volunteer local emergency medical service agencies that serve the area.

Non-Trauma Centers

All facilities that receive emergency patients, including hospitals and free-standing diagnostic and treatment clinics, should have the capabilities for resuscitating and stabilizing injured patients of all ages. Therefore, protocols should be in place for sustentative trauma care (including education of medical and nursing staff in early care of injured patients) and for identification of patients in need of transfer to hospitals capable of providing definitive trauma care (which should be known to all urgent care personnel through prior development of formal transfer agreements). All such facilities should be:

- Considered part of the regional trauma system.
- Prepared to provide, within their communities, anticipatory guidance related to injuries that is consistent with programs advocated by regional experts in injury prevention.
- Participants in regional programs for performance improvement of community trauma care, with special emphasis on the outcomes of patients transferred to local trauma centers.

Treatment

Treatment of blast trauma involves full integration of the regional EMS system and the regional trauma system, in accordance with plans developed in collaboration with regional public safety and emergency management agencies. Although most blast trauma is caused by explosive or incendiary agents, the possibility of other weapons of mass destruction (WMD), such as biological, chemical, or nuclear weapons, should always be

considered (see Chapter 4, Biological Terrorism; Chapter 5, Chemical Terrorism; and Chapter 6, Radiological and Nuclear Terrorism).

Trauma

The treatment of victims of major trauma, including blast trauma, follows well-established protocols. The American College of Surgeons Committee on Trauma has developed and disseminated such protocols through its support of the *Advanced Trauma Life Support® for Doctors* Course (see Table 7.3). The Emergency Nurses Association and the Society of Trauma Nurses have undertaken like responsibilities for nurses through the *Trauma Nursing Core Course* and the *Advanced Trauma Care for Nurses* Course. All three courses focus on a practical approach to the initial care and management of the injured patient, assuming no special knowledge of trauma care, including the steps to be taken during the "golden hour" of trauma care—the critical first hour after injury has occurred.

Burns

Major burns and major trauma are often seen together in victims with injuries caused by explosive or incendiary devices. The treatment of victims of major burns also follows well-established protocols. Specific education on the initial resuscitation of these victims is included in both the *Advanced Trauma Life Support® for Doctors* course (American College of Surgeons Committee on Trauma) and the *Advanced Burn Life Support* course (American Burn Association) (see Table 7.3, Table 7.4).

Multiple Casualties

The strict definition of a multiple casualty incident is an incident involving more than one casualty that overwhelms the capacity of emergency medical providers at the scene. In general, this happens when a local EMS system must care for five or more victims who have the same illness or injury at the same place and time. Because local hospital emergency departments may also be overwhelmed by such events, EMS systems usually attempt to transport multiple victims to several hospitals in the vicinity of the event when feasible. In such circumstances, attempts are usually made to transport members of the same family to the same hospital, particularly if ill or injured children are involved. However, the availability of specialized pediatric health care resources, such as children's hospitals, may justify preferential transport of pediatric victims of multiple casualty incidents to these facilities.

Mass Casualties

The strict definition of a mass casualty event is an event involving large numbers of casualties, generally 20 or more, that overwhelms and disrupts the resources and capabilities of the entire regional trauma and EMS systems to provide immediate care for all ill or injured victims. This situation develops when the need for ambulances, hospitals, or both exceeds the emergency resources of the regional health care system. The definition further implies the following:

- The need to activate regional disaster plans that mobilize all available ancillary resources to assist with providing emergency medical care. This includes using the surge capability of both the regional EMS system to deploy extra ambulances (via mutual aid agreements) and of the regional hospital system to maximize the number of victims who can be cared for by opening spare beds, discharging stable patients, canceling elective procedures, and conscripting off-duty staff.
- The need to prioritize care such that those at greatest risk of loss of life or limb are treated first (unless they are unlikely to survive). The most widely used pediatric resource is JumpSTART, modified by Romig (www.jumpstarttriage.com) from the Simple Triage and Rapid Treatment (START) triage system used for adults.

Planning and Mitigation

The approach to planning for the possibility of blast injury after a terrorist attack should combine knowledge of the epidemiology of blast injury with awareness of the resources available to the regional trauma system. The Federal Government has adopted a similar approach for routine trauma system planning that allies the regional public health system with the regional health care system to form regional partnerships for the purpose of developing and implementing comprehensive injury control strategies at the community level.

Medical disaster planning should fully integrate regional public health agencies, regional health care organizations, EMS, emergency departments, and trauma centers before a disaster occurs. Public health officials and trauma care professionals should collaborate to evaluate, and redesign if needed, each system component for optimal performance.

Current regional trauma system design maintains an artificial separation between the pre-event, event, and post-event phases of injury control. The comprehensive public health approach to regional trauma system design integrates all phases of injury control into a single system. Regional injury control systems that have adopted such an approach (e.g., San Diego County, CA) have seen steady improvement in the quality of their injury prevention programs and the outcomes of their trauma patient care.

Public health reasons to apply this approach to blast terrorism include the documented lack of public health preparedness of most regions for terrorist attacks, despite excellent resources that describe the necessary elements for treatment of victims.

Planning

The enormous variability in the following characteristics hinders comparative analysis, and hence accurate prediction, of needs and resources for victims of blast terrorism:

- Type, quality, quantity, force, and delivery (human, bicycle, motorcycle, car, truck, plane) of explosive.
- Environment (closed space vs. open air).
- Time (day vs. night).
- Distance (proximate vs. distant).

- Circumstances (weather conditions, hazardous materials, etc.).
- Protection (clothes, barriers, etc.).
- Sequelae (structural collapse, structural fire, etc.).
- Victims (ages, number, density).

In general, small, frequent blasts in open air usually result in less serious injury than large, single blasts in closed spaces, which historically have resulted in life-threatening injury.

Regional trauma system planning should also consider the special needs of children who are injured due to blast terrorism and the special resources needed to care for them. Children and young adults are at higher risk of serious injury than adults for several reasons (see Children Are Not Small Adults in Chapter 1). Specific to blast trauma is that while blast tolerances in children are poorly defined, there is good reason to believe that children may absorb more blast energy per unit body mass than adults after blast trauma. This predisposes children to morbidity and mortality rates higher than those of adults as compressive shock waves passing through the body are compacted into a smaller total body mass.

Mitigation

Because most blast terrorism in recent years has involved children, with the notable exceptions of the terrorist airliner attacks on the World Trade Center in New York and the Pentagon in Washington on September 11, 2001, significant personal experience has been gained with pediatric disaster and emergency preparedness and management by child health professionals. Reports in the literature (summarized below) point out the woeful state of emergency preparedness for disasters that involve children. They also describe the common problems in pediatric disaster planning and management such that pediatric professionals involved in disaster planning will be knowledgeable about these problems and thus can seek to anticipate and thereby avoid them in future disasters.

In the Avianca jetliner crash in New York in January 1990, 22 of 25 (80%) children survived versus 70 of 132 (50%) adults, despite the fact that pediatric patients were inadequately treated and transported (State, regional, and county disaster plans did not address pediatrics). Only three children died, and only seven survivors sustained high-risk injuries. The spectrum of injuries resulting from this event were as follows:

- A 3-month-old boy with intracranial bleeding and aortic rupture (died).
- A 5-year-old boy with massive hemothorax (died).
- A 7-year-old boy with severe traumatic brain injury (died).
- Six children with traumatic brain injury.
- Five children with hypotensive shock.
- Three children with femur fractures with either hypotensive shock or traumatic brain injury.

Triage and transport of pediatric patients:

- Of seven children with a pediatric trauma score (PTS) <8, only one was taken to a Level I Pediatric Center.

- Of five high-risk children (greater risk of death) initially taken to a Level III Pediatric Center, only two were subsequently transported to a higher level Pediatric Center.
- Two high-risk patients and one low-risk patient (low risk of death) were transported by helicopter.

After the bomb blast that destroyed the Alfred P. Murrah Federal Building in Oklahoma City, OK, in April 1995, there were 816 casualties, including 66 children. Of these, 19 children died, and 47 survived. Of the 20 children in the day care center who were seated by windows, 16 died and 4 survived. The spectrum of injuries resulting from this event was as follows:

Of the 35 children who died—

- 90% had skull fractures, most with skull capping.
- Associated injuries: 37% trunk, 31% amputations, 47% arm fractures, 26% leg fractures, 21% burns, 100% soft-tissue injuries.

Of the children who survived—

- 15% required hospitalization.
- Documented injuries: two open depressed skull fractures with partially extruded brain, two closed head injuries, three arm fractures, one leg fracture, one arterial injury, one splenic injury, five tympanic membrane perforations, four burns (one burn >40% total BSA).

No children were injured in the terrorist airliner attack on the Pentagon on September 11, 2001, because the Pentagon daycare center was located on the opposite side of the building from the location of attack. However, as a result of the attack, issues were raised about children's hospital disaster preparedness. Immediately after the disaster, the hospital disaster plan was invoked, resulting in the discharge of more than 50 patients and the cessation of all nonurgent activities. Although hospital staff had conducted disaster drills in preparation for Y2K, hospital leaders continued to question their actual state of readiness. Emergency preparations were complicated by the fact that all of their news came not from official sources, but from local television, leaving hospital leaders unsure about what to expect.

These experiences highlight a number of vitally important issues regarding blast terrorism mitigation in children.

- After a blast, injuries in children are to be expected with most children injured in closed or confined spaces, which greatly increases the magnitude of forces of injury.
- As with blast injuries in adults, most children will either die at the scene or sustain minor injuries. Only a small number of children in the "penumbra" of the blast wind who sustain major injuries will survive to require hospital care, but typically they will not begin to arrive at the trauma center until 30–60 minutes after the blast event.
- Most surviving children with major injuries will require early surgery and subsequent care in a pediatric critical care unit, followed by lengthy hospitalization and rehabilitation, both physical and psychological.

Bibliography

Explosives, Incendiary Devices, and Aviation Terrorism

- Blackwell T. Medical response to terrorism (CD-ROM). University of North Carolina School of Medicine. Chapel Hill, NC. Available free from the National Association of EMS Physicians via e-mail to info-naemsp@goamp.com; request MRT CD-ROM.
- Bolz F, Dudonis K, Schulz D. Bomb incidents. In: *Counterterrorism Handbook—Tactics, Procedures, and Technique*.: Boca Raton, FL: CRC Press LLC; 2002;129–153.
- Cooper GJ, Maynard RL, Cross NL, et al. Casualties from terrorist bombings. *J Trauma*. 1983;23:955.
- Federal Bureau of Investigations Bomb Data Center. U.S. Department of Justice. Bomb Data Reports 1997-1999.
- Ho AM-H, Ling E. Systemic air embolism after lung trauma. *Anesthesiology*. 1999;90(2):564–575.
- Ho AM-H. Is emergency thoracotomy always the most appropriate immediate intervention for systemic air embolism after lung trauma? *Chest*. 1999;116(1):234–237.
- Hull JB, Cooper GJ. Patterns and mechanisms of traumatic amputation by explosive blast. *J Trauma*. 1996;40(3S):198S–205S.
- Irwin RJ, Lerner MR, Bealer JF, et al. Cardiopulmonary physiology of primary blast injury. *J Trauma*. 1997;43(4):650–655.
- Katz E, Ofek B, Adler J, et al. Primary blast injury after a bomb explosion in a civilian bus. *Annals Surg*. 1989;209:484–488.
- Kerr AG, Byrne JET. Concussive effects of a bomb blast on the ear. *J Laryngol Otolaryngol*. 1975;89:131–143.
- Leibovici D, Gofrit O, Shapira S. Eardrum perforation in explosion survivors: is it a marker for pulmonary blast injuries? *Annals Emerg Med*.1999;34(2):168–172.
- Liebovici D, Gofrit ON, Stein M, et al. Blast injuries: bus versus open-air bombings—a comparative study of injuries in survivors of open-air versus confined space explosions. *J Trauma*. 1996;41(6):1030–1035.
- Maxson RT. Management of pediatric trauma: blast victims in a mass casualty incident. *Clin Ped Emerg Med*. 2002;3(4):256–261.
- Mellor SG. The pathogenesis of blast injury and its management. *Br J Hosp Med*. 1988; 39:536–539.
- Ost D, Corbridge T. Independent lung ventilation. *Clin Chest Med*. 1996;17:591–601.
- Pahor AL. The ENT problems following the Birmingham bombings. *J Laryngol Otolaryngol*. 1981;95:399–406.
- Paran H, Neufeld D, Schwart I, et al. Perforation of the terminal ileum induced by blast injury: delayed diagnosis or delayed perforation? *J Trauma*. 1996;40(3);472–475.

- Phillips Y, Richmond DR. Primary blast injury and basic research: a brief history. In: Bellamy RF, Zajtchuk R (eds). *Conventional Warfare: Ballistics, Blasts, and Burn Injuries.* Washington, DC: Office of the Surgeon General of the US Army; 1991:221–240.
- Phillips YY, Zajtchuk JT. Management of primary blast injury. In: Bellamy RF, Zajtchuk R (eds). *Conventional Warfare: Ballistics, Blasts, and Burn Injuries.* Washington, DC: Office of the Surgeon General of the US Army; 1991:295–335.
- Pizov R, Oppenheim-Eden A, Matot I, et al. Blast lung injury from an explosion on a civilian bus. *Chest*. 1999;115(1):165–172.
- Virginia Department of Emergency Management. A Reporter's Guide to Terrorism. A Practical Guide to the Threat of Terrorism. Virginia Department of Emergency Management. Virginia Department of Emergency Management. 2001. Available at: http://www.vaemergency.com/newsroom/index.cfm. Accessed August 24, 2006.
- Riley D, Clark M, Wong T. World Trade Center terror: explosion trauma—blasts, burns and crush injury. *Topics Emerg Med*. 2002;24(2):47–59.
- Schardin H. The physical principles of the effects of detonation. In: *German Aviation Medicine. World War II.* Vol II. Washington, DC: Prepared under auspices of the U.S. Air Force Surgeon General; 1950:1207–1224.
- Sharpnack DD, Johnson AJ, Phillps YY. The pathology of primary blast injury. In: Bellamy RF, Zajtchuk R (eds). *Conventional Warfare: Ballistics, Blasts, and Burn Injuries*. Washington, DC: Office of the Surgeon General of the U.S. Army; 1991:271–294.
- Sorkine P, Szold O, Kluger Y, et al. Permissive hypercapnia ventilation in patients with severe pulmonary blast injury. *J Trauma*. 1998;45(1):35–38.
- Stein M, Hirshberg A. Medical consequences of terrorism. *Surg Clin No Am*. 1999;79(6):1537–1552.
- Stuhmiller JH, Phillips YY, Richmond DR. The physics and mechanisms of primary blast injuries. In: Bellamy RF, Zajtchuk R (eds). *Conventional Warfare: Ballistics, Blasts, and Burn Injuries.* Washington, DC: Office of the Surgeon General of the U.S. Army; 1991:241–270.
- Uretsky G, Coter S. The use of continuous positive airway pressure in blast injury of the chest. *Crit Care Med*. 1980;8:486–489.
- U.S. Department of Justice. 1997 Report on the Availability of Bomb Making Information. U.S. Department of Justice. April 1997.
- Virginia Department of Emergency Management. VDEM Terrorism Toolkit. Weapons of mass destruction—explosives. Available at http://www.vaemergency.com/threats/terrorism/toolkit/wmd.cfm. Accessed August 24, 2006.
- Wightman JM, Gladish SL. Explosions and blast injuries. *Annals Emerg Med*. 2001;37(6):664–678.
- Wikoff RP, Lerner MR, Mantor PC, et al. Fluid resuscitation is detrimental in a blast injury model. *J Trauma*. 1999;46(1):204.

Trauma Systems and Planning/Mitigation

- American Burn Association. *Advanced Burn Life Support Course*. Available at: http://www.ameriburn.org/ABLS/ABLSNow.htm. Accessed August 24, 2006.
- Briggs SM, Brinsfield KH, eds. *Advanced Disaster Medical Response*. Boston: Harvard Medical International, Inc., 2003.
- Committee on Trauma and Committee on Shock, Division of Medical Sciences, National Research Council, National Academy of Sciences. *Accidental Death and Disability: The Neglected Disease of Modern Society*. Washington: National Academy of Sciences, 1966.
- Committee on Trauma, American College of Surgeons. *Advanced Trauma Life Support® for Doctors 1997 Student Course Manual*. Chicago: American College of Surgeons, 1997.
- Committee on Trauma, American College of Surgeons. *Resources for Optimal Care of the Injured Patient 1999*. Chicago: American College of Surgeons, 1999.
- Cooper G, Dawson D, Kaufmann C, Esposito T, Cooper A, et al. *Trauma Systems Planning and Evaluation: A Model Approach to a Major Public Health Problem*. Washington: National Highway Traffic Safety Administration, 2004.
- Dieckmann R, Brownstein D, Gausche-Hill M, eds. *Pediatric Emergencies for Prehospital Professionals*. Sudbury: Jones and Bartlett Publishers, 2000.
- Farmer JC, Jimenez EJ, Talmor DS, et al, eds. *Fundamentals of Disaster Management*. Des Plaines: Society of Critical Care Medicine, 2003.
- Freishtat RJ, Wright JL, Holbrook PR. Issues in children's hospital disaster preparedness. *Clin Ped Emerg Med* 2002;3:224-230.
- Goldfrank LR, ed. Public Health. Washington: Institute of Medicine, National Academy of Sciences, 2001.
- MacKenzie EJ, Hoyt DB, Sacra JC, et al. National inventory of hospital trauma centers. *JAMA* 2003;289:1515–1522.
- Quintana DA, Jordan JB, Tuggle DW, et al. The spectrum of pediatric injuries after a bomb blast. *J Pediatr Surg* 1997;32:307–311.
- Society of Trauma Nurses. *Advanced Trauma Care for Nurses Course.* Available at: http://www.traumanursesoc.org/education.html.
- *Trauma Nursing Core Course*. Emergency Nurses Association.
- van Amerongen RH, Fine JS, Tunik MG, et al. The Avianca plane crash: emergency medical system response to pediatric survivors of the disaster. *Pediatrics* 1993;92:105–110.
- West JG, Trunkey DD, Lim RC. Systems of trauma care: a study of two counties. *Arch Surg* 1979;114:455–460.

Table 7.1. Expected injuries at relative distances from detonation in open air

Injury	Closest						Farthest
Body disruption	●						
Burn/inhalation	●						
Toxic inhalation	●	●					
Amputation	●	●	●				
Primary blast injury, lung and bowel	●	●	●	●			
Tertiary mechanism	●	●	●	●	●		
Primary blast injury, ear	●	●	●	●	●	●	
Secondary mechanism	●	●	●	●	●	●	●

Table 7.2 Spectrum of primary blast injury on body systems

System	Injuries
Respiratory	
	Pulmonary contusion
	Pulmonary hemorrhage
	Respiratory failure
	Stripped-epithelium lesion
	Alveolo-venous fistula
	Bronchopleural fistula
	Pneumothorax
	Traumatic emphysema
	Air embolism
	Acute respiratory distress syndrome
GI	
	GI hemorrhage
	GI perforation
	Spleen or liver hemorrhage or rupture
	Retroperitoneal hemorrhage
	Tunica albuginea (testis) hemorrhage
Auditory	
	Tympanic membrane rupture
	Ossicular chain disruption
	Cochlear nerve injury
	Labyrinthine fistula
	Perilymphatic fistula
Circulatory	
	Coronary embolism
	Myocardial contusion
	Thrombosis/disseminated intravascular coagulation
CNS	
	Cerebral air embolism
	Retinal artery embolism

Source: Sharpnack DD, Johnson AJ, Phillips Y. The Pathology of Primary Blast Injury. In: Bellamy RF, Zajtchuk R, eds. *Conventional Warfare: Ballistics, Blasts, and Burn Injuries*. Washington, DC: Office of the Surgeon General of the U.S. Army; 1991: 271-294.

Table 7.3 Principles of Advanced Trauma Life Support®

Primary Survey and Resuscitation	
Airway/cervical spine	Open: jaw thrust/spinal stabilization
	Clear: suction/remove particulate matter
	Support: oropharyngeal/nasopharyngeal airway
	Establish: orotracheal/nasotracheal intubation[1]
	Maintain: primary/secondary confirmation[2]
	Bypass: needle/surgical cricothyroidotomy
Breathing/chest wall	Ventilation: chest rise/air entry/effort/rate
	Oxygenation: central color/pulse oximetry
	Support: distress – NRB/failure – BVM[3]
	Chest wall: ensure integrity/expand lungs Tension pneumothorax: needle, chest tube[4] Open pneumothorax: occlude, chest tube Massive hemothorax: volume, chest tube
Circulation/external bleeding	Stop bleeding: direct pressure, avoid clamps
	Shock evaluation: pulse, skin CRT, LOC[5]
	Blood pressure: avoid over/undercorrection Infant/child: low normal = 70 + (age × 2) mmHg Adolescent: low normal = 90 mmHg
	Volume resuscitation: Ringer's lactate → packed cells Infant/child: 20 mL/kg RL, repeat x 1-2 → 10 mL/PRBC Adolescent: 1–2l, repeat 1–2 × → 1–2 U PRBC
Disability/mental status	Pupils: symmetry, reaction
	LOC: GCS Track and trend as a vital sign Significant change = 2 points Intubate for coma = GCS ≤8
	Motor: strength, symmetry
	Abnormality or deterioration: call neurosurgeon Mild TBI (GCS 14–15): observe, consider CT for history of LOC Moderate TBI (GCS 9–13): admit, obtain CT, repeat CT 12–24 hr Severe TBI (GCS 3–8): intubate, ventilate, obtain CT, repeat CT 12–24 hr
Exposure and environment	Remove clothing
	Logroll: requires four people
	Screening examination: front and back
	Avoid hypothermia: keep patient warm
Adjuncts	
	Foley catheter unless contraindicated[6]
	Gastric tube unless contraindicated[7]

Table 7.3 Principles of Advanced Trauma Life Support®, continued

Secondary Survey and Reevaluation	
	History and physical: SAMPLE history, complete examination
	Imaging studies: plain radiographs[8], special studies[9]

1 RSI technique: etomidate then succinylcholine
2 Primary: chest rise, air entry; secondary: exhaled CO_2 detector, esophageal detector device; watch for DOPE: **D**islodgement, **O**bstruction, **P**neumothorax, **E**quipment failure
3 NRB: nonrebreather mask; BVM: bag valve mask
4 Do not wait for confirmatory chest x-ray.
5 CRT = capillary refill time, LOC = level of consciousness; consider obstructive and neurogenic as well as hypovolemic shock: exclude tension pneumothorax, cardiac tamponade, spinal shock
6 Meatal blood, scrotal hematoma, high riding prostate
7 CSF oto/rhinorrhea, basilar skull fracture, midface instability
8 Chest, pelvis, lateral cervical spine; others as indicated
9 FAST, CT as indicated

Source: Adapted with permission from the American College of Surgeons Committee on Trauma, Advanced Trauma Life Support® for Doctors Student Course Manual.

Table 7.4 Principles of Advanced Burn Life Support®

Primary Survey	
Airway	Rapid upper airway closure may occur following inhalation injury and require early intubation
Breathing	Circumferential full thickness burns of the trunk may impair ventilation and require escharotomy
Circulation	Circulation in a limb with full thickness burns may be impaired and require escharotomy
Disability	Consider carbon monoxide poisoning if the patient is not initially alert and oriented
Exposure and environment	Remove all jewelry to avoid constriction of digits
Adjuncts	
Foley catheter	Needed to titrate fluid replacement against urine output
Gastric tube	Needed to decompress stomach due to burn ileus
Resuscitation	
Stop the burning process	
Fluid resuscitation	*Parkland formula*: 3–4 mL Ringer's lactate × body weight in kg × total BSA burned; administer half the amount during the first 8 hr after injury, half over the next 16 hr *Modification for pediatric patients:* Add maintenance fluid to Parkland formula for children and infants; 100 mL/kg for first 10 kg + 50 mL/kg for next 10 kg + 20 mL/kg for each additional kg
Secondary Survey	
History and physical	
Obtain as much information as possible regarding the circumstances of injury	***Flame*** How did the burn occur? Did the burn occur outside or inside? Did the clothes catch on fire? How long did it take to extinguish the flames? How were the flames extinguished? Was gasoline or another fuel involved? Was there an explosion? Was there a house fire? Was the patient found in a smoke-filled room? How did the patient escape? If the patient jumped out a window, from what floor? Were others killed at the scene? Was there a motor vehicle crash? How badly was the car damaged? Was there a car fire? Are there other injuries? Are the purported circumstances of the injury consistent with the burn characteristics?

	Scald How did the burn occur? What was the temperature of the liquid? What was the liquid? How much liquid was involved? What was the temperature setting of the water heater? Was the patient wearing clothes? How quickly were the patient's clothes removed? Was the burned area cooled? Who was with the patient when the burn took place? How quickly was care sought? Where did the burn occur? Are the purported circumstances of the injury consistent with the burn characteristics? ***Chemical*** What was the agent? How did the exposure occur? What was the duration of contact? What decontamination occurred? Was there an explosion? ***Electrical*** What kind of electricity was involved? What was the duration of contact? Did the patient fall? What was the estimated voltage? Was there loss of consciousness? Was cardiopulmonary resuscitation administered at the scene?
Determine the severity of the burn	***Extent of burn*** Rule of nines for adolescents Lund-Browder chart for children and infants (Table 6.5) ***Depth of burn***
Adjuncts	
Imaging and laboratory studies	Carboxyhemoglobin
Reevaluation	
Continued assessment of ventilatory effort Continued assessment of extremity perfusion Pain management Psychosocial assessment	
Burn Wound Care	
Thermal burns	Cover the area with a clean, dry sheet Ice applications are appropriate only in small burns
Chemical burns	Flush the chemical agent from the body surface with copious amounts of water Powdered chemicals should be brushed from the skin prior to flushing the involved area

	Remove all contaminated clothing Chemical eye injuries require continuous irrigation until otherwise instructed by an expert
Electrical burns	Continuous cardiac monitoring may be necessary during the first 24 hr after injury Maintain urine output at twice normal target volumes if myoglobinuria
Burn Center Referral	
Partial thickness burns >10% total BSA Burns that involve face, hands, feet, genitalia, perineum, or major joints Full-thickness burns Chemical burns Electrical burns, including lightning injury Inhalation injury Burn injury in patients with significant comorbidities; concomitant trauma; or who require special social, emotional, or rehabilitative intervention Burned children in hospitals without qualified personnel or equipment for care of children	

Note: The principles of Advanced Burn Life Support are identical to the principles of Advanced Trauma Life Support® (Table 7.3), with additional caveats pertinent to Advanced Burn Life Support.

Source: Courtesy American Burn Association. Used with permission.

Note: The principles of Advanced Burn Life Support are identical to the principles of Advanced Trauma Life Support® (Table 7.3), with additional caveats pertinent to Advanced Burn Life Support.

Source: Courtesy American Burn Association. Used with permission.

Chapter 8. Mental Health Issues

Mental Health and the Role of the Pediatrician

Schools and pediatricians have generally become the de facto mental health providers for children. Children are most likely to receive treatment from primary care physicians for symptoms associated with mental disorders, and most psychotropic drug prescriptions for children and adolescents are prescribed by primary care physicians. In a disaster or terrorist event, the need for mental health services will be far greater and the resources even less adequate. Pediatricians and other health professionals that care for children will play many critical roles in identifying and addressing the mental health needs of children and families in a disaster or terrorist event.

For many, if not most, children affected by a critical event, pediatricians and other health care providers for children will be the first responders. Therefore, pediatricians need to be able to identify psychological symptoms, perform timely and effective triage of mental health complaints, initiate brief supportive interventions, and make appropriate referrals when necessary. Many children (and their parents) with emotional reactions to a disaster (manmade or otherwise) will not identify their problems as psychological in nature. Pediatricians will have to be vigilant for somatization and help children, and their families, recognize and address the underlying psychological cause of these physical complaints. Because children's adjustment depends to a great extent on their parents' own ability to cope with the situation, pediatricians should also attempt to identify parents who are having difficulties adjusting to the event and encourage them to seek support for themselves. Pediatricians can also help families identify and access appropriate supportive or counseling services, and they can help support families who are reluctant to seek mental health services because of misunderstandings related to the nature of the treatment or associated stigma.

Trauma-Related Disorders

Children are not immune to the emotional and behavioral consequences of disasters and terrorism. Their reactions depend on their own inherent characteristics and experiences and their developmental level, family and social influences, and the nature and magnitude of the event and their exposure to it.

Exposure to disasters and terrorism can be direct, interpersonal, or indirect. Children who are physically present during an incident are directly exposed. Interpersonal exposure occurs when relatives or close associates are directly affected. Indirect exposure occurs through secondary negative consequences of an event such as chaos and disruption in daily activities. Children who are far away from an incident may be remotely affected with fear and generalized distress as they perceive the societal impact of these experiences.

Exposure to media coverage may play a role in the child's reaction to an event. Studies have documented an association between viewing television coverage of terrorist

incidents and posttraumatic stress reactions, but these associations do not establish a causal relationship. Aroused children may be drawn to the information provided by the media, and it is possible that other factors are responsible for the link between exposure to media coverage and these emotional states.

Reaction to Disasters and Terrorism

Children may develop psychiatric symptoms and disorders—including posttraumatic stress disorder (PTSD), anxiety, depression, and behavioral problems—after exposure to disasters or terrorist incidents. Grief in these situations can be compounded by the traumatic circumstances associated with the loss.

Risk factors for adjustment difficulties. The following factors are associated with an increased risk of posttraumatic symptoms and other adjustment difficulties:

- Children or others close to them are direct victims, especially if injury is involved (or the death of significant others).
- Children directly witness the event, especially if there was exposure to horrific scenes (indirect exposure through the media to these scenes is also associated with increased risk).
- Children perceive during the event that their life is in jeopardy (even if the perception is inaccurate).
- Event results in separation from parents or other caregivers.
- Event results in loss of personal property or other disruption in regular environment.
- Children have a history of prior traumatic experiences.
- Children have a history of prior psychopathology.
- Parents have difficulty coping with the aftermath of the event.
- Family lacks a supportive communication style.
- Community lacks the resources to support children after the event.

Posttraumatic stress disorder. The essential feature of PTSD is the development of characteristic symptoms after exposure to a traumatic event that arouse intense fear, helplessness, or horror or that lead to disorganized or agitated behavior. Signs and symptoms are categorized into three clusters:

1. Persistent intrusive re-experiencing (one symptom required for diagnosis).
    - Unwanted memories, images, thoughts, or perceptions of the experience including repetitive play with themes of the trauma.
    - Bad dreams and nightmares.
    - Acting or feeling like the event is recurring, including flashbacks or re-enactment of the experience.
    - Feeling intense distress when reminded of the experience.
    - Physiological reactivity to reminders.
2. New onset and persistent avoidance and numbing (three symptoms required for diagnosis).
    - Avoiding thoughts, feelings, or conversations associated with the experience.
    - Avoiding activities, places, or people that arouse memories of the experience.

- Amnesia for important parts of the experience.
- Loss of interest or involvement in usual activities.
- Feeling distant and isolated.
- Decreased range of emotions.
- Sense of foreshortened future.

3. New onset persistent hyperarousal (two symptoms required for diagnosis).
   - Sleep difficulties.
   - Irritability and angry outbursts,
   - Difficulty concentrating.
   - Hypervigilance.
   - Exaggerated startle response.

The symptoms must last for more than 1 month and must cause clinically significant distress or impaired functioning. Because of developmental influences, symptoms in young children may not correspond exactly to those in adults.

Other conditions. Other conditions, especially anxiety and affective disorders, are common after crisis events and may occur independently or together with PTSD. These conditions may precede, follow, or develop at the same time as PTSD. Establishing the temporal relationship in onset of disorders may aid in treatment. For example, PTSD stems from the primary traumatic event, while depression may result secondarily from persistent severe PTSD symptoms, intervening stresses, or unresolved grief. Fear and avoidance of situations reminiscent of the trauma may persist for years.

Behavioral reactions. Signs of trauma may be evident in children's behavior, mood, and interactions with others. Traumatized children may adopt behavior more appropriate of younger children. Although they may not share their concerns and they may be especially compliant in the aftermath of an incident, compliant behavior does not mean the child is unaffected. Withdrawal is a cause for concern as it may represent a symptom of PTSD, and it potentially distances the child from adults who could provide support and assistance. Girls are more likely to express anxiety and sadness; boys tend to exhibit more behavior problems.

The child's reaction will reflect his or her developmental level. Infants may experience sleep and feeding problems, irritability, and failure to achieve developmental milestones. Problems in preschool children include separation anxiety, dependence, clinging, irritability, misbehavior, sleep disturbance, and withdrawal. Problems in school-aged children include irritability, somatic complaints, withdrawal, misbehavior, and change in academic performance. Problems in adolescents include anxiety, irritability, isolation and withdrawal, guilt, anger and hate, and preoccupation with death.

Grief and traumatic grief. Although grief is not a mental disorder, it may require professional attention, especially if it is complicated by depression or PTSD. Traumatic deaths are of particular concern in disasters because of the implications for assessment, which should include an evaluation of the circumstances of the death and the child's exposure, and for treatment, which should address trauma symptoms as well as grief. In

some ways, any death may be perceived by survivors as subjectively traumatic; however, five factors have been described that are likely to be present in death circumstances that are considered "traumatic deaths":

- Sudden, unanticipated deaths.
- Deaths involving violence, mutilation, and destruction.
- Deaths that are perceived as random or preventable, or both.
- Multiple deaths.
- Deaths witnessed by the survivor that are associated with a significant threat to personal survival or a massive or shocking confrontation with the death and mutilation.

Deaths that occur in the context of a disaster or terrorist situation often meet these criteria and pose an increased risk of traumatic grief. Referral to a pediatric mental health professional is often indicated in these situations.

Assessment and Treatment

Assessment and treatment of children after a disaster will vary, depending on the characteristics of the disaster and the child's exposure, the setting, and the length of time since the event.

Early interventions. In the acute-impact and early post-impact phases, supportive interventions should ensure the child's safety and protection from additional harm, address immediate physical needs, provide reassurance, minimize exposure to traumatic aspects of the event, validate experiences and feelings, and restore routine. Children benefit from accurate information, but it should be age-appropriate and measured. If possible, pediatric mental health professionals can help other health professionals and family members with the process of death notification. Reuniting family members is a priority.

Assessment and screening. Assessment should include a history of the child's exposure and reactions. When children or their close family members have been directly exposed, the children may require more comprehensive assessment. Children with less direct exposure may also need attention. Children and their parents should be educated about trauma reactions and coping, and they may welcome opportunities to ask questions and correct misperceptions. Children may not spontaneously describe their feelings, and adults may underestimate trauma in children. Therefore, it is essential to ask children directly about their experiences. Observation and the use of projective techniques, such as play and the use of art, aid in assessment and are useful in treatment as well.

Screening to identify children at risk and those needing referral can be conducted with symptom rating scales (such as the UCLA PTSD Reaction Index), which typically measure the type and degree of exposure, subjective reactions, personal consequences, PTSD symptoms, and other related symptoms such as fear and depression, grief, and functioning. Group interviews, conducted in classrooms or other small clusters, may also be used to assess the need for more comprehensive individual evaluation and to begin the process of healing after large-scale or mass casualty events.

Treatment. Treatment should be guided by the child's exposure and reactions. Directly traumatized and bereaved children should be seen individually, but groups are useful for identifying children in need of more comprehensive evaluation and treatment. Cognitive behavioral therapy and educational information provide structure and support and may be used in individual or group sessions after disasters.

Group sessions are ideal for providing age-appropriate explanations of acute and longer term reactions, reactions to traumatic reminders, secondary effects, anniversaries, and coping. Some children are uncomfortable sharing in a group, and group discussions may traumatize the children again through re-exposure to their own experiences or through exposure to the experiences of others. Groups vary with respect to structure and may include play, art, and other projective techniques. Parallel parent groups provide a means to address parental reactions and concerns and to discuss effective management. These groups also provide an opportunity to teach parents how to parent their traumatized child.

The family has a major role in the child's adjustment to trauma, and parents should be included in treatment. Often, more than one family member will be traumatized, although specific aspects of exposure may differ among family members. Helping parents resolve their own emotional distress can increase their perceptiveness and responsiveness to their children. Parents may also benefit from psychoeducation about symptoms, how to manage symptoms effectively, and ways to decrease traumatic reminders and secondary stresses.

Medication is rarely indicated in children after disasters but might be used for those with severe reactions. Consultation with a child psychiatrist is recommended when medication is being considered. When used, medication should be coupled with psychotherapeutic interventions such as play therapy or cognitive behavioral approaches. Specific symptoms determine whether to use a drug, which drug to use, and how long to use it. Comorbid conditions should be considered in selecting an agent. Selective serotonin reuptake inhibitors may be effective in treating childhood PTSD and comorbid anxiety and depression (see also the section on medication, later in this chapter).

School-based interventions. Schools are an excellent setting to deliver mental health services to children and families after a disaster. They provide access to children, encourage normalcy, and minimize stigma. PTSD and associated symptoms are likely to emerge in the school setting. For example, intrusive thoughts and difficulty concentrating may interfere with academic performance and social adaptation. Therefore, school consultation about the consequences of trauma and the recovery process may be indicated. School-based interventions, which can include curricular materials and activities, should be appropriate for the setting and should not supplant efforts to identify and refer children in need of more intensive individual evaluation and treatment.

Long-term and staged interventions. Long-term interventions may be necessary, especially for children with direct or interpersonal exposure and for those with enduring symptoms, pre-existing or comorbid conditions, prior or subsequent trauma, or family

problems. New issues related to trauma may emerge as children mature. Thus, developmentally appropriate staged interventions, which anticipate and address the course of recovery, should be considered during developmental transitions and at marker events such as anniversaries.

Death Notification and Pediatric Bereavement

Considerations in Notifying Individuals About an Unexpected Death

At the time of a disaster or terrorist attack, it is very unlikely that pediatric health care providers will have the time and resources to deliver death notification in an optimal manner. Nonetheless, sensitivity to the issues discussed here can help minimize the short- and long-term impact on survivors.

- Verify the identity of the deceased and identify the next of kin.

- Establish contact as soon as possible. Do not delay contact waiting for a time thought to be more convenient for the survivors (e.g., if the death occurs in the middle of the night, do not wait until the following morning).

- Contact the next of kin. Phone calls can be used to contact next of kin, but death notification is preferably done in person. Alternatively, someone (e.g., police) can be sent to the home of the next of kin to ask them to come to the hospital for notification purposes.

- Minimize the likelihood that you will be compelled to notify the family members of the death over the phone. If you contact the survivor(s) by phone to request they come to the hospital, try to contact the family before the death has been declared (i.e., during resuscitation) or have someone else who has not been directly involved in the care call on your behalf. Someone not directly involved in the care could make a statement such as: “I know that your husband was seriously hurt in the bombing, but I don’t have any further information. If you come to the hospital now, someone who has been taking care of your husband will be available to talk with you when you arrive.” If family members demand information on the phone, the caller can state: “I would prefer to talk with you about this in person when you arrive at the hospital.”

- Consider inviting additional family members or friends to accompany the next of kin to the hospital for notification. If a child has died, it is best to notify both parents at the same time. When any family member has died, survivors may benefit from being told with at least one other family member or friend present. Family members and friends can provide support to the next of kin and help notify other relatives and friends (instead of the entire burden being placed on one survivor).

- Before notifying the family, briefly review the basic facts, including the name of the deceased, the relationship to individual(s) that will be notified, the basic circumstances of injury and death, and the nature of medical care provided. For example, the individual was in a building when a bomb detonated and was found under rubble; CPR was done until arrival in the emergency department, where after an attempt at resuscitation, he was pronounced dead. Identify who else will participate in the notification, and consider planning in advance how to initiate the conversation.

- When the family arrives at the hospital (or site where death notification will be occurring), have them escorted to a private location if possible. Try to inform them in as private a site as possible; if there is no opportunity for a private room, make every reasonable effort to maximize privacy (e.g., use a curtain or notify the family while standing behind the building instead of in front). Do not inform family members in view of the media; anticipate the presence of media and try to offer survivors the opportunity to maintain their privacy as much as possible immediately after notification.

- If possible, have the notification conducted by a physician who was involved in the care, especially if he or she knows the family or had some direct involvement. Comments such as "I was with your husband when he first arrived at the hospital. He was not conscious at the time and therefore was not feeling any pain" can be very helpful to families. Inform the patient's primary care provider whenever possible.

- Consider involving at least one other professional on the health care team, such as a social worker, chaplain, nurse, etc. If more than one family member is receiving the notification, conducting the notification with another professional is especially helpful; however, one staff person should be in charge of the discussion. Try to include at least two staff people for notification, even when notification is conducted in the field, but limit the number of staff to those directly involved. It can be overwhelming for a family member to be notified by a large team.

- Introduce yourself and any other member(s) of the health care team who are participating by name and title and offer to shake hands.

- Offer seating to the survivors. Sit close to them and face them so that eye-to-eye contact can be easily maintained.

- Refer to the deceased by name and/or relationship to the survivor (e.g., "Mr. Smith" or "your husband"). Avoid referring to the person as "the deceased" or "the victim."

- If children are included, involve professionals with training and experience in working with children in the notification process. Notification of the death of a family member is preferably provided to children by family members (such as the

surviving parent), rather than by professionals unknown to the child. However, parents may wish for professionals to be present when children are told to provide support and to help answer questions.

- Remember that informing survivors of a death is a process, not an act. Pacing of the discussion is important. Do not start by stating that the individual is dead because survivors are unlikely to hear any further information.

- Start by asking the family what they have already been told or know. Then provide a brief description of the circumstances of the injury and the relief efforts. This information helps the survivors understand the context of the death; not knowing what happened introduces a discontinuity in the history that impairs adjustment. After giving brief background information, it is useful to give a "warning notice" and then proceed fairly quickly to stating that the individual died. Ideally, the family will be present during the resuscitation efforts, and medical staff can provide the background information when the resuscitation begins and return to deliver updates that may serve as a "warning notice." For example, "The team has given several medications to try to get your husband's heart starting again, but so far there has not been any response."

- An example of a notification initiated after a death might be: "There was an explosion 2 hours ago that we believe was caused by a bomb in the building where your husband works. The explosion started a fire that spread rapidly. Firefighters arrived on the scene within several minutes, but the exits were blocked and flames spread quickly. Many individuals were unable to get out of the building before they were overwhelmed by smoke. I am sorry to say that your husband did not get out of the building in time. We believe he died as a result of the smoke from the fire. His body was recovered by a firefighter, and we identified him by the wallet that we found in his pocket. We found your phone number in the wallet. I am very sorry to have to be telling you this news."

- After notifying the survivor(s) of the death, pause to allow both the information to be processed and emotions to be expressed. Do not try to fill the silence, even though it may seem awkward. Listen more than you speak. Silence is often better than anything you can say. Stay with the family members as they are reacting to the news, even if they are not talking.

- Use clear and simple language. Avoid euphemisms such as terminated, expired, or passed away. State that the individual died or is dead.

- Don't provide unnecessary graphic details. Begin by providing basic information and allow the individual to ask questions for more details.

- Don't lie or speculate. If you do not know the answer to a question, say so. Try to get the answer if possible.

- Be conscious of nonverbal communication and cues, both those of the family as well as your own.

- Be aware of and sensitive to cultural differences. If you do not know how a particular culture deals with a death, it is fine to ask the family. Be particularly attentive to difficulty speaking or understanding English. If there is any doubt whether the family members are fluent in English, make sure to have a professional translator present unless you are fluent in the family's preferred language. Using family and friends as unofficial translators often leads to inadequate translation in the general medical setting. Such reliance on family and friends as translators for death notification is particularly burdensome to them and should be avoided.

- Consider the use of limited physical contact (e.g., placing a hand on the family member's shoulder or providing a shoulder to cry on). Monitor the individual's body language and if at all in doubt whether such contact would be well received, ask first.

- Realize that the individual may initially appear to be in shock or denial. Expect additional reactions, such as sadness, anger, guilt, or blame. Acknowledge emotions and allow them to be expressed without judgment.

- Do not ignore or dismiss suicidal or homicidal statements or threats. Investigate any such statements (often this will be facilitated by the involvement of mental health professionals) and if concerns persist, take appropriate action.

- Just before and during the notification process, try to assess if the survivors have any physical (e.g., severe heart disease) or psychological (e.g., major depression) risk factors, and assess their status after notification has been completed.

- If possible, write down your name and contact information in case the family wants further information at a later time. If the situation is not appropriate for providing your name and contact information, then consider how the family may be able to obtain additional information in the future (even months later). For example: "I work as a volunteer for the Red Cross. Here is my name and the contact information for the Red Cross Chapter. If later you wish more information about what happened to your husband, you can call them at this number and they should be able to look at the records." Survivors may not be ready to think of or ask questions and may later regret not asking for critical information.

- Do not try to "cheer-up" survivors by making statements such as "I know it hurts very much right now, but I know you will feel better within a short period of time." Instead, allow them their grief. Do not encourage them to be strong or to cover up their emotions by saying "You need to be strong for your children; you don't want them to see you crying, do you?"

- Feel free to express your own feelings and to demonstrate empathy, but do not state you know exactly how family members feel. Comments such as "I realize this must be extremely difficult for you" or "I can only begin to imagine how painful this must be to hear" can demonstrate empathy. Avoid statements such as "I know exactly what you are going through" (you can't know this) or "You must be angry" (let the individual express his or her own feelings; don't tell the person how to feel) or "Both my parents died when I was your age" (don't compete with the survivor for sympathy). Provide whatever reassuring information you may be able to, such as "It appears your husband died immediately after the explosion. It is unlikely he was even aware of what happened and did not suffer before he died." However, do not use such information as an attempt to cheer up family members (e.g., "You should be happy, many people suffered painful burns or were trapped under rubble for an hour before they died. At least your husband didn't experience that.")

- Feel free to demonstrate that you are upset as well—it is fine to cry or become tearful. If you feel, though, that you are likely to become overwhelmed (e.g., sobbing or hysterical), then try to identify someone else to do the notification.

- After you have provided the information to the family and allowed adequate time for them to process the information, you may wish to ask questions to verify comprehension.

- Offer the family the opportunity to view the body of the deceased and to spend some time with their loved one. Before allowing the family to view the body, the health care team should prepare it for viewing by others. A member of the health care team should escort the family to the viewing and remain present, at least initially.[1]

- Help families figure out what to do next. Offer to help them notify additional family members or close friends. Tell them what needs to be done regarding the disposition of the body. Check to see if they have a means to get home safely (if they have driven to the notification, they may not feel able to drive back safely), and inquire if they have someone they can be with when they return home.

- Help survivors identify potential sources of support within the community (e.g., member of the clergy, their pediatrician, family members, or close friends).

- Take care of yourself. Death notification can be very stressful to health care providers. Health care providers need to explore and come to understand their own reactions to patient death and associated emotions, which may include

[1] For further information about preparation of the body for viewing, as well as additional recommendations about the death notification process, *see* Leash RM. *Death Notification: A Practical Guide to the Process*. Hinesburg, VT: Upper Access, 1994.

sadness, anger, guilt, or a sense of responsibility. It is important to provide informal support and debriefing to professionals who provide death notification, especially if related to tragic deaths or when multiple deaths are involved (as would be anticipated in a major disaster or terrorist event).

Explaining Death to Children

Children's understanding of death may be very different from that of adults. Children have had far less personal experience of loss and have accumulated less information about death. They can also have difficulty understanding what they have seen and what they are told unless the basic concepts related to death are explained to them. Adults will need to provide especially young children with both the basic facts about what happens to people after they die, as well as the concepts that help them to explain those facts. For example, young children may be told that after people have died, their body is buried in a cemetery or turned to ashes that can then be buried or scattered. Children can be very distressed by these facts unless they are helped to understand the concept that at the time of death, all life functions end completely and permanently—the body can no longer move, and the person is no longer able to feel pain. That is why it is okay to bury or cremate the body.

Children need to understand four concepts about death to comprehend what death means and to adjust to a personal loss: irreversibility, finality, inevitability, and causality (Table 8.1). Most children will develop an understanding of these concepts between ages 5 and 7, but this varies widely among children of the same age or developmental level, based in part on their experience and what others have taught them. When faced with a personal loss, some children 2 years old or younger may demonstrate at least some comprehension of these concepts. Adults should not underestimate the ability of young children to understand what death means if it is explained to them properly. Therefore, it is best to ask children what they understand about death, instead of assuming a level of comprehension based on their age. As children explain what they already understand, it will be possible to identify their misunderstandings and misinformation and to correct them accordingly.

When providing explanations to children, use simple and direct terms. Be sure to use the words "dead" or "died" instead of euphemisms that children may find confusing. If young children are told that the person who died is in "eternal sleep," they may expect the deceased to later awaken and be afraid to go to sleep themselves. This description does little to help children understand death and may cause more confusion and distress. Religious explanations can be shared with children of any age, but adults should appreciate that religious explanations are generally very abstract and therefore difficult for young children to comprehend. It is best to present both the facts about what happens to the physical body after death, as well as the religious beliefs that are held by the family.

Even when children are given appropriate explanations, they still may misinterpret what they have been told. For example, some children who have been told that the body is placed in a casket worry about where the head has been placed. After explanations have

been given to children, it is helpful to ask them to review what they now understand about the death.

Common Reactions Among Children Who Have Experienced a Personal Loss

Like adults, children may be reluctant to talk about a death. They may at first be shocked by the news or fail to understand its implications. Young children have difficulty sustaining strong emotions, so they may appear upset for a brief period of time and then return to play. They may also use play or other creative activities, such as artwork or writing, to both express and work through their feelings associated with a loss. By observing play and the products of children's creative activities, we may find some clues as to what is bothering them, but it is important not to jump to conclusions about the meaning or relevance of what is observed.

Soon after notification, children often ask questions about the deceased and the meaning it has for them personally. These questions may cause surviving family members distress because they often are particularly poignant. Children pick up readily on the cues from others in their family that adults are made uncomfortable by these inquiries. They may conclude that such questions are unwelcome, inappropriate, or even represent misbehavior and stop asking. The silence that results is not an indication that children do not understand what has happened or have already coped. Rather, it may be a sign that they are trying to protect their parents who appear overwhelmed or that they do not feel comfortable asking questions or expressing their emotions, leaving them to deal with both alone. Therefore, it is important for adults to explicitly invite children to share their questions and feelings. Often it is helpful for an adult who is familiar with child development and who knows the child personally to provide an additional outlet for discussion. The child's pediatrician or a social worker from the child's school, for example, may be in a good position to start such a conversation.

Older children and adolescents may initially decline the assistance of adults because they are more accustomed to turning to peers for support and to address issues of concern. It is important to extend an open invitation to these young people to talk with you when they have questions or want to talk about the situation and to help them identify other adults in their lives they can turn to for support and assistance (e.g., a chaplain, coach, or teacher).

Even in the setting of a natural disaster or terrorist event, children may still wonder if they were in some way responsible for the death. After a traumatic death, such guilt feelings may increase posttraumatic symptoms and complicate the grieving process. Young children in particular have a very limited understanding of why things occur and tend to be self-centered. As a result, they often use magical thinking to explain situations that they do not understand. This may result in extreme feelings of personal responsibility for a death that has occurred, even in situations when there is absolutely no logical reason why the child should feel responsible.

Often such feelings of guilt are irrational. "If only I hadn't gone to school that day, my dad would never have gone to the office and wouldn't have been killed by the bomb," "I

was mean to my father yesterday and that's probably why he died," etc. Understandably, children are often reluctant to share their guilt feelings with adults; adults may not anticipate these feelings (or be burdened with their own guilt feelings). It may be helpful to reassure children of their complete lack of responsibility, even if they do not express feelings of guilt, and there is no logical reason why you might anticipate they would feel guilty.

At the time of a traumatic loss, children often think first about their own needs. Parents should be warned that this self-centeredness is not a sign that children are selfish; more likely, it is a sign that they are under considerable stress and in need of more support and assistance.

Children often regress in response to the stress of a personal loss. Children who had been successfully toilet-trained may now begin to wet their bed; children who had not had difficulty attending daycare may now begin to show separation problems; children who had good social skills may now argue more or have difficulty getting along with peers. Children and adolescents may also develop somatic complaints, such as headaches, stomach aches, or generalized fatigue.

A disaster or terrorist event may uncover children's concerns about another loss or personal crisis that has not been fully resolved. Children may react strongly to the death of someone that they did not know well or perhaps did not know at all. Or, children may be more preoccupied with their own personal crises than they are affected by the death of someone in their family or community.

Indications of the Need for Referral

Not every child who has experienced the death of a family member or friend requires professional counseling, and in the setting of a major disaster or terrorist event, such resources are unlikely to be available. It generally is helpful though for children who have experienced the death of a family member or friend to speak with someone outside of the immediate family who understands child development and can attend to the child's needs (without being burdened with his or her own grief), such as their pediatrician or a school counselor or social worker. When a community disaster or crisis has occurred, it is important to help establish access to supportive services within community sites, such as schools, to provide services to larger numbers of children.

Significant stigma continues to be associated with receiving mental health services, and this stigma remains even in the setting of a major crisis event. Parents and other caregivers need to understand that even though bereavement is a normative experience, it still can be profoundly difficult. People, including children, can be helped through supportive services and, when indicated, group or individual counseling.

Children who have extreme reactions (e.g., anxiety, posttraumatic symptoms, depression, or thoughts of suicide), atypical reactions (e.g., appearing happy or disinterested), or prolonged reactions (e.g., prolonged sleep problems or somatization) should be evaluated by their pediatrician and likely referred to a mental health professional experienced in the

management of pediatric bereavement. Children should be referred who are having difficulty returning to their normal daily routines several weeks after the death or are demonstrating the new onset or worsening of problems interacting with peers. Children who are experiencing traumatic grief may require treatment of posttraumatic symptomatology before they are able to continue with normal grieving.

Soon after a death has occurred, many children may find comfort in returning to school, spending time with their friends, and taking part in the same activities that they did before the death. Allowances and adjustments should be made for a time (such as extra help with homework because of difficulty concentrating and learning) so that they can return to their day-to-day life as soon as possible. Some children may resist returning to school or resuming their regular daily activities. They may be fearful to leave other family members, worrying that they may die in their absence or that grieving family members may need their support. These children require reassurance of the safety and well-being of surviving family members and encouragement to return to school. Other grieving family members, especially parents or guardians, should receive the support and assistance they need, so that the children do not feel it is their responsibility.

Attendance of Children at Funerals and Memorial Services

Children can be told in simple terms what to expect at a funeral or memorial service. If an open casket or gravesite ceremony is planned, children should be told and given explanations about what this involves. Children can be invited to participate to their level of comfort but should not be forced or coerced to attend. They should be encouraged to ask questions, which should be answered simply and honestly but without unnecessary details. At the ceremony, children should be accompanied by an adult they know and like (who is not personally grieving to the same extent as close family members) who can monitor the child's reactions, answer questions, and step out of the ceremony with the child if the child appears distressed or indicates a desire to leave. Even if children play quietly in the lobby of a funeral home, they may still have a sense of having participated in the ritual. Children who are not allowed to attend the funeral or memorial service often feel angry and hurt and lose out on the benefits of religious, family, and community support. They also may create fantasies about what occurs during funerals that are actually more frightening than the reality. It is also helpful if children can perform a small task at the funeral, such as handing out Mass cards at the entrance of the funeral home or selecting flowers to be placed near the coffin. Such tasks should be predominantly symbolic, of the child's choosing, and not overwhelming for the child.

Therapies for Psychic Trauma

Crisis Response

Mass violence presents unique issues that differ from other episodes of interpersonal, community, and other forms of violence. Responding to those individuals who are directly affected by the event is not enough—a multilevel strategy is required and should include victims and witnesses, individuals with whom they are associated, and the broader community. Although crisis response providers do not have to perform all of

these roles, they should work closely and collaborate with a number of individuals and agencies to ensure that the psychological impact of mass violence is addressed.

The first and foremost response to mass events is both directed and performed by the government and its agents. These are usually under the auspices of law enforcement, fire personnel, and/or emergency medical services, which are typically managed by an Incident Command System (ICS). Mental health early responders should have pre-existing relationships with the ICS to perform their duties effectively. In most States and other jurisdictions, ICS staff members meet regularly to ensure efficient operation when needed. During episodes of mass violence, mental health providers need to be part of the ICS staff whenever possible. The pre-existing relationship with emergency response commanders permits more expeditious access to affected individuals and for the community's psychological needs to be considered consonant with emergency responses.

In addition, when mental health providers are members of the ICS, access to and allocation of resources for mental health crisis responders in situations of mass violence improves. Situating providers in the most useful locations, ensuring the flow of needed information and communications, and preventing well-intended, but inexperienced and unlinked clinicians from arriving en masse in an attempt to provide services are essential to lessen the general confusion and chaos that accompany disasters.

A useful way of defining and understanding a traumatic response is that the affected individual experiences the loss of both internal and external control. Therefore, maximizing organization and structure is a necessary prerequisite in providing mental health crisis response and early intervention. Mental health crisis models are best equipped to achieve this organization and structure when they are firmly rooted in the ICS.

Crisis Response for Children and Families

Unfortunately, there is no clear empirical evidence for the effectiveness of any crisis response intervention. In fact, the frequently used and previously heralded Critical Incident Stress Debriefing or Management (CISD or CISM) strategies have not demonstrated effectiveness, and in some studies they have proved detrimental. Indeed, it has been recommended that compulsory debriefing of victims of trauma should cease. However, it is possible that an alternative method of early crisis intervention may be helpful for assisting people who may be recently traumatized. The following recommendations and guidelines for early intervention strategies are based on evidence from research on the risk factors for PTSD as well as some intervention research. Thus, they provide an empirical foundation for appropriate and useful approaches to assist potentially traumatized individuals.

Currently, there is no evidence that global intervention for all trauma survivors serves a function in preventing subsequent psychopathology. However, there is consensus that providing comfort, information, and support and meeting the immediate practical and emotional needs of affected individuals can help people cope with a highly stressful event. This intervention should be conceptualized as supportive and noninterventional but

definitely not as a therapy or treatment. This suggestion recognizes that most people do not develop PTSD and other posttraumatic symptoms immediately. Instead, they usually will experience transient stress reactions that will abate with time. The goal of early intervention is to create a supportive (but not intrusive) relationship that will result in the exposed individual being open to followup, further assessment, and referral to treatment when necessary. Inherent in this early intervention is the recognition that interpretation or directive interventions are not to be provided.

After assuring that basic necessities are available and are not a pressing concern, the basic principles of intervention should be followed. These principles should ensure that no harm is being done in the intervention process and hopefully prevent or reduce symptomatology and impairment.

- Interventions should be grounded in the basic principles of child development, and providers should be experienced in working with children of different ages and levels of development.
- Mental health providers should have collaborative relationships with community providers to ensure access and community support for children and families.
- Children and families should be assessed for risk factors and symptoms, and interventions should be crafted to address the findings.
- An essential objective is to improve parental attention and family cohesion through assessment, psychoeducation, and treatment, when necessary, to parents and primary caregivers.
- Providers should make concerted efforts to prevent social disruption and displacement.
- Providers should identify, assess, and attempt to ameliorate or remove children and families from the continued threat of danger.
- Providers should have continued contact and monitor children for symptoms or impairment.

Handouts or flyers that describe trauma, what to expect, and where to get help should be made available. Individuals should be given an array of intervention options that may best meet their needs. The goal is not to maximize emotional processing of horrific events, as in exposure therapy, but rather to respond to the acute need that arises in many to share their experience, while at the same time respecting those who do not wish to discuss what happened.

Medication

There is not yet clear evidence to support the use of pharmacotherapy in the treatment of posttraumatic symptoms in children, and no randomized controlled trial has been completed. The first line of treatment for posttraumatic symptoms in children is trauma-focused cognitive behavioral treatments (CBT), which includes such interventions as graded desensitization and other CBT.

So, why consider the use of psychopharmacology for children with posttraumatic symptoms?

- Lack of availability of trauma-focused psychotherapy (CBT). In many areas of the country, these therapies are not available for children and although nondirective therapies can be helpful for children with posttraumatic symptoms, they do not appear to directly treat the symptoms.
- Some children do not respond to trauma-focused psychotherapies. As in all situations, first-line treatments do not always work, and other interventions are required.
- Randomized controlled trials have demonstrated the efficacy of medications in adults with posttraumatic symptoms. Although the analogy between medication effectiveness in adults and children is frequently made, extreme care is indicated. Metabolic and neurodevelopmental factors have great influence on dosage and frequency of medications and responses in children. Some medications that are effective in adults are not effective in children, and furthermore, they may have different or more dangerous side effects.

Considering the use of medications for children with early posttraumatic symptoms prompts certain considerations:

- Is a trauma-focused therapy available, and if so, has the child completed a trial without responding?
- What specific symptoms are being targeted?
- Different psychotropic medications are better for different symptoms (see below).
- What is the child's level of distress (symptomatic and functional)? In nearly 60% of cases, early posttraumatic symptoms are transient and resolve without treatment. So the question is whether the current amount of distress and functional impairment warrants a medication trial.
- Are other family members symptomatic?
- Ascertaining the level of untreated symptomatology of other family members, especially parents, is essential, because posttraumatic symptoms—especially anxiety and hypervigilance—are highly transferable to the child. So, treating the child may have no impact on symptoms if they originated with the parent.
- What is the developmental or neuromaturational level of the child?

Again, psychotropics are much more difficult to use in younger children and may have an increased propensity to cause side effects.

Neurotransmitters implicated in PTSD symptoms include the following:

- Adrenergics (norepinephrine, epinephrine).
- Dopamine.
- Serotonin (5-HT).
- Endogenous opioids.

Symptoms that appear to be directly related to dysregulation in the adrenergic system include increased heart rate, blood pressure, and anxiety, as well as hyperarousal symptoms such as nightmares, poor sleep, hypervigilance, and panic attacks.

Blocking norepinephrine and epinephrine may treat these symptoms. The first choices, especially for sleep difficulties, are the α_2-blockers clonidine and guafacine. These agents are especially good at improving sleep and decreasing nightmares acutely and may improve other hyperarousal symptoms such as impulsivity and hypervigilance.

Symptoms associated with dysregulation of the dopaminergic system include anxiety, hypervigilance, aggressive impulsivity, flashbacks, and paranoia. Studies of risperidone indicate that it may be useful for this constellation of symptoms.

There is clear evidence of dysregulation of the serotonergic system in individuals with posttraumatic symptoms. Deficit of 5-HT is associated with depression, thoughts of suicide, aggression, impulsivity, anxiety, and obsessive thoughts. This suggests that increasing 5-HT availability may treat these symptoms, and studies in adults have found selective serotonin reuptake inhibitors (SSRIs) to be the only class of drugs to decrease all three PTSD symptom clusters. However, with acute posttraumatic symptoms, 6–8 weeks may be needed for effect, so it is better used as a first-line therapy for children who meet the criteria for PTSD. At present, the SSRIs of choice are fluoxetine and citalopram.

Although benzodiazepines would seem an obvious choice for acute posttraumatic symptoms, their use is controversial. In two randomized controlled trials of benzodiazepine use for this purpose, one demonstrated moderate positive effects, while the other showed increased likelihood of the individual developing PTSD.

School Crisis Response

Most children benefit from receiving supportive services in the aftermath of a disaster or terrorist attack. Pediatricians can play a vital role in advocating for, consulting for, and actively participating in school crisis response teams to ensure that such supportive services can be provided to children within schools and other community sites.

School administrators, teachers, and other school staff will be affected by the same crisis event that is affecting their students. During such times, organizing and implementing an effective crisis response can be difficult or even impossible. Therefore, it is imperative that schools begin planning for potential crisis events before they occur, both to avert disasters whenever possible and to decrease the negative impact on students and staff when disasters cannot be prevented.

The school crisis response plan should include generic protocols for the following:

- Notification of team members, school staff, students, and parents of a crisis event.
- Delivery of psychoeducational services and brief crisis-oriented counseling, such as through support rooms or short-term support groups.
- Memorialization and commemoration (see guidelines at http://www.csee.net/pageview.aspx?id=55).
- Followup.

The structure provided by a preexisting plan can be very comforting in times of crisis and helps to ensure that key issues are considered, appropriate steps are taken, and necessary resources are in place.

In addition, the crisis response plan should include guidelines on the following:

- Crisis team membership.
- Roles of crisis team members.
- Protocols for delivery of crisis intervention services.
- Specific guidelines for responding to unique situations, such as large-scale natural disasters or a terrorist attack.
- Physical safety and security.
- Rapid dissemination of accurate and appropriate information.
- Attention to the emotional impact of the events and the crisis response.

All areas should be addressed concurrently and in a coordinated fashion.

Delivery of supportive services to children during a crisis can be demanding work for school staff and community mental health providers working within the schools. Plans should also include mechanisms to ensure that supportive services for staff are included as a key component of a crisis response.

Anniversary Reactions and Commemorative Activities

As the anniversaries of stressful, critical, or traumatic events approach, many children and adults still have significant reactions. Throughout the year, reminders of the original crisis may add to children's sense of further danger and emotional distress. Those reminders of the events may also increase the reactions of peers, parents, teachers, and other adults.

Remember:

- Memorial activities can further the process of healing and learning.
- The planning process is as important as the memorial activities themselves.
- Doctors, teachers, parents, and children all benefit from the planning process.
- Symptoms and reactions vary from child to child.
- There is no one "best way" to acknowledge an anniversary.
- Helping children deal with a difficult event is hard work; pediatricians need to take care of themselves and their staff as well.

Anniversaries

At the time of the anniversary, children frequently experience a recurrence of some of the feelings associated with a loss or tragedy. These reactions vary widely, and they can be seen in both children and adults. Some children may not be interested in revisiting the events. For these children, it may be more appropriate that they are occupied with the typical concerns of childhood.

It is important to find ways within the school to recognize the anniversary of such an important event without imposing personal emotions or expectations on either students or staff.

Some children directly affected by the traumatic event may appear to be "back to normal" but may still be feeling sad, scared, anxious, or angry. Children do not always demonstrate their feelings directly, and we should pay special attention to signs of concern or distress. Children who are known to have histories or ongoing exposure to trauma or loss, even if they are not directly related to the traumatic event, may be especially vulnerable in the days and weeks surrounding the anniversary.

Heightened media coverage and publicity of memorial events may increase reactions in children. Parents should monitor and supervise their watching of television and, especially for younger children, consider limiting the amount of television exposure.

Some signs of distress to look for include the sudden appearance of or noticeable change in the following:

- Depressed or irritable mood.
- Oppositional and defiant attitude.
- Attention-getting or other behavioral problems.
- Difficulties getting along with classmates and peer group.
- Social isolation or withdrawal.
- Deterioration in academic performance.
- Physical complaints.
- Changes in appetite.
- Sleep disturbances.

The extent and nature of potential difficulties may be related to many factors, including the following:

- Age and developmental level.
- Personal history (e.g., prior trauma, loss, or emotional difficulties).
- Support from peers, parents, and school staff.

Memorialization

Memorialization is any activity designed to formally mark the anniversary or memory of a significant event. Memorial events can help children express and cope with their feelings that might otherwise seem overwhelming to deal with alone. By actively planning and participating in a memorial event, children can exercise some control over how they will remember the disturbing event.

Children may have needs similar to those of adults in times of crisis, but they often meet those needs in very different ways. It is important to find out from the children what they would like to remember and what they think would be the best way to acknowledge the anniversary. Children need to be part of the planning process for memorial events. A memorial planned by adults for children is likely to be more helpful to the adults and not

necessarily meet the children's needs. The planning of a memorial activity can be more therapeutic than participating in the activity itself.

Remember also that different groups of children and adults will have different needs and wishes at the time of the anniversary. Memorial activities do not need to be formal or elaborate. It is best to take cues from children, considering their age and developmental level, when planning memorial activities. Discussion allows children to explore how they are feeling and to think about what might help them feel better.

Some children may wish to acknowledge the anniversary in a personally meaningful way (e.g., drawing a picture, writing a poem or essay) but resist a group activity centered around the anniversary. Some children may prefer not to mark the anniversary with any formal or even informal activity. It is important to remember that those children who are grieving their own personal losses may resent or feel frustrated if the memorial event focuses only on the heroic efforts of rescue workers.

Planning a Memorial Activity

Memorial activities can be planned at various levels, including individual consultation with the pediatrician, with family members, in small student groups, or in larger community or school-wide committees. Children should be involved in the planning process, but it is equally important for adults to provide guidance, structure, and support.

- Consider the children's ages and developmental levels when planning activities.
- Some children may wish to involve other friends or family members in the planning process.
- Coordinate the planned events with the family and the school.
- Not all children will want to be involved in the planning process, and participation should be voluntary.
- Don't feel pressured to plan the "perfect event." Any memorial event or activity, big or small, may be a helpful means for children to understand and mark an anniversary.
- Activities within a school or individual classroom may affect other students and staff within the school as well as children's families at home. Therefore, other families should be informed about plans for memorial events within a school.
- Other adults will benefit from additional support and guidance on how to mark an anniversary in a sensitive manner.
- Awareness of school activities and plans often can help to initiate discussions at home, where children may be most comfortable talking about critical events and anniversaries.
- Parents should be invited to share any concerns related to the anniversary or relevant family experiences with the pediatrician, teachers, and school staff. Pediatricians, teachers, and school personnel should keep the lines of communication open with parents throughout the planning process. Parents should be encouraged to continue to discuss the planned activities with their children at home.
- Open discussion communicates to children that adults are available for further discussion and support.

- Look for signs of distress in students, such as agitation, acting out, or other unexpected behaviors, and help teachers, parents, and school personnel to be aware of them.

In some instances, families may not wish to have their children participate in memorial activities. Remember that many children and their families choose not to disclose personal losses, and their privacy should be respected.

Supporting School Staff

Some adults may find it difficult to discuss traumatic events, especially if dealing with their own losses. Adults should seek out support from other adults and colleagues when needed. This is difficult work for everyone, and it is important for staff to think about what their own feelings are in relation to the events. Providing an opportunity for staff to talk about their own reactions may be useful to them personally and may better prepare them to meet the children's needs.

Remember that children look to adults for guidance and support during difficult times. We need to think about how our own reactions may impact children. Children's questions may sometimes take us off guard and make us confront issues we would rather not think about.

Having a plan to address these concerns in advance will help make the task easier. If the task seems too difficult, staff should share the responsibility with a colleague or invite someone else to help with the planning and process of memorialization.

Impact on Health Care Providers

Very often, first responders and other adult service providers show signs of stress and emotional disruption after responding to situations involving disasters, including terrorism, that result in trauma. Typically, programs such as Critical Incident Stress Debriefing or Management (CISD or CISM) are effectively used with first responders. Fire departments and emergency medical teams have incorporated these strategies into the response programs to critical incidents. A short but specialized training is needed for an individual to apply this model, which basically walks the responder through a guided exploration of their experience in responding to a critical situation.

Pediatricians may be approached by a colleague, a fellow worker, or any health care provider in distress. The following basic, common sense rules can help:

- Make psychological contact.
- Assess the individual's level of coping.
- Explore possibilities for getting further help.
- Assist in taking action.
- Followup.

Make Psychological Contact

Tune in—empathetic listening is a precondition for any helping activity:

- Invite the person to talk.
- Listen for what happened (facts).
- Listen for the person's reaction to the events (feelings).
- Maintain a calm, controlled manner.

Level of Coping

Examine the dimensions of the problem. Ask questions that focus on three areas:

- Immediate situation. What were the events leading up to the current difficulties in coping?
- Present. What life situations may increase or decrease the level of stress (family, work, children, etc.)? Have the person tell the story.
- Immediate future. What are the likely future difficulties? What coping strategies have been attempted?

Explore Possible Solutions

Take a step-by-step approach; ask first about what has already been tried, then try to get the individual to generate alternatives, and finally, add other possible solutions. Some people may need coaching to even consider the idea that a possible solution exists. Examine obstacles to implementation and address these issues before a plan is implemented. Part of the solution may include helping the individual to establish a supportive social network. Finally, it may be appropriate to suggest that the individual seek professional help for emotional difficulties.

Take Action

The objective is very limited, i.e., no more than taking the best next step given the situation. Help the individual implement the agreed upon immediate solutions(s) aimed at dealing with the immediate need(s).

Followup

The last component involves establishing a way to provide the support the individual needs to ensure that the necessary steps are taken toward a satisfactory resolution of the situation.

Basic Objectives

- Help caregiving adults recognize the immediate emotional, cognitive, and behavioral impact of overwhelming events on themselves, typically through psychoeducational approaches.

- Engage the responder or care provider for purposes of followup assessments and identification of service needs across multiple domains, including home, clinical, work, and community settings that involve individuals, families, and groups.

Benefits

One of the primary ways to reduce stress for first responders and other service providers is through "psychoeducational programs," especially training that emphasizes skills in the following:

- Coping with people. Stress reduction also occurs through increased confidence and abilities to have an impact on and help other people rather than just oneself (e.g., microcounseling, "psychological first aid," human relations training, assertiveness training).
- Interpersonal awareness. Stress reduction also occurs through increased understanding and awareness of your own and other people's cognitive and emotional reactions to traumatic events.

These programs significantly decrease the following:

- Depression.
- Anxiety.
- Psychological strain.
- Physical strain.
- Emotional exhaustion.
- Vocational strain.
- Interpersonal strain.

Additionally, these programs decrease anger and improve relationship adjustment.

Risk Communication and Media Issues

Information should be communicated to the public in timely, accurate ways that do not heighten concern and fear. Communicating effectively during a crisis requires the following:

- Planning.
- Preparation.
- An understanding of communications protocols, messaging, and the media.
- The ability to manage the flow of information.

Each element is a challenge that can be met effectively, to the benefit of those receiving messages in times of crisis.

Developing Goals and Key Messages

People often fail to communicate effectively due to a lack of clear communications goals and key messages to support them. Setting such goals and identifying support messages are tasks that should be accomplished before issuing any public comment and are especially important in a crisis.

A communications goal of "educating the public on the complexities of bioterrorism and preparing them for any eventuality" is not realistic. Informing the public of the problem and specific dangers, providing guidance on appropriate responses, and easing concerns are achievable goals. Messages in support of these goals should also be direct and speak effectively to the audience.

A risk message is a written, verbal, or visual statement containing information about risk that may or may not include advice about behaviors to reduce risk. A formal risk message is a structured written, audio, or visual package developed with the express purpose of presenting information about risk. Risk messages may aim to ease public concern or provide guidance on how to respond.

Messages to ease public concern. Examples of messages to ease public concern are:

- The risk is low.
- The illness is treatable.
- It is not easily contracted.
- Symptoms are easily recognized.

Messages on how to respond. Examples of messages that give guidance on how to respond include:

- Take these precautions.
- If possibly exposed, contact a physician.
- If symptomatic, contact a physician.
- Note possible symptoms in others.

If the goal is to ease concern and the message in support of that goal is "the risk to the public is low," that message should be clearly stated at the outset and returned to as often as possible.

- Raise points often enough that the audience leaves with a clear understanding of the message you wanted them to hear.
- Take opportunities to begin or end statements with a reiteration of your message.
- Don't be so repetitious with a single message that you appear to be trying to convince people of something that isn't true.
- Don't repeat messages word-for-word every time you answer a question.

Exercise some control over the conversation you are having, be it an interview, press conference, or questions from an audience. Don't allow the conversation to be led down paths that are not pertinent to the goals or message—no matter how persistent the questioner might be in pursuing a line of inquiry.

Delivering Accurate and Timely Information

In a risk-communication situation, there is constant tension between providing accurate information and providing information quickly. Both demands pose challenges. To wait for all information to be complete and verified before releasing it to the public can create

an information vacuum that will almost certainly be filled with rumor and speculation. To release information that has not been confirmed and turns out to be inaccurate, however, runs the risk of misleading the public and undermining your credibility as a spokesperson.

- Goals and messages should be simple, straightforward, and realistic.
- Information should be delivered with brevity, clarity, and effectiveness.

Most importantly, always provide statistics and key information to the media in written form. In presenting information, always know how the information was gathered and how any conclusions were reached.[2]

Bibliography

Trauma-Related Disorders

- American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders-IV-TR* (4th ed). Washington, DC: American Psychiatric Association; 2000.
- Bolton D, O'Ryan D, Udwin, et al. The long-term psychological effects of a disaster experienced in adolescence: II: General psychopathology. *J Child Psychol Psychiatry.* 2000;41(4):513–523.
- Cohen JA, Bernet W, Dunne JE, et al. Practice parameters for the assessment and treatment of children and adolescents with posttraumatic stress disorder. *J Am Acad Child Adolesc Psychiatry.* 1998;37(Suppl 10):4S–26S.
- Eth S, Pynoos RS. Developmental perspective on psychic trauma in childhood. In Figley CR (ed), *Trauma and Its Wake*. New York: Brunner/Mazel;1985:36–52.
- Goenjian AK, Pynoos RS, Steinberg AM, et al. Psychiatric comorbidity in children after the 1988 earthquake in Armenia. *J Am Acad Child Adolesc Psychiatry.* 1995;34(9):1174–1184.
- Pfefferbaum B, Nixon SJ, Krug RS, et al. Clinical needs assessment of middle and high school students following the 1995 Oklahoma City bombing. *Am J Psychiatry.* 1999;156(7):1069–1074.
- Rando T. *Treatment of Complicated Mourning*. Champaign, IL: Research Press, 1993.
- Schuster MA, Stein BD, Jaycox LH, et al. A national survey of stress reactions after the September 11, 2001, terrorist attacks. *N Engl J Med.* 2001;345(20):1507–1512.
- Steinberg AM, Brymer M, Decker K, et al. The UCLA PTSD Reaction Index. *Curr Psychiatry Rep.* 2004;6:96–100.

Crisis Response

- Leash RM. *Death Notification: A Practical Guide to the Process*. Hinesburg, VT: Upper Access; 1994.

[2] From Communicating in a Crisis: Risk Communication Guidelines for Public Officials. Rockville, MD: Substance Abuse and Mental Health Services Administration; 2002.

- Schonfeld D. Crisis intervention for bereavement support: A model of intervention in the children's school. *Clin Pediatr.* 1989; 28(1):27-33.
- Schonfeld D. Almost one year later: looking back and looking ahead. *J Dev Behav Ped.* 2002;23(4):1–3.
- Schonfeld D. School-based crisis intervention services for adolescents: Position paper of the Committees on Adolescence and School Health, Connecticut Chapter of the American Academy of Pediatrics. *Pediatrics.* 1993;91(3):656–657.
- Schonfeld D, Kline M, and Members of the Crisis Intervention Committee. School-based crisis intervention: an organizational model. *Crisis Intervention and Time-Limited Treatment.* 1994;1(2):155–166.
- Schonfeld D, Lichtenstein R, Pruett MK, et al. *How to Prepare for and Respond to a Crisis* (2nd ed). Alexandria, VA: Association for Supervision and Curriculum Development; 2002.
- Substance Abuse and Mental Health Services Administration. National Child Traumatic Stress Network. Available at: http://www.nctsnet.org Accessed September 18, 2006.

Risk Communication and Media Issues

- Substance Abuse and Mental Health Services Administration. *Communicating in a Crisis: Risk Communication Guidelines for Public Officials.* Rockville, MD: 2002. SAMHSA Publication No. SMA02-3461.

Table 8.1 Concepts of death and implications of incomplete understanding for adjustment to loss

Concept	Example of incomplete understanding	Implication
Irreversibility		
Death is seen as a permanent phenomenon from which there is no recovery or return.	Child expects the deceased to return, as if from a trip.	Failure to comprehend this concept prevents child from taking the first step in the mourning process, that of appreciating the permanence of the loss and the need to adjust ties to the deceased.
Finality (Nonfunctionality)		
Death is seen as a state in which all life functions cease completely.	Child worries about a buried relative being in pain or trying to dig himself or herself out of the grave; child wishes to bury food with the deceased.	Can lead to preoccupation with physical suffering of the deceased and may impair readjustment; serves as the basis for many horror stories and films directed at children and youth (e.g., zombies, vampires, and other "living dead").
Inevitability (Universality)		
Death is seen as a natural phenomenon that no living being can escape indefinitely.	Child views significant individuals (i.e., self, parents) as immortal.	If child does not view death as inevitable, he or she is likely to view death as a punishment (either for actions or thoughts of the child or the deceased), leading to excessive guilt and shame.
Causality		
A realistic understanding of the causes of death is developed.	Child who relies on magical thinking is apt to assume responsibility for death of a loved one by assuming bad thoughts or unrelated actions were causative.	Tends to lead to excessive guilt that is difficult for child to resolve.

Adapted from Schonfeld D. Crisis intervention for bereavement support: a model of intervention in the children's school. *Clin Pediatr* 1989;28(1):27-33. Reprinted with permission of Sage Publications, Inc.

Chapter 9. Integrating Terrorism and Disaster Preparedness into Your Pediatric Practice

Relevance for Office-Based Pediatricians

Emergency preparedness should be exercised at all organizational levels, and office-based physicians (either in the hospital or free-standing practice) should understand the role of their specific office in the general system response to disasters. Integrating the office's response to disaster within the overall Federal, State, regional, and community response is essential. Office-based policies and procedures ideally should be specific to the location of the practice and its characteristics and be consistent with the policies of affiliate institutions and public and governmental agencies.

Preparedness for disaster by office-based physicians can be subdivided into two broad categories:

- Internal operations of the practice.
- External operations related to communication and coordination with other agencies, institutions, and the community.

Internal Operations of the Practice: Office Readiness

Framework for disaster preparedness. A child-oriented, comprehensive, emergency care system maintains the concept of systematic intervention in response to disasters while viewing the needs of the child in the context of family and community. This framework is particularly suited to the office-based physician, who attends to the whole child. Pediatric health care professionals bring knowledge about responses and needs of children involved in disasters and should work across public systems to render effective medical, educational, and community interventions. The objective is to ensure that the biological and psychological needs of children are addressed before, during, and after trauma.

Basic office readiness. There are several aspects to basic office readiness. They involve facilities, equipment and supplies, and records.

Facilities. In the face of a natural or manmade disaster, two modes of mitigating the results have been termed "hard" or "soft." Soft mitigation refers to emergency preparedness or emergency response as discussed throughout this chapter. Hard mitigation refers to engineering efforts in the built environment to withstand destruction. These include building standards for structures to withstand destruction from earthquakes, hurricanes, floods, fires, technological hazards, etc., and on-site permanent emergency systems such as fire suppression systems, uninterruptible power supplies, and standby generators.

Office-based physicians should be aware of the particular vulnerabilities of free-standing practice buildings based on geographic location, and their practices should comply with strict building code regulations and be equipped with emergency system back-ups. In the event of structural damage to the practice, there should be a plan for its relocation (e.g., by making

arrangements to share facilities with another practice). Considerations in such planning include the following:

- Partner with a practice that is unlikely to have incurred the same damage due to geographic location and clearly work out the operations of the practices ahead of time with respect to volume of patients, sharing of staff and resources, etc.
- Contact vendors for a change in delivery address, notify laboratories of relocation, and select alternative vendors and laboratories (in case the usual vendors have also been affected).
- Ensure that all staff members are apprised of the plans.
- Develop specific evacuation plans and conduct periodic in-service and practice drills.
- Periodically review the location of fire extinguishers, first aid, and emergency equipment.

Equipment and supplies. Offices should have emergency kits assembled that contain water, a substantial first-aid kit (including thermometer, blood pressure cuff, and stethoscope), radios, flashlights, batteries, heavy-duty gloves, food, sanitation supplies, and medical reference books and cards. Emergency supplies should be located both on-site and off-site. Lack of refrigeration for medications and vaccines is a likely scenario in a disaster. Back-up generators are important in case of outages. Back-up communication systems such as cellular phones, direct telephone lines that are not part of the regular telephone system, two-way radios, beepers, and ham radios should be considered. Practices located within hospitals should comply with JCAHO requirements.

Records. The Health Insurance Portability and Accountability Act (HIPAA) mandates that copies of records be stored off-site (some experts recommend at least 50 miles away) in case of catastrophe. This includes copies of patient charts and other vital records, even if most records are stored electronically. In addition to patient charts, other records that should be stored off-site include the following:

- Contact lists.
- Chain of command list.
- Pertinent contact information for government and emergency agencies.
- Copies of insurance policies.
- Loan applications.
- Real estate leases.
- Other materials relevant to the practice operations.

A number of Web sites that provide computer data storage capability are currently available. The choice of vendors should be researched carefully, and the various options for secure access (e.g., wireless) should be explored. Free-standing practices need to consider all contingencies. Practices located within larger hospital institutions need to be aware of the provisions made by the larger institution.

Communication system. Having a communication system in place for the office—including the chain of command, a listing of contact information for all staff members, and the delineation of staff responsibilities—is vital to office readiness in the event of a manmade or terrorist disaster.

Chain of command. A chain of command in case of emergency situations is routine in hospital settings. However, office-based physicians also need to consider this organizational step, which includes the following:

- Deciding a hierarchy (or who will be in charge) and their responsibilities vis-à-vis patient records.
- Informing staff members about recovery plans.
- Informing patients about new location and hours.
- Listing other important contacts.

Reviewing the nature of the catastrophe that may close or affect the practice should incorporate ways to contact employees and patients and take into account the possibility that phone lines will be inaccessible.

Contact list. A confidential contact list of all physicians, nurses, and other staff that includes home numbers, cellular phones, and alternative phone numbers should be kept by individuals designated in the chain of command. The contact list should also include back-up providers. Importantly, this confidential list should be kept in multiple places (e.g., the practice, outside location, and/or with the designated individuals). Practices may need to consider keeping a list of technologically dependent children who, during a disaster or emergency situation, may need specific planning (e.g., availability of back-up generators) and instructions about where to go in case equipment fails.

Staff responsibilities. Staff should be made aware that they have a professional responsibility to discuss their availability in case of disasters. This would include calling in to check where their services are needed and the feasibility of responding based on previously discussed office-readiness plans. Discussion with staff of their multiple responsibilities for their own families, work, and the community will help to alleviate concerns and anticipate problem areas. Staff should prepare a "Family Emergency Plan" so that they will be assured that the needs of their own families will be addressed while they are performing critical health care duties. The roles of the physician, nurse, and support staff should be briefly outlined. For example, everyone should know who will contact the police or fire department, who will aid in evacuation, who will reschedule patients, etc. Periodic exercises or drills can help to ensure that each staff member knows his or her role in the event of a disaster. Practices may choose to hire a consultant to advise staff members in the development of their plans. Again, individual practice efforts should be coordinated with more regional efforts as described below.

Infectious disease identification and control; chemical and radiological injuries and exposures. The office-based physician may be the first contact for an individual who is the victim of a biological agent and may be called on to treat or answer questions with regard to chemical and radiation exposure. Pediatricians should understand the following:

- The classification and qualities of possible biological agents.
- The natural history and management of biological, chemical, and radiological injuries and exposures.
- Chemical agents that may be used and their properties.

- Types of radiological terrorism.
- Decontamination procedures, especially those specific to children.
- Availability of antidotes and other therapeutics.

The procedure and numbers for alerting the proper authorities (e.g., Department of Health, CDC, etc.) should be detailed in the practice policies and procedures and chain of command protocol.

Triage, screening, and prioritization. Although office-based physicians may feel that they have no role to play in disaster planning or management, emergency pediatricians may need to draw on community pediatricians to provide the best possible management of children. Office-based physicians may be asked to help hospital-based pediatricians to determine which pediatric patients can be discharged or transferred to another hospital. In addition, office-based physicians will need to triage their own patients who show up at the practice to determine whether they need to go to the hospital or can be safely managed without emergency care. Office-based pediatricians also play a critical role in screening for psychological distress of the child and family (see also Chapter 8, Mental Health Issues).

Increasingly, risk assessment involves screening protocols applied at the school or community level to detect psychological needs of the population after a disaster. Leadership from the office-based physician can be instrumental in guiding these processes in a coherent and rational way.

Practice readiness and staff development. Staff development programs that provide accurate, reliable, and timely information on disaster preparedness for the office practice are essential. These programs can be organized by the practice, or the practice may choose to participate in programs developed by affiliate institutions specific to the needs of office-based physicians.

Insurance. Adequate business insurance is stressed in office readiness in the face of disaster. This may include the following:

- Determining how much revenue the practice can afford to lose, preferably slightly overestimating the necessary coverage.
- The details of the insurance policy (e.g., does it cover acts of terrorism, floods, etc.).
- Overall cost and possible liens against the business.

Keep in mind that building code requirements are essential in determining if insurance coverage applies. Reading and re-reading the policy is critical to upgrade coverage based on evolving or changing potential threats.

External Operations: Communication and Coordination with Other Agencies

Communication systems. The office-based physician should determine how the practice will link and coordinate efforts with affiliate hospitals, schools, daycare centers, local response teams, the local department of health, and city, State, regional and Federal efforts.

The chain of command established by the practice should identify the points(s) of contact within the organizational structure for communication with the various larger agencies.

Ideally, a list of all the relevant relationships should be developed in advance with contact numbers listed:

- Local and regional hospitals.
- Local emergency management agency.
- State and city departments of health.
- State and city police.
- State office of mental health.
- City fire department.
- City department of sanitation.
- Utility services.
- Medical equipment/supply resources.
- Pharmacies.
- Shelters.
- Poison Control Center (1-800-222-1222).
- Centers for Disease Control and Prevention (1-800-311-3435).
- American Red Cross (Disaster Assistance Info: 1-866-GET-INFO [1-866-438-4636]).
- Occupational Safety and Health Administration (OSHA).

The city Office of Emergency Management (OEM) can serve as a conduit for coordination of the various local agencies, including other Federal and State agencies. Practices can take advantage of already established relationships (e.g., with school-based health centers, daycare centers, hospitals). Communication before, during, and after a disaster would allow the efforts of the larger community to be coordinated with knowledge of the available resources at the practice level. Realistic scenarios can be investigated as part of community disaster drills.

Community as a resource. Community resources can mitigate the adverse effects of disasters. Successful programs include extensive training provided by fire, police, mental health, and emergency services personnel. The office-based physician can be integrated into this larger community response and should participate in drills and exercises. Three examples of such community programs include citizen disaster preparedness programs, the Citizen Emergency Response Training (CERT) programs, and the Collaborating Agencies Responding to Disasters (CARD) programs.

Citizen disaster preparedness programs. These include basic preparedness (e.g., a course to assist individuals in preparing their homes), neighborhood response teams (e.g., choosing a block captain, setting up a response area, establishing teams to address such things as search and rescue, safety and utilities, damage assessment), and advance training to augment public safety and response.

Citizen Emergency Response Training (CERT) programs. The CERT programs (established in Los Angeles, CA in 1985) provide citizen training through the fire department.

Collaborating Agencies Responding to Disasters (CARD) programs. The CARD programs (established in 1994 after the Northridge earthquake in Los Angeles) were originally organized to provide services for vulnerable and underserved populations (e.g., homeless individuals, veterans, at-risk youth, people with special needs). This group of agencies has extended its services to develop disaster preparedness plans. This kind of program has the advantage of linking culturally sensitive community services and preparedness, thereby addressing ongoing issues such as homelessness, cardiopulmonary resuscitation (CPR) training, and maintaining a set structure and mission. The clusters of service teams provide support services such as transportation, counseling, shelter and housing, health services, and commodities. Conceivably, this structure could accommodate a pediatric focus in collaboration with community-based pediatric health care professionals.

Policies and procedures. Office-based physicians should be aware of the emergency numbers and protocols of their local department of health. These policies should be included in the overall office practice manual for easy reference. If practices are affiliated with a hospital, also being aware of those policies and procedures will facilitate collaboration. When they exist, current best practices can be adopted with respect to emergency response plans and to physician and nurse training for disasters.

Communicating Directly with Children and Families

The pediatrician plays a central role in disaster and terrorism preparedness with families and children. Families view pediatricians as their expert resource, and most expect pediatricians to be knowledgeable in areas of concern. Providing expert guidance entails both educating families in anticipation of events and responding to questions during and after actual events.

In many areas of the country, the threat of natural disasters is ongoing, and guiding and educating families on home disaster preparedness can be done in the pediatrician's office or as a community focus. Family preparedness may include training in cardiopulmonary resuscitation, rendezvous points, lists of emergency telephone numbers, and an out-of-the-area friend or relative whom all family members can contact after the event to report their whereabouts and condition. Home preparedness (such as installing storm shutters or earthquake-proofing the home) should be covered. Parents should maintain emergency supplies of food, water, and medicine; a first-aid kit; and clothing. Family members should know the safest place in the home, make special provisions, know community resources, and have a plan to reunite. Medications for chronic illness and resources for children who depend on technological means for survival should be included in the plan (see also the AAP Family Readiness Kit at www.aap.org/family/frk/frkit/htm).

Answering questions during events. During any event, children and families will receive good and bad information from a multitude of sources, including friends, media, and public officials. The problems caused by panic, overreaction, and overwhelming the emergency health care system with anxious families cannot be overstated. A well-educated and available pediatrician who can appropriately respond to numerous and varied questions can be of great service.

Advice for families of children with special health care needs. Pediatricians should provide guidance to families of children with special health care needs. This may include the following:

- Notifying utility companies to provide emergency support during a disaster.
- Maintaining a supply of medications and equipment in case availability is disrupted during a disaster.
- Knowing how to obtain additional medications and equipment during times of a disaster.
- Training family members to assume the role of in-home health care providers who may not be available during a disaster.
- Keeping an up-to-date emergency information form to provide health care workers with the child's medical information in case the regular care provider is unavailable.
- Knowing back-up hospitals/providers in the region in case primary hospital/specialists/providers become unusable or are unavailable.
- Providing advice on power of attorney, living wills, advance directives, and other important legal tasks/documents.

Relevance for Hospital-Based Pediatricians

In mass casualty incidents, including those involving chemical and biological agents, casualties among children and adults could be significant. Because children are likely to become victims in many disaster events, pediatricians should assist in preparedness planning to ensure coordinated responses of local hospitals that may in fact each have limited pediatric resources. In addition, health care facilities could be a primary or secondary target themselves. Also, facilities may be overwhelmed by massive numbers of anxious individuals and families. Pediatricians working in or supporting hospitals can play a vital role in ensuring the enhanced care of the pediatric disaster victim by participating in all levels of disaster preparedness planning.

Emergency Department Readiness

The hospital disaster alert system is designed to triage victims in the field and carefully distribute them among available resources to keep a single facility from being overwhelmed. However, in many crisis situations, facilities are vulnerable to inundation with patients who arrive in large numbers without EMS transport and pre-entry triage. Pediatricians working in or supporting hospitals should interact with the planning committee to ensure adequate training and preparation of supplies and treatment areas in the emergency department. Pediatricians working in hospitals can be key facilitators between emergency department services, critical care services, and regular inpatient services. Coordination with the local community should involve primary/prehospital/infrastructure response (with liaison planning to State and Federal agencies) and community/citizen response. Considerations should be made as usual referral patterns may not typically include accepting pediatric patients.

Primary/prehospital/infrastructure response includes the following:

- EMS.
- Fire.

- Police, Environmental Protection Agency (EPA), sheriff.
- Military (local or regional).
- Regional poison centers.
- Local health department.

Community/citizen response involves the following:

- Schools, public and private.
- Daycare centers, public and private.
- Service groups (Kiwanis, Rotary, Salvation Army, parent/teacher associations (PTAs), etc.).
- Nonsecular groups (churches, synagogues).
- Public recreation administrations (zoos, amusement parks, sports stadiums, museums, etc.).

Inpatient Service Readiness

Anticipating surge capacity for inpatient care is vital in preparedness planning and perhaps is the greatest contribution of pediatricians working in hospitals. Areas that should be considered include the following:

- Increasing the number of inpatient beds within a community (e.g., by using several strategies such as converting cafeterias and meeting spaces into ward capacity or making arrangements with other community hospitals). Local hotels, school gymnasiums, etc., may be converted into low-acuity medical facilities with some planning.
- Contingency plans for acquiring or maintaining essential services, such as water, electricity, portable oxygen, garbage/trash removal, etc.
- Planning for stockpiling or readily acquiring medical supplies such as vaccines, antitoxins, and antibiotics (in dosages, formulations, etc., appropriate for pediatric patients). In addition, pediatric-specific supplies and equipment in a full range of sizes to accommodate pediatric patients should be available.
- Networking community resources to organize volunteers to become proxy caretakers for orphaned children.

Hospital Infrastructure Needs

Crisis drills. Hospital and community-wide drills are essential to preparedness planning. They need to be done on both a wide scale and with a narrow focus. Drills should include not only the initial triage and decontamination, but also the 48-72 hours after impact to measure readiness with all provider and support services that will be needed. Specific drills should be planned and practiced for evacuation in response to fire or other disasters.

Infection control plans. Infection control plans closely parallel quarantine procedures in the community and on a public health basis. The in-hospital level involves quarantine or isolation and control measures to limit spread of infection to staff and other patients.

Quarantine procedures. Quarantine procedures should be a well-established means of limiting the spread of infection. Quarantine may become an active part of inpatient needs in a given hazard-related disaster. Children who become ill may require isolation to prevent spread of disease to other patients and health care providers. The exact nature and severity of quarantine will depend on the specific hazard involved. Close coordination with the public health service, CDC, and local poison centers is essential in both the planning and execution stage.

Staff training. Staff training should include the following:

- Training in use of protective gear.
- Orientation to all aspects of the plan from the disaster site to the emergency department to hospital floors, as well as to rehabilitation and rebuilding in the community.
- Staff preparedness for notification, transportation to treatment sites, self preparedness (emergency packages of personal items), strategies for coping with family demands, psychological demands, and plans for personal health and hygiene.
- Support for families of health care workers so that the health care workers are available to provide services.
- Mechanism for tracking resources.
- Media/public communication issues.

Bibliography

- Cascardo D. Preparing your medical practice for disaster. *Med Pract Manage*. 2002;18(1):33–35.
- Caffo E, Belaise C. Psychological aspects of traumatic injury in children and adolescents. *Child Adolesc Psychiatric Clin No Am.* 2003;12:493–535.
- Chemtob CM, Nakashima JP, Hamada RS. Psychosocial interventions for postdisaster trauma symptoms in elementary school children. *Arch Pediatr Adolesc Med*. 2002;156:211–216.
- Cowan ML, Cloutier MG. Medical simulation for disaster casualty management training. *J Trauma.* 1988;28(1Suppl):S178–S182.
- Flowers LK, Mothershead JL, Blackwell TH. Bioterrorism preparedness: II. the community and emergency medical services systems. *Emerg Med Clin No Am*. 2002;20(2):457–476.
- Harner A. Will you be ready when your patients need you the most? Disaster planning for the medical practice. *MGMA Connexion*, Nov/Dec 2001: 40–41.
- Hazinski FM, Markenson D, Neish S, et al. Response to cardiac arrest and selected life-threatening medical emergencies: the medical emergency response plan for schools. A statement for healthcare providers, policymakers, school administrators, and community leaders. American Heart Association, Emergency Cardiovascular Care Committee. Policy Statement, *Pediatrics*. 2004;113(1):155–168.
- Laor M, Wolmer L, Spriman S, Wiener Z. Facing war, terrorism, and disaster: toward a child-oriented comprehensive emergency care system. *Child Adolesc Psychiatric Clin No Am*. 2003(12);343–361.

- Leonard RB. Role of pediatricians in disasters and mass casualty incidents. *Ped Emerg Care*. 1988;4(1):41–44.
- Lichterman JD. A "community as resource" strategy for disaster response. *Public Health Rep*. 2000;115(2–3):262–265.
- Mattox, K. The World Trade Center attack disaster preparedness: health care is ready, but is the bureaucracy? *Crit Care* 2001;5:323:325.
- Perry RW, Lindell MK. Preparedness for emergency response: guidelines for the emergency planning process. *Disasters*. 2003;27(4):336–350.
- Redlener I, Markenson D. Disaster and terrorism preparedness: what pediatricians need to know. *Adv Ped*. 2003;50:1–37.
- Rose MA, Larrimore KL. Knowledge and awareness concerning chemical and biological terrorism: continuing education implications. *J Cont Ed Nursing*. 2002;33(6):253–258.
- Somers GT, Maxfield N, Drinkwater EJ. General practitioner preparedness to respond to a medical disaster. *Aust Fam Physician.* 1999;28(Suppl 1):S10–S14.
- Veenema GT. Chemical and biological terrorism preparedness for staff development specialists. *J Nurses Staff Devel*. 2003;19(5):215–222.

Chapter 10. Working with Government Agencies

Introduction

The challenge of dealing with the threat of terrorism in the United States is daunting not only for disaster planners, but also for our medical system and health professionals of all types, including pediatricians. All possible forms of terrorism should be considered, including chemical, biological, explosive, radiological, and nuclear events. Pediatricians need to be able to answer concerns of patients and families, recognize signs of possible exposure to a weapon of terror, understand first-line response to such attacks, and sufficiently participate in disaster planning to ensure that the unique needs of children are satisfactorily addressed in the overall process.

Community, Government, and Public Health Preparedness

Emergency preparedness is important at many levels—personal, family, community, regional, State, and Federal—with the State and Federal Governments having pivotal roles. The Federal Government provides significant funding for disaster preparedness and response and to a large extent establishes the framework that is then followed by States, regions, and communities. In disaster response, the funding and planning tends to be top down, while the response and use of resources tends to be from the bottom up. In other words, as resources are exhausted at the local level, assistance is requested from the next level, such as the State, which then requests Federal assistance.

Volunteer organizations, such as the Red Cross and Salvation Army, also have key roles in disaster response. Recent concepts of disaster and aftermath response and planning include the involvement of neighborhoods and families and have begun to include needs at the individual level. A successful response to a disaster requires the interaction of personnel and resources from multiple agencies in an organized and coordinated manner according to a well-formulated plan. While this planning has increased in recent times, the attention to the unique needs of children and the inclusion of pediatric expertise in the planning phases is still insufficient and deserves much more attention.

Community Response

Local governments are the first line of defense in emergencies and are primarily responsible for managing the response to most disasters. The primary responsibility for the protection of citizens belongs to such local elected officials as mayors, city councils, and boards of commissioners. When a local government receives a warning that an emergency could be imminent, its first priorities are to warn citizens and to take whatever actions are needed to minimize damage and protect life and property. If necessary, an evacuation may be ordered. The emergency operations plan is at the center of comprehensive emergency planning. This plan spells out the scope of activities required for community response. It should be a living document that accurately describes what the community can realistically do. Historically, these documents usually do not address

pediatric considerations and in only the rarest of cases have pediatricians been part of the planning process.

State Government

All States have laws that describe the responsibilities of the State government in emergencies and disasters. These laws provide governors and State agencies with the authority to plan for and carry out the necessary actions to respond to and recover from emergencies. State emergency management legislation describes the duties and powers of the governor, whose authority includes the power to declare a state of emergency and to decide when to terminate this declaration.

Performing and maintaining the provisions of emergency management legislation is generally the responsibility of the State emergency management offices (some municipalities also have offices of emergency management). These offices are organized in a number of ways and have different names. Emergency managers are responsible for preparing for emergencies and for coordinating the activation and use of resources controlled by the State government when they are needed to help local governments respond to and recover from emergencies and disasters. In its coordinating role, the State emergency management office is involved in virtually all serious emergencies, terrorist events, and natural or manmade disasters.

Using procedures specified in the State emergency operations plan, the State emergency management organization coordinates deployment of personnel and resources to the affected areas. Again, pediatric concerns are rarely considered. The emergency operations plans of many States do not include child-specific guidelines. Pediatricians need to be involved in the State emergency planning committees to ensure that pediatric considerations are included in State plans and become part of the plans of each State agency and part of funding for disaster and terrorism preparation. This pediatrician involvement can be accomplished through both grassroots efforts and involvement in local chapters.

Federal Government

The authority for Federal involvement in disasters is based on provisions of the Stafford Act.[1] This Act establishes the presidential declaration process for major disasters and emergencies, provides for the implementation of disaster assistance, and sets forth the various disaster assistance programs. FEMA's role is to coordinate the delivery of Federal assistance by managing its own programs and by coordinating disaster assistance from other Federal departments and agencies. FEMA coordinates these activities using the interagency Federal response system, also called the Federal Response Plan (FRP). The FRP describes the basic mechanisms and structures by which the Federal Government mobilizes resources and conducts activities to augment State and local response efforts. To facilitate the provision of Federal assistance, the FRP uses a functional approach to group the types of Federal assistance that a State is most likely to

[1] Robert T. Stafford Disaster Relief and Emergency Assistance Act, Public Law 93-288, Sections 5121-5206, et seq of Title 42 United States Code.

need under the 12 Emergency Services Functions (ESFs). Each ESF is headed by a primary agency that has authorities, resources, and capabilities in the particular functional area. Other agencies have been designated as support agencies for one or more ESFs based on their resources and capabilities to support the functional area. The FRP is also important in that most States use it as a base for the structure and some content of their emergency operations plans. In turn, local emergency operations plans are based on the State plans. (See Chapter 2, Systems Issues, for more information.)

One of the major functions physicians will be involved with and should become familiar with is Emergency Services Function Number 8 (ESF #8), the Health and Medical Services Function. ESF #8 provides assistance to supplement State and local resources for public health and medical care needs during a disaster. In a federally declared disaster, the U.S. Public Health Service (USPHS), a component of the U.S. Department of Health and Human Services (DHHS), forms a Crisis Action Team and activates the National Disaster Medical System (NDMS) as needed to provide health and medical care assistance. A Disaster Medical Assistance Team (DMAT) is a deployable unit of 35 physicians, nurses, technicians, equipment, and supplies for austere medical care. A Management Support Unit (MSU) manages field health and medical resources of deployed DMATs. A Disaster Mortuary Assistance Team (DMORT) provides a temporary morgue facility, victim identification, processing, preparation, and disposal of remains. A Metro Medical Strike Team (MMST) provides assistance in the medical treatment/management of chemical, biological, or radiological incidents resulting from deliberate or accidental acts. In some cities, there are Metropolitan Medical Response Teams that provide additional medical response capability. The parent agency of all of these groups is the United States USPHS, with assistance from the Department of Homeland Security (DHS).

Public Health Preparedness

On a larger scale, we should also recognize the need for public health preparedness. This requires the existence of a strong public health system. To allow for rapid and efficient response, we need both central organization as specified by Federal planning and State implementation and decentralization of some resources, such as diagnostic capabilities.

It is important for pediatricians to understand the importance of public health and their relationship to departments of health. This includes their role in public health, reporting requirements and mechanisms, and mechanisms for receiving and soliciting information from departments of health.

Advocating for Children and Families in Preparedness Planning

To ensure that children are included in government and community planning and preparedness activities, informed and motivated pediatricians should advocate for children at all levels. This advocacy can take several forms. Grassroots advocacy can include efforts to ensure legislation and funding to support an emphasis on children in disaster planning at every level. Pediatricians can also serve as expert advisors to local,

State, and Federal agencies and committees. Often, this activity can be through involvement in professional organizations such as the American Academy of Pediatrics, its chapters, and special committees and task forces.

Pediatricians as the experts in the care of children should:

- Advocate for inclusion of the needs of children in all Federal, State, and local disaster planning.
- Advocate for research on the pediatric aspects of biological, chemical, and radiological terrorism, including mechanisms, pathophysiology, and treatments. This includes the availability of appropriate medications and antidotes.
- Work with DMATs to ensure that they are equipped and trained for the care of children.
- Assist in developing a hospital disaster plan that provides for the proper care of children.
- Provide on-site emergency and primary health care at emergency shelters.
- Be involved in emergency medical services (EMS), e.g., develop proficiency in cardiopulmonary resuscitation and first aid, train first responders in pediatric assessment, assist in development of prehospital pediatric protocols, help establish protocols for consent to treat and identification of minors, and ensure the availability of pediatric resources.

At the local and community level, pediatricians should:

- Work with local police, fire, and EMS departments to ensure that their plans and equipment address the needs of children.
- Be involved with local and community emergency preparedness task forces and committees.
- Work with schools, child care centers, and other locations where children spend their time to ensure that they have adequate emergency plans.

Similarly, at the State level, pediatricians should:

- Ensure that State emergency management, department of health, and EMS advisory committees have pediatric expertise as part of their membership.
- Advocate for all State disaster and terrorism education and funding to require inclusion of provisions for children.

Resources Available from Government Agencies

A number of State and Federal Government agencies have information, tools, electronic media, and other materials to assist physicians, including pediatricians, and other health care providers in disaster planning, preparedness, and response. Many of these resources can be found in the bibliographies that follow chapters in this report.

Another Federal agency, the Agency for Healthcare Research and Quality (AHRQ), has provided support for research on bioterrorism preparedness and response and the development of materials to improve the Nation's capacity to respond to bioterrorism and other disasters. AHRQ's multimillion dollar investment in bioterrorism research

recognizes that community clinicians—including pediatricians—have a vital role to play in disaster planning, preparedness, and response at the Federal, State, and local levels. Hospitals, health care systems, professional organizations, and other groups also have essential roles in the public health infrastructure and need evidence-based information and tools to help them respond appropriately in the event of a disaster.

To inform and assist these groups in meeting the health care needs of the U.S. population in the face of bioterrorist threats and other disasters, AHRQ-supported research has focused on the following:

- Emergency preparedness of hospitals and health care systems for bioterrorism and other rare public health events.
- Technologies and methods to improve the linkages between the personal health care system, emergency response networks, and public health agencies.
- Training and information needed to prepare community clinicians to recognize the manifestations of bioterrorist agents and manage patients appropriately.

AHRQ contracted with the American Academy of Pediatrics to develop this report to ensure that the needs of children are an integral part of disaster planning and preparedness activities.

AHRQ has developed a number of other reports, tools, and other materials that focus on disaster planning, preparedness, and response. They are available online at the Agency's Web site (http://www.ahrq.gov/browse/bioterbr.htm), and in many cases, print copies are available from the AHRQ Publications Clearinghouse. Information about print availability is also online at the above Web site.

Chapter 11. Conclusion

This report has presented detailed information to assist pediatricians—both those based in the community and those based in hospitals and other health care systems—in becoming active participants in disaster planning, preparedness, and response. Additional sources of information and many other valuable resources are provided in the bibliographies that follow each chapter. In addition, valuable lessons can be drawn from several disasters, both manmade and natural, that have occurred in the United States.

The hurricanes in the Gulf Coast and their aftermath serve as a reminder that nature is capable of destruction greater than that of most terrorist attacks. An important lesson regarding the organization and focus of disaster response is to include natural as well as manmade events in planning and preparedness efforts. Consideration also should be given to the organizational and logistical issues involved in evacuating a major city.

Systems Issues

Activities pertaining to disaster planning, preparedness, and response take place at all levels, including Federal, State, regional, and local, and everyone has a role to play. For example, at the Federal level, the Centers for Disease Control and Prevention (CDC) has provided excellent information on the impact of hurricanes and on methods to help mitigate the impact and facilitate management immediately after the event (see http://www.bt.cdc.gov/disasters/hurricanes/). Much of this information can be applied to both natural disasters and terrorism. The Web site includes information on family readiness, evacuation, power loss, recovery (including keeping food and water safe, preventing injury, monitoring infections, etc.), animal and insect hazards, environmental issues, clean-up safety, returning home, and mental health issues.

Regional, State, and Local Efforts

Valuable lessons have been learned from our experiences with Hurricane Katrina and its aftermath. A large part of the problem was insufficient coordination and planning at all levels. The situation was exacerbated by the need for evacuation of large numbers of people and their long-term displacement from their homes and schools.

Future planning and preparedness efforts in major U.S. cities should recognize the potential for severe and long-term consequences. In disasters, State and Federal aid is always going to take some time to arrive. Local and regional authorities, as well as individual families, should have an effective plan in place that includes efforts to be undertaken before other outside assistance can arrive. This should include an effective evacuation plan that covers quickly setting up emergency shelters, providing food and managing water sanitation for large numbers of people, and providing affected individuals—including children who may or may not have chronic illnesses and other special needs—with medical and mental health care.

Vulnerable Populations

In a disaster, the most vulnerable are going to suffer the most and need the most assistance. Clearly, vulnerable populations include the poor, the infirm, individuals with mental illness, the elderly, and children. Hospitalized patients, people in nursing homes, and incarcerated individuals are at special risk in a natural or manmade disaster.

Hospitalized children, particularly premature infants and those in neonatal intensive care units, are particularly vulnerable. Their lives may depend on the ability of hospitals to maintain critical emergency power and life support equipment until transfer to other regional specialty facilities can be arranged.

These situations highlight the need for including large numbers of technologically dependent patients, many of whom require highly specialized regionalized care, in planning efforts to mitigate the impact of a disaster.

Separation of Children from Families

Hurricane Katrina also highlighted the need to plan for the likelihood of numerous children being separated from their parents or other caregivers. The National Center for Missing and Exploited Children has a hotline (1-800-843-5678) and Web link (http://www.missingkids.com/) to report missing children, missing adults, and found children. The Red Cross also has a hotline (1-866-GET-INFO) and Web link (http://www.redcross.org) to help separated family members find each other. As of March 2006, the National Center reported that all children separated from families as a result of Hurricane Katrina have been reunited with their families.

This issue should receive more attention in preparedness and mitigation plans. Pre-disaster identification of children (e.g., name tags, other forms of ID, etc.), especially for those who are not verbal, or who cannot give their own name, a parent's name, or other critical information, should be considered. Neonates and their mothers are purposefully given matching ID bracelets in hospitals immediately after delivery so the identity of the maternal-child pair is never in doubt. Similar identification of parent-child pairs at the time of separation (e.g., during rescue or evacuation) could greatly aid in the identification of the child and more accurately track and reunite children separated from their parents.

Sheltering Families

The immediate first response to a disaster the magnitude of Hurricane Katrina includes the mobilization and evacuation of a large region. Subsequently, those displaced from their homes, schools, and neighborhoods will require basic necessities, including a place to stay, water, food, clothing, health and mental health care, etc.

The unique needs of children in shelter situations include the need for special foods (e.g., formula), clothing and sanitation (e.g., diapers), and sleeping accommodations (e.g., cribs). Efforts to distract, entertain, comfort, and even separate families with crying newborns and toddlers will help calm other evacuees in the shelter who find these sounds discomforting.

Planning for special medical needs and for mental health care that focuses on children's unique developmental stages is also critical.

The immediate issues of emergency sheltering, providing food and water, and other necessities do not take into account the problem of returning some semblance of stability and normalcy to children and families. These significant issues will likely create severe mental health issues for an extended period of time for many of these displaced children.

Providing Urgent Medical Care to Large Populations of Displaced Children

Following Hurricane Katrina, a Mobile Pediatric Emergency Response Team (MPERT) staffed a temporary pediatric clinic near the displaced population. As Houston, TX, became the new home for 300,000 people, a percentage of evacuated children needed medical triage, evaluation, and management. They otherwise would have been likely to seek care at local emergency departments, many of which were already at surge capacity. To prevent severe overcrowding in these local emergency departments, a large children's hospital expended the resources necessary to care for these displaced children near the site of their temporary shelter.

A temporary pediatric clinic provided a high level of care for the evacuated children. The clinic cared for approximately 2,000 patients. Notably, not a single child died, and less than 50 required EMS transport to area hospitals. Other cites—e.g., Dallas, Birmingham, Ft. Worth, and Baton Rouge—also set up temporary clinics.

Setting up a Temporary Pediatric Clinic—Lessons Learned

In every large metropolitan area that received evacuees, temporary pediatric clinics evolved. These temporary clinics matched caregivers who were comfortable with and accustomed to providing care to children with incoming newborns, children, and adolescents with medical needs. Equally important, children were removed from adult waiting lines, allowing moe effective care to be given to that population as well.

- Physicians and nurses who are trained and experienced in the emergency care of children should always be included, even at the most basic level, when planning for and responding to a disaster.
- Access to local tertiary pediatric care resources should be arranged for in advance, and the tertiary care provider supplying those resources should be involved in the planning discussions.
- While volunteerism is essential in the event of a mass casualty, guaranteed staffing of the MPERTS should be a priority.
- The medical director of the MPERTS should be knowledgeable in pre-hospital medicine and associated or familiar with the local EMS system.
- The appropriate allocation of physician and nursing resources is vitally important to patient flow:
    - Physicians trained and experienced in pediatric emergency management should be placed at main triage and as charge physicians at the pediatric arena clinic.
    - A good charge nurse will either make or break patient flow.

- MPERTS should be able to mobilize rapidly.
- Cooperation with regional disaster command is essential.
- Choosing the appropriate venue for the staging of the disaster response is critical:
    - Providing enough room upfront to create a scaled-down version of an emergency department, including an area for observation and isolation, is critical.
- Mental health care and social services should be made available to the evacuated population as early as possible.
- A centrally located functional communication device (e.g., phone, cell phone, radio) is crucial to successful implementation; it provides access to additional essential staff and services quickly, on an ongoing basis.
- There should be a planned exit strategy:
    - The clinic should not be allowed to outlive its resources.
    - Once the cost of running the clinic outweighs the cost of referring those patients to the emergency department, the clinic should close its doors.

Environment

Many of the environmental problems that occur after a natural or manmade disaster are typical and predictable. These problems include temperature extremes; lack of clean water, food, and electricity; and environmental hazards not usually present before the event. See Table 11.1 for some of the more common environmental conditions and hazards that are likely to be present following a disaster.

Medical Needs

The medical needs of children and families are also predictable and consistent because they closely match the needs of the affected children and communities before the disaster. See Table 11.2 for common pediatric medical issues, challenges, and adaptations that can be expected after a disaster.

A Final Word

Timely response and appropriate medical management are essential to minimizing injuries and maximizing survival when a disaster occurs. Being prepared ahead of time is the key to timely and appropriate medical care. Children and other vulnerable populations have special needs that must be considered in the course of planning for a mass casualty event.

Pediatricians can play a very important and unique role in advocating for the needs of children and families who seldom receive enough attention in disaster planning. Response resources dedicated to pediatric populations remain unavailable or extremely limited for most emergency medical response activities related to disasters, even though victims often include children. To address this shortcoming, it is vitally important that pediatricians and other representatives of special populations take part in local, State, regional, and Federal disaster planning to ensure appropriate care for the most vulnerable populations.

Table 11.1 Environmental constraints to pediatric medical care after large-scale natural disasters

Physical constraint	Consequences/examples
Temperature/exposure	Heat Cold Overexposure to the sun Dehydration
Lack of clean water	Dehydration Poor hygiene Inability to comply with wound care instructions Potential for GI complications
Lack of food	Inadequate nutrition Inappropriate diet Potential for GI complications
Lack of electricity	Inability to use non-battery-powered medical devices such as nebulizers, pumps, ventilators Inability to maintain medications at appropriate temperatures Difficulty maintaining safe thermal environment Inadequate light/ventilation as a safety hazard Difficulty receiving critical information about medical care and available medical and non-medical assistance
Hazardous environments	Chemicals Physical hazards (e.g., nails, tree limbs, debris, roofs, unregulated traffic intersections) Tools (e.g., chainsaws) Weapons Animals or insects Allergens/plants (e.g., poison ivy)

Table 11.2 Pediatric medical complaints after large-scale natural disasters: challenges and adaptations based on post-hurricane responses

Challenges	Adaptations to Usual Care	Decisions
	Pulmonary	
• Bronchospasm is common in those with and without histories of asthma. • Children with bad/labile asthma present early due to stress, environmental triggers, lack of medication. • Stable asthmatics start showing up as triggers increase or medication runs out. • Children with bronchospasm due to respiratory infection start to present after the first 3–5 days. • October storms correspond to high allergy season and a slight peak in RSV incidence. • Winter storms may occur during RSV outbreaks.	• Need adequate supplies to treat patients. • Premixed beta agonists for nebulizers (infant and child dosing). • Nebulizer capability with and without oxygen. • Pediatric nebulizer masks and pipes. • Oral and parenteral steroids. • Peak flow monitoring helpful but not essential. • *Outpatient treatment:* • Allow use of facility's electricity for families giving their own nebulization. • Using MDIs with spacer chambers more frequently. • Be liberal with steroids. • Counsel regarding allergen exposure.	• Lower threshold for admission based on available resources and ongoing hazards. • Consider recommendation to temporarily remove child from the area to healthier environment. • Temper decisions with consideration of family's existing resources and demands on family members.

Table 11.2 Pediatric medical complaints after large-scale natural disasters: challenges and adaptations based on post-hurricane responses, continued

Challenges	Adaptations to usual care	Decisions
	Gastrointestinal	
• Close living quarters may lead to transmission of GI viral illnesses, • Limited water and facilities for washing; limited diaper/hygiene supplies. • Inadequate sanitation in field kitchens/food distribution points. • Norovirus precautions go beyond soap and water or alcohol. • Erratic availability of potable water and oral rehydration solutions. • MREs have high sodium/high calorie content.	• Ask about sheltering situation. Give specific infection control instructions (written if possible). • Health care sites can act as distribution points for hygiene items such as alcohol solution, diaper wipes, diapers, soap, garbage (biohazard?) bags/gloves, bleach. • Maintain contact with public health officials. • Ask about diet specifics, including origin of drinking water and food storage conditions. • Warn families of need to increase fluid intake if eating MREs. • Consider unusual electrolyte abnormalities in clinically dehydrated children. • Distribute oral rehydration solutions. • Focus on oral rehydration protocols unless staff and IV fluids are in adequate supply. • Limit use of antiemetics and antidiarrheals in children. • Minimize infant formula-switching. • Use fecal volume replacement techniques in cases of diarrhea.	• Admission decisions must include consideration of shelter status. • Lower admission threshold if adequate outpatient management is doubtful. • If in doubt, schedule patient rechecks.

Table 11.2 Pediatric medical complaints after large-scale natural disasters: challenges and adaptations based on post-hurricane responses, continued

Challenges	Adaptations to usual care	Decisions
	Infectious Diseases	
• Infections will mostly follow existing community patterns. • "Third world" epidemics have not occurred in the US. • Isolation/segregation of infected people is difficult in the post-storm environment. • Children need different preparations of antibiotics, some requiring controlled environmental conditions. • Pharmacies and drug supplies may be limited and may focus on adult medications. • Skin infections are common; good hygiene is not. • Penetrating injuries to the foot are common; Pseudomonas must be suspected. • Community-acquired MRSA is an increasing problem. • Animal control may be problematic; may need to vaccinate patients against rabies.	• Contact local public health or hospital officials for intelligence regarding existing infection patterns and sensitivities. • Cooperate with public health officials in monitoring efforts. • Assist in informing shelter staffs of infection patterns seen and what to look for. • Assist public health personnel with projects needed to protect exposed high-risk groups, such as giving VZIG to exposed immunocompromised victims or tetanus boosters to those who need them. • Educate patients and families about infection control issues, especially if they are shelter residents. • Prescribe antibiotics judiciously; use the simplest appropriate form for the shortest practical course. • Use alternative medication formulations (chewable tabs, crushed tabs) and those that do not require refrigeration. • Obtain and distribute information about pharmacies in operation. • Inform local pharmacies about prescribing privileges for federal responders. • Consider distribution of starter doses of medications. • Distribute hygiene and wound care supplies, insect repellant, and topical or oral medications for itching/inflammation. • Plan follow-up for penetrating and contaminated injuries (especially nails into feet). • Consider using ciprofloxacin for children with penetrating wounds into feet. • May use first-generation cephalosporins for most skin infections. • Consider adding TMP-SMX or clindamycin if community-acquired MRSA is suspected. • Communicate with local public health authorities about rabies exposure. Recognize that most children will not need a tetanus booster.	• Consider family's environment and mobility when making decisions about admission vs. outpatient treatment with rechecks. • May need to admit children with highly contagious diseases to avoid exposing others in a crowded environment. • Consider sending infected children out of the area if more appropriate shelter is available. • Maintain low admission thresholds for the very young with fever and for immunocompromised patients. • Use antibiotics judiciously.

Challenges	Adaptations to usual care	Decisions
Trauma		
• The post-storm environment is hazardous! • Children may not have adequate supervision or may be asked to perform inappropriate tasks. • Children are risk-takers. • Minor skin and musculoskeletal injuries are common. • Penetrating injuries by contaminated objects are common. • Skin foreign bodies are common. • Major trauma is not common. • Increased chance of: o Carbon monoxide exposure o Hydrocarbon and bleach ingestion/aspiration o Ingestion of medications o Drowning o Traffic incidents due to unregulated intersections • Intentional injury	• Carefully document mechanisms of injury. • Be prepared to stabilize a badly injured child while arranging for transfer. • Identify local pediatric trauma and burn care resources. • Have access to poison control resources. • If lacking x-ray capabilities, splint the injured extremity on any child with bony tenderness, regardless of lack of deformity. • Emphasize elevation and splinting of an injured extremity for control of pain and swelling; ice may not be a viable option. • Provide the best possible initial wound care in an environment as comfortable for the patient as possible. • Consider delayed/no closure for contaminated wounds or possible retained foreign bodies. • Consider self-absorbing sutures for children with lip, finger or toe lacerations. • Use skin glue only if wound is clean and can be kept dry. • Don’t forget pain management!	• Follow-up care may be biggest issue; patients may need to go to another facility to initiate contact with follow-up caregivers. • Make some allowances for unusual circumstances but be alert for potentially negligent or dangerous family situations. • Depending on available medical resources, conscious sedation/analgesia should remain a consideration for painful or stressful procedures.

Challenges	Adaptations to usual care	Decisions
Psychosocial		
• Families may have difficulty coping with their child's illness or injury. • Delays in seeking care may be more common than in ordinary circumstances. • Families may not have had primary care resources before the disaster. • Compliance with treatment recommendations may be difficult for many reasons. • Stress may lead to higher risk for domestic and child abuse. • Pediatric mental health goes beyond PTSD. • Children with mental health issues may present with acute or prolonged nonspecific physical symptoms. • Parents are often not well informed about children's reactions to catastrophic stress.	• Assume family members do not get your message the first time. • Write down instructions for the family. • Always ask, "Is there anything else we can help you with?" • Address children directly; let them know what they have to say is important and that they have a role in feeling better. • Encourage children to express their feelings. • Make the visit as pleasant as possible for the child. • Explore alternatives with the family to help ensure compliance with treatment recommendations. • Avoid judgmental attitudes. • Identify local resources for family psychosocial support. • Use available mental health resources.	• Recognize risks for abuse or intentional neglect; know the local reporting mandates and procedures. • Try to keep family members together. • Remember that a child's reactions will reflect what's going on with the rest of the family. Ask! • Be willing to accept reasonable therapeutic compromises that help increase family coping abilities without jeopardizing patient care.

Notes: MDI = metered-dose inhaler
MRE = Meal, Ready-to-Eat
MRSA = methcillin-resistant *Staphylococcus aureus*
RSV = respiratory syncytial virus
VZIG = Varicella zoster immunoglobulin

Acronyms

AAP	American Academy of Pediatrics
ACEP	American College of Emergency Physicians
ACIP	Advisory Committee on Immunization Practices
AFRRI	Armed Forces Radiobiology Research Institute
AHRQ	Agency for Healthcare Research and Quality
AMU	Atomic mass unit
ARDS	Acute respiratory distress syndrome
ARS	Acute radiation sickness/syndrome
BAL	British antilewisite (dimercaprol)
BAT	Biodosimetry assessment tool
BIGIV	Botulism immune globulin intravenous
BSA	Body surface area
BWC	Biological Weapons Convention
CARD	Collaborating Agencies Responding to Disasters
CBT	Cognitive behavioral treatment
CDC	Centers for Disease Control and Prevention
CERT	Citizen Emergency Response Training
cGy	centiGray
CISD/CISM	Critical incident stress debriefing/management
CNS	Central nervous system
CPAP	Continuous positive airway pressure
CPR	Cardiopulmonary resuscitation
CR	Dibenzoxazepine
CRS	Cutaneous radiation syndrome
CS	2-chlorobenzylidene
CSF	Cerebrospinal fluid
DEOC	Director’s Emergency Operations Center (CDC)
DHHS	Department of Health and Human Services
DHS	Department of Homeland Security
DMAT	Disaster Medical Assistance Team
DMORT	Disaster Mortuary Assistance Team
DOD	Department of Defense
DOE	Department of Energy
DTPA	Diethylenetriaminepentaacetate
DU	Depleted uranium
ED	Emergency department
EEE	Eastern equine encephalitis
EMDR	Eye movement desensitization and reprocessing
EMS	Emergency medical services
EMT	Emergency medical technician
EPA	Environmental Protection Agency
ESF #8	Emergency services function #8
FBI	Federal Bureau of Investigation
FEMA	Federal Emergency Management Agency

FRP	Federal Response Plan
G-CSF	Granulocyte colony stimulating factor
GI	Gastrointestinal
GIS	Geographic information systems
G-M	Geiger-Mueller
GM-CSF	Granulocyte macrophage colony stimulating factor
Gy	Gray
HEICS	Hospital emergency incident command system
HIPAA	Health Insurance Portability and Accountability Act
HVA	Hazard vulnerability analysis
HVAC	Heating, ventilation, air conditioning
IAEA	International Atomic Energy Agency
ICS	Incident command system
IND	Improvised nuclear device
JCAHO	Joint Commission on Accreditation of Healthcare Organizations
KT	Kiloton
LET	Linear energy transfer
LRN	Laboratory Response Network
MCI	Mass casualty incident
MEN	Multiple endocrine neoplasia
MMST	Metro Medical Strike Team
MPERT	Mobile Pediatric Emergency Response Team
MSU	Management Support Unit
NCRP	National Council on Radiation Protection and Measurements
NDMS	National Disaster Medical System
NHTSA	National Highway Transportation Safety Administration
NIMS	National Incident Management System
NIOSH	National Institute for Occupational Safety and Health
NRC	Nuclear Regulatory Commission
OEM	Office of Emergency Management
OSHA	Occupational Safety and Health Administration
PBI	Primary blast injury
PCR	Polymerase chain reaction
PEPP	Pediatric Emergencies for Prehospital Professionals
PPE	Personal protective equipment
PPV	Positive-pressure ventilation
PSI	Pound(s) per square inch
PTA	Parent/teacher association
PTS	Pediatric trauma score
PTSD/S	Posttraumatic stress disorder/syndrome
Rad	Radiation absorbed dose
RADIAC	Radiation detection, indication, and computation
RBC	Red blood cell
RDD	Radiological dispersal device
REAC-TS	Radiation Emergency Action Center Training Site
REM	Roentgen equivalent man

RIA	Radioactive iodine ablation
RTG	Radioisotopic thermoelectric generators
SCIWORA	Spinal cord injury without radiographic abnormality
SEB	*Staphylococcus* enterotoxin B
SI	System International
SNS	Strategic National Stockpile
SSRI	Selective serotonin reuptake inhibitor
START	Simple Triage and Rapid Transport
Sv	Sievert
TLD	Thermoluminescent device
TMI	Three Mile Island
TNT	Trinitrotoluene
TPN	Total parenteral nutrition
TSA	Transportation Security Administration
TSH	Thyroid stimulating hormone
USPHS	United States Public Health Service
VEE	Venezuelan equine encephalitis
VHF	Viral hemorrhagic fever
VIG	Vaccinia immune globulin
WEE	Western equine encephalitis
WHO	World Health Organization
WMD	Weapons of mass destruction

Appendix A. Pediatric Terrorism and Disaster Preparedness: Learning Objectives

Chapter 1. Introduction

Goal: To prepare pediatricians to participate in pediatric disaster planning, taking into consideration the differences between children and adults.

Learning Objectives:

At the end of this chapter, the reader will be able to:

1. Describe the phases of terrorism and disaster planning.
2. Access the major Web sites available for further information on disaster preparedness to meet the needs of children.
3. Work in concert with response agencies when providing disaster relief.
4. Advocate for resources and products that currently do not exist for children, especially for children with special health care needs.
5. Identify children's anatomic, physiologic, immunologic, developmental, and psychological considerations that are different from adults and potentially could affect children's vulnerability to injury and response in a disaster.
6. Participate in community planning efforts that consider all potential aspects of a child's life.
7. Prepare for disasters at all phases of planning, response, recovery, and mitigation.

Chapter 2. Systems Issues

Goal: To prepare pediatricians to participate in pediatric disaster planning, by ensuring their familiarity with the U.S. disaster response system infrastructure.

Learning Objectives:

At the end of this chapter, the reader will be able to:

1. List the five major types of natural disasters.
2. Define and describe anthropogenic disasters.
3. Identify the lead federal agencies involved in planning, preparedness, emergency medical services (EMS), and other disaster response.
4. Access the Clinician Information Line run by the Centers for Disease Control Director's Emergency Operations Center.

Chapter 3. Responding to a Disaster

Goal: To prepare pediatricians to respond to a disaster.

At the end of this chapter, the reader will be able to:

1. Identify and describe the four phases of response to a disaster or act of terrorism (preparedness, actual response to the event, mitigation, and recovery).
2. Describe the function of the National Incident Management System and how it could be used as part of local emergency response plans.
3. Determine who would be in charge of an incident when there is a multi-agency response.
4. Describe the various types of EMS personnel and their function.
5. Identify problems that may be encountered by hospitals when dealing with a disaster or act of terrorism, including issues related to regional coordination, surge capacity, protection of personnel, and levels of precaution.
6. Describe potential problems with using personal protective equipment (PPE).
7. Describe the structure and function of an Incident Command System, including a Hospital Emergency Incident Command System, and its key participants.
8. Describe how hospitals coordinate their response to a disaster with other community and medical entities.
9. Provide a number of examples of the roles pediatricians may play in regional hospital to community planning.
10. Participate in the organization, implementation, and quality assurance activities related to disaster drills.
11. Assist in integrating children's services into disaster life support drills.
12. Refer children and families to existing support services in their communities.
13. Explain the new Joint Commission on Accreditation of Healthcare Organizations (JCAHO) disaster preparedness standards.
14. Explain the importance of appropriate triage.
15. Describe the major management functions of the Incident Command System (incident command, operations, planning, logistics, finance/administration).
16. Recognize the standardized position titles used for ICS supervisory personnel.
17. Describe the roles of smaller ICS functional units (staging, triage, treatment/decontamination, transport).

Chapter 4. Biological Terrorism

Goal: To prepare pediatricians to recognize signs and symptoms, diagnose, treat, perform infection control measures, and report biological terrorism agents and events.

Learning Objectives:

At the end of this chapter, the reader will be able to:

1. List which biological agents (microorganisms and toxins) pose the greatest potential for use in a bioterrorist attack, designated as “Category A” agents.
2. Explain the reasons why Category A agents have the greatest adverse public health, medical, and social impact if used as bioterrorist agents.
3. Recognize a wide number of clues that should heighten suspicion that a bioterrorist attack has occurred.
4. Describe how biological agents affect and manifest themselves in pediatric respiratory, nervous, gastrointestinal, and dermatologic systems.
5. Promptly diagnose and isolate a patient who has an illness potentially related to bioterrorism and notify the proper authorities.
6. Access and properly use PPE.
7. Describe the structure of his or her local public health system and the point of contact for reporting illnesses suspected of being related to bioterrorism.
8. Rapidly detect and isolate patients with an infectious illness related to bioterrorism to prevent transmission in health care settings.
9. Care for patients using standard precaution, contact precautions, droplet precautions, and airborne infection isolation techniques.
10. Access the major Web sites available for further information on isolation and environmental control guidelines.
11. Stock and use equipment and supplies necessary to diagnose and treat a patient suspected of being infected with a bioterrorist agent according to the level of care that will be provided at a particular facility or pediatrician’s office.
12. Create an emergency plan and train office staff to follow it.
13. Rapidly treat high-risk populations via administration of smallpox or anthrax vaccines.
14. Access the Strategic National Stockpile managed by the Department of Homeland Security and the Department of Health and Human Services.
15. Plan for surge capacity strains on existing pediatric resources.
16. Provide families with the information they need to plan and prepare for a disaster.
17. Provide clinical evaluation of patients suspected of being infected by Category A agents including all forms of anthrax, botulinum toxin, plague, smallpox, tularemia, and viral hemorrhagic fevers; as well as Category B and C Agents, including Ricin, Q Fever, *Staphylococcus* enterotoxin B, *Brucella*, B*urkholderia mallei* (Glanders), encephalitis viruses and yellow fever virus, and C*lostridium perfringens.*

Chapter 5. Chemical Terrorism

Goal: To prepare pediatricians to recognize signs and symptoms, diagnose, treat, perform infection control measures, and report chemical terrorism agents and events.

Learning Objectives:

At the end of this chapter, the reader will be able to:

1. Define chemical terrorism.
2. Explain how careful community planning, robust research and development (by academic, private, and governmental collaborative efforts), and rigorous medical education could mitigate a chemical terrorism catastrophe.
3. Explain why pediatricians need to understand the approach to mass casualty incidents involving traditional military chemical weapons and other toxic chemicals that might be used as "weapons of opportunity."
4. Explain the similarities and differences between a chemical terrorist attack and a more conventional chemical disaster in terms of the epidemiology and medical consequences associated with each.
5. Create a management plan for decontamination and initial care of small children by personnel wearing bulky PPE.
6. Recognize the risks and challenges to pediatric care providers posed by contact with many exposed children who are not critically injured but have been taken by parents to hospitals and pediatricians' offices without prior on-scene decontamination.
7. Describe how children have inherent physiologic, developmental, and psychological differences from adults that may enhance susceptibility and worsen prognosis after a chemical agent exposure.
8. Recognize the epidemiology of acute mass exposure to a toxin as an aid in recognizing a covert chemical attack with unknown agents.
9. Formulate an approach for managing chemical injuries sustained by children from an unknown chemical agent.
10. Describe clinical syndromes and management after exposure to various chemical agents (nerve agents, vesicants, pulmonary agents, cyanide, and riot-control agents).
11. Categorize chemical weapons based on the predominant symptoms they cause: neurologic (nerve agents or cyanide); respiratory (phosgene or chlorine, high-dose riot-control agents, or sulfur mustard with a delay of several hours from time of exposure); and mucocutaneous syndromes (vesicants).
12. Access and alert the CDC, local public health authorities, and/or regional poison control center.
13. Differentiate between cyanide and nerve agent attacks.
14. Plan for the initial protection of everyone in a community exposed to a hazardous chemical using CDC Guidelines for Evacuation and for Sheltering in Place in a Chemical Emergency.
15. Discuss treatment of contaminated victims via extrication, triage, resuscitation as needed, and decontamination performed by rescue workers or health care providers wearing appropriate PPE.
16. Provide cardiopulmonary and airway support, including endotracheal intubation, and emergent intramuscular antidotal therapy as necessary and appropriate for the specific exposure.
17. Prepare to treat potential victims of a terrorist attack involving industrial sources of hazardous chemicals.
18. Participate in disaster management training, including stocking appropriate antidotes, practicing decontamination strategies, and learning the use of PPE.

19. Identify chemical terrorism toxins such as nerve agents (tabun, sarin, soman, and VX ("Venom X") and their delivery systems, toxicology and clinical manifestations, central and autonomic systems effects, neuromuscular effects, diagnostic tests, treatment, isolation, and control measures.
20. Describe comprehensive plans to address terrorist threats to infection control (for biological agents), epidemiology, public affairs, and administration of health care facilities.
21. Explain the lethality of cyanide, its toxicology, clinical presentation, and treatment.
22. List the agents known as vesicants (sulfur mustard, the nitrogen mustards, lewisite, and phosgene oxime) and their characteristics, clinical effects, treatment, and special pediatric considerations.
23. Describe pulmonary agents (chlorine and phosgene) and their characteristics, clinical effects, treatment, including decontamination and management.
24. Identify riot control agents (CS [2-chlorobenzylidene], CN [1-chloroacetophenone, Mace®], and pepper spray [*Oleoresin capsicum*]), their transmission and pathogenesis, clinical manifestations, diagnosis, treatment, and control.

Chapter 6. Radiological and Nuclear Terrorism

Goal: To prepare pediatricians to recognize the signs and symptoms associated with radiologic and nuclear exposure and to diagnose, treat, and report nuclear and radiologic terrorism events and agents.

Learning Objectives:

At the end of this chapter, the reader will be able to:

1. List sources of radioactivity and the terrorism threats they pose.
2. Describe the scope, implications, and incident management of radiological threats.
3. Differentiate between nuclear and conventional explosions.
4. Explain the process of radiation from nuclear detonation and fallout.
5. Describe the purpose and effects of radiological dispersal devices ("dirty bombs").
6. Describe medical and industrial sources of radiation.
7. Define the potential hazards of destruction or sabotage of a nuclear power plant.
8. Recount the historical overview of radiation injury.
9. Explain the physical principles of ionizing radiation.
10. Describe radiation interactions including electron excitation, ionization, nuclear excitation, nuclear fission, activation, and secondary radiation.
11. Provide information to patients concerning the safety of consuming milk after a reactor accident.
12. List natural and man-made types of radiation exposure.

13. Record radiation-induced signs, symptoms, and erythema during the course of medical management for radiation casualties to help triage and guide medical management of casualties.
14. Recognize the pattern of biological responses associated with acute radiation sickness (ARS).
15. Describe the pathophysiology and effects of radio- and chemotherapy including bone marrow damage, diarrhea, and hair loss.
16. Explain the time-dependent clinical stages of radiation exposure (prodrome, latent period and manifest illness).
17. List the diagnostic steps to assess a dose of radiation exposure.
18. Medically diagnose external radiological contamination.
19. Assess and care for externally contaminated patients.
20. Properly use PPE to eliminate personal risk while caring for victims of radiation exposure.
21. Explain the mechanisms of internal contamination when radioactive material enters the bodies of unprotected people through inhalation, ingestion, or wound contamination.
22. Differentiate between public concern about terrorism involving radioactive materials and medical errors during diagnosis that have been the most common cause of significant internal contamination.
23. Describe the three pathways through which radioactive material can enter the body (inhalation, ingestion and skin penetration).
24. List the various instruments available for detecting and measuring radiation.
25. Discuss the need for PPE, type needed for radiation contamination, disposal of PPE, and Department of Energy Guidance on PPE.
26. Differentiate between PPE for radioactive isotopes and chemical or biological exposures.
27. Describe emergency precautions when PPE is not available in a radioactive environment.
28. Explain the basic principles of decontamination and priorities.
29. Explain the characteristics that render pediatric patients uniquely sensitive to the effects of radiation exposure.
30. Explain the well-documented long-term effects of radiation exposure to the fetus and child.
31. Recount the psychological vulnerability of children involved in a radiation related incident.
32. Treat acute pediatric radiation syndrome both supportively and with specific actions and medications.
33. Prioritize external decontamination before treatment of internal decontamination.
34. Use potassium iodide (KI) to reduce thyroid uptake of radioactive iodine.
35. Keep available and use a table of threshold radioactive exposures and recommended prophylactic single doses of KI.
36. Treat internal contamination with appropriate chelating agents.
37. Prioritize steps in the treatment of victims of combined injuries, that is, stabilization of life threatening injuries prior to radiation injuries.
38. Manage the patient with embedded radioactive material and depleted uranium.

39. Provide followup care, being mindful of the risk of carcinogenesis.
40. Describe environmental issues affecting children after a terrorist incident involving radioactive materials.
41. Make knowledgeable decisions and actions to prevent radiation exposure related to short-term evacuation versus sheltering.
42. Access the FDA guidance for accidental radioactive contamination of human food and animal feed.

Chapter 7. Blast Terrorism

Goal: To prepare pediatricians to recognize signs and symptoms, diagnose, treat, and report blast terrorism events and agents.

Learning Objectives:

At the end of this chapter, the reader will be able to:

1. Explain the four primary mechanisms involved in blast injuries.
2. List the three primary causes of death after a blast.
3. Describe four characteristic results of urban bomb blasts.
4. Explain how the anatomic structure of the lung makes it susceptible to the effects of blast barotraumas.
5. List typical clinical findings in blast lung injuries.
6. Treat blast lung injury and resulting complications.
7. List the signs and symptoms that suggest arterial air embolization.
8. Treat arterial air emboli.
9. List the clinical findings in GI injury from a blast.
10. Manage gastrointestinal blast injuries by controlling internal bleeding and identifying and repairing any perforated viscus.
11. Describe the clinical findings in blast auditory injuries.
12. Explain the cardiovascular effects of a blast wave.
13. Recognize sentinel injuries (subtle injuries that can increase the risk of developing serious blast injury).
14. Provide emergency management of victims of incendiary devices.
15. Describe a trauma center.
16. Describe the key organizational characteristics common among all trauma centers.
17. Delineate the services provided by the three levels of trauma centers.
18. Access the *Advanced Trauma Life Support® for Doctors Course* protocols.
19. Access the *Advanced Burn Life Support® Course* protocols.
20. Define a mass casualty event.
21. Participate in planning for and mitigation of an attack involving blast terrorism.
22. Recognize the need for proper triage and transport of pediatric patients to an appropriate trauma center.

Chapter 8. Mental Health Issues

Goal: To prepare pediatricians to address the mental health needs of children and families in a disaster or terrorist event.

Learning Objectives:

At the end of this chapter, the reader will be able to:

1. Recognize pediatric psychiatric symptoms and disorders, including posttraumatic stress disorder (PTSD), anxiety, depression, and behavioral problems, after exposure to disasters or terrorist incidents.
2. List the risk factors that are associated with an increased risk of posttraumatic symptoms.
3. Describe three characteristic symptoms that are essential features of PTSD after exposure to a traumatic event.
4. List five factors that are likely to be present in death circumstances that are considered "traumatic deaths."
5. Assess, screen and treat (or refer for treatment), children who are at risk for mental health problems after exposure to a traumatic event.
6. Follow procedures for notification about an unexpected death with sensitivity to key issues that will minimize the short- and long-term impact on survivors.
7. Explain death to children in a manner they can understand.
8. Recognize common reactions that children may have in response to a personal loss.
9. Identify children who have extreme reactions, atypical reactions, or prolonged reactions that require referral to a mental health professional experienced in the management of pediatric bereavement.
10. Explain the controversy that surrounds the use of psychopharmacology in children with PTSD.
11. Advocate for, consult with, and actively participate in school crisis response teams.
12. Prepare families to cope with anniversary reactions and commemorative activities of a traumatic event.
13. Recognize the emotional impact experienced by school staff, first responders, and other health care professionals following a disaster.
14. Communicate effectively during a crisis.

Chapter 9. Integrating Terrorism and Disaster Preparedness into Your Pediatric Practice

Goal: To assist general pediatricians in integrating the information in this resource into their office- or hospital-based practices.

Learning Objectives:
At the end of this chapter, the reader will be able to:

1. Explain their role in the general system response to disasters.
2. Create a disaster-preparedness framework for a general practice pediatric office.
3. Describe the components for basic internal office readiness (facilities, equipment, records, communication system, chain of command, contact list, staff responsibilities, triage, screening, prioritization, practice readiness, staff development, and insurance).
4. Determine how the practice will link and coordinate efforts with affiliate hospitals, schools, daycare centers, local response teams, the local department of health, and city, State, and Federal efforts.
5. Develop a list of contact numbers for all existing agencies that are components in the external disaster management system.
6. Establish relationships with the local or regional Office of Emergency Management and strengthen already established relationships with organizations/institutions in the community.
7. Integrate into the larger community response effort.
8. Participate in disaster drills and exercises.
9. Describe disaster protocols of local departments of health.
10. Communicate directly with children and families about disaster and terrorism preparedness.
11. Answer the questions of children and families during events.
12. Provide advice for families of children with special health care needs.
13. Participate in creating contingency plans to increase hospital surge capacity.
14. Help to produce contingency plans for acquiring and maintaining essential services (water, electricity, oxygen, garbage disposal).
15. Plan for stockpiling medical supplies.
16. Participate in a network of community resources that will be able to organize volunteers to become proxy caretakers for orphaned children.
17. Participate in planning hospital infrastructure needs for children (disaster drills involving children, infection control plans, quarantine procedures, staff training).

Chapter 10. Working with Government Agencies to Enhance Preparedness

Goal: To assist pediatricians working with government agencies to enhance preparedness for meeting children's needs.

Learning Objectives:
At the end of this chapter, the reader will be able to:

1. Describe the role of various government agencies in disaster preparedness and response.

2. Work with community, State, and Federal Government entities in disaster preparedness for children.
3. Explain the importance of the pediatrician's role in determining public health preparedness.
4. Advocate for children and families in preparedness planning.

Chapter 11. Conclusion

Goal: To assist pediatricians in understanding the consequences of a recent major disaster and applying the lessons learned to current planning and preparedness efforts.

Learning Objectives:

At the end of this chapter, the reader will be able to:

1. Describe the problems that emerged following Hurricane Katrina and how these experiences can inform future planning efforts.
2. Ensure that preparedness plans include provisions for identification and care of displaced children who have been separated from their families.
3. Understand the needs of children and families in sheltering situations.
4. Recognize the importance of and maintain up-to-date professional and personal contact information for themselves and their families and staff members.
5. Participate and assist in coordination of disaster relief efforts, both locally and farther away, depending on areas of greatest need.

Appendix B. List of Contributors[1]

Lead Editors

George L. Foltin, MD, FAAP, FACEP
Director, Center for Pediatric Emergency Medicine
Director, Division of Pediatric Emergency Medicine
Associate Professor of Pediatrics and Emergency Medicine
New York University School of Medicine
Bellevue Hospital Center, New York, NY
System Issues; Blast Trauma; Integrating Preparedness Into Your Practice
Working With Government Agencies and Advocacy
Conclusion/Lessons Learned—First Impressions

David J. Schonfeld, MD, FAAP
Thelma and Jack Rubinstein Professor of Pediatrics
Director, Division of Developmental and Behavioral Pediatrics
Cincinnati Children's Hospital Medical Center, Cincinnati, OH
Professor Adjunct of Pediatrics
Yale University School of Medicine, New Haven, CT
Children Are Not Small Adults; Mental Health Issues

Michael W. Shannon, MD, MPH, FAAP
Chief and CHB Chair
Division of Emergency Medicine
Director, The Center for Biopreparedness
Children's Hospital Boston
Professor of Pediatrics
Harvard Medical School, Boston, MA
Biological Terrorism; Chemical Terrorism; Nuclear and Radiological Terrorism

Children Are Not Small Adults

Section Editor and Contributor

Jane Knapp, MD, FAAP
Co-Director, Center for Childhood Safety
Professor of Pediatrics
Clinical Scholar in Pediatric Emergency Medicine
Children's Mercy Hospital
University of Missouri-Kansas City School of Medicine, Kansas City, MO

[1] Note: For all contributors who are affiliated with the U.S. Army, the views, opinions and assertions contained in this resource are those of the authors and should not be construed as official Department of the Army policy.

Systems Issues

Section Editors

Marion J. Balsam, MD, FAAP
Executive Secretary of the Federal Advisory Committee
Research Partnerships Program Director for the National Children's Study
National Institute of Child Health and Human Development
National Institutes of Health, Bethesda, MD

Lorraine M. Giordano, MD, FACEP
Medical Director
NYC Office of Emergency Management, New York, NY
Program Director
Medical Oversight and Quality Assurance
NYC Health & Hospitals Corporation, New York, NY

Contributors

Steven Baldwin, MD, FAAP
Associate Professor of Pediatrics and Emergency Medicine
Children's Hospital of the University of Alabama, Birmingham, AL

Marie Diglio, EMT-P
Executive Director, Operations
Regional Emergency Medical Services Council**,** New York, NY

Jessica C. Foltin, MD
Director, Pediatric Emergency and Transport Services
Associate Professor of Pediatrics and Emergency Medicine
New York University School of Medicine
New York University Medical Center, New York, NY

Katherine Gnauck, MD
Division of Pediatric Emergency Medicine
Department of Emergency Medicine
University of New Mexico Health Science Center
Albuquerque, NM

Robert Iannarelli
Chief (retired)
NYC Fire Department, New York, NY

Francesca C. Music
Director, Homeland Security and National Medical Contingency Planning
Office of the Assistant Secretary of Defense for Health Affairs
Force Health Protection & Readiness
Office of the Assistant Secretary of Defense for Homeland Defense
Department of Defense, Washington, DC

James Tsung, MD, FAAP
Attending Physician
Division of Pediatric Emergency Medicine
Departments of Pediatrics and Emergency Medicine
Bellevue Hospital Center and New York University Medical Center
Assistant Professor of Pediatrics and Emergency Medicine
New York University School of Medicine

Biological Terrorism

Section Editor

Julia McMillan, MD
Vice Chair for Pediatric Education
Professor of Pediatrics
Residency Program Director
Department of Pediatrics
Johns Hopkins University School of Medicine, Baltimore, MD
Editor-in-Chief, *Contemporary Pediatrics*

Contributors

Joanne Cono, MD, ScM, FAAP
Assistant Associate Director for Science
Coordinating Office of Terrorism Preparedness and Emergency Response
Medical Epidemiologist, Bioterrorism Preparedness and Response Program
National Center for Infectious Diseases
Centers for Disease Control and Prevention, Atlanta, GA

Hanoch A. Patt, MD
Fellow in Pediatric Cardiology
Department of Pediatrics
Baylor College of Medicine, Texas Children's Hospital, Houston, TX

Chemical Terrorism

Section Editor

Frederick Henretig, MD, FACMT, FAAP
Emergency Medicine Attending Physician
Director, Section of Clinical Toxicology
Senior Toxicologist and Associate Medical Director, Poison Control Center
Children’s Hospital of Philadelphia
University of Pennsylvania Medical Center, Philadelphia, PA

Contributors

Thomas R. Burklow, MD
Lieutenant Colonel, Medical Corps, United States Army
Chief, Department of Pediatrics
Walter Reed Army Medical Center, Washington, DC
Uniformed Services University of the Health Sciences, Bethesda, MD

James M. Madsen, MD, MPH
Colonel, Medical Corps, United States Army
Former Deputy Director, National Capital Consortium Residency in Occupational and Environmental Medicine
Former Assistant Professor of Preventive Medicine and Biometrics, of Military and Emergency Medicine, of Pathology, and of Emerging Infectious Diseases
Uniformed Services University of the Health Sciences, Bethesda, MD

Michele McKee, MD
Department of Pediatrics and Toxicology
University of Pennsylvania Medical Center, Philadelphia, PA

Joshua S. Rotenberg, MD, MS, FAAP
Major, Medical Corps, United States Army
Founding member of *The Working Group on Chemical and Biological Terrorism*
Departments of Child and Adolescent Neurology and Pediatrics
Walter Reed Army Medical Center, Washington, DC
Uniformed Services University of the Health Sciences, Bethesda, MD

James F. Wiley II, MD
Associate Professor of Pediatrics and Emergency Medicine/Traumatology
Head, Division of Pediatric Emergency Medicine
University of Connecticut School of Medicine
Medical Consultant, Connecticut Regional Poison Control Center
Connecticut Children's Medical Center, Hartford, CT

Clifton E. Yu, MD
Lieutenant Colonel, Medical Corps, United States Army
Assistant Professor of Pediatrics
Director, National Capital Consortium Residency in Pediatrics
Uniformed Services University of the Health Sciences, Bethesda, MD

Nuclear and Radiological Terrorism

Section Editor

David E. Jarrett, MD, FS, FACEP
Colonel, Medical Corps, United States Army
Director, Armed Forces Radiobiology Research Institute
US Army Medical Research Institute of Infectious Diseases, Bethesda, MD

Contributors

William F. Blakely, PhD
Head of Radiation Biology
Biological Dosimetry Team Leader
Armed Forces Radiobiology Research Institute, Bethesda, MD
Adjunct Assistant Professor of Preventive Medicine/Biometrics
Adjunct Assistant Professor of Environmental and Occupational Health
Uniformed Services University of the Health Sciences, Bethesda, MD

Gerald S. Braley, PhD
Major, United States Air Force
Science Advisor, Office of Emergency Response
National Nuclear Security Administration, Department of Energy, Germantown, MD

Itzhak Brook, MD
Professor of Pediatrics
Georgetown University School of Medicine, Washington DC

Curtis Cummings, MD, MPH
Associate Professor of Environmental and Occupational Health
Drexel University School of Public Health, Philadelphia, PA

William E. Dickerson, MD
Colonel, Medical Corps, United States Air Force
Director of Military Medical Operations
Staff Radiological Oncologist
Armed Forces Radiobiology Research Institute, Bethesda, MD

Gary Francis, MD, PhD
Professor of Pediatrics and Pediatric Endocrinology
Director of Pediatric Endocrinology Fellowship Program
Uniformed Services University of Health Sciences, Bethesda, MD

Sarah K. Kirtland, MS
Commander, United States Navy
Deputy Director of Military Medical Operations
Armed Forces Radiobiology Research Institute, Bethesda, MD

Philip L. Liotta, PhD
Former Chief Health Physicist, Former Chairman
Radiation Sciences Department
Armed Forces Radiobiology Research Institute

Robert W. Miller, MD, DrPH
Pediatric Epidemiologist, Clinical Epidemiology Branch
National Cancer Institute, Bethesda, MD

Pataje Prasanna, PhD
Radiation Biologist
Armed Forces Radiobiology Research Institute, Bethesda, MD

Richard Sedlak, MD, MPH
Colonel, United States Army
Director, Military Medical Operation
Armed Forces Radiobiology Research Institute, Bethesda, MD

Horace Tsu, MD
Lieutenant Colonel, Medical Corps, United States Air Force
Former Deputy Head, Military Medical Operations
Armed Forces Radiobiology Research Institute, Bethesda, MD

Clifton E. Yu, MD
Lieutenant Colonel, Medical Corps, United States Army
Assistant Professor of Pediatrics
Director, National Capital Consortium Residency in Pediatrics
Uniformed Services University of the Health Sciences, Bethesda, MD

Acknowledgments

Donna Solyan
Head of Illustration and Editing Section
Armed Forces Radiobiology Research Institute, Bethesda, MD

Nimae Angwafo, MD, AFRRI
LCDR Charles R. Woodruff, AFRRI
Jamie Waselenko, MD, AFRRI

Blast Trauma

Section Editor

Arthur Cooper, MD, MS, FACS, FAAP, FCCM
Director of Trauma and Pediatric Surgical Services
Columbia University Affiliation
Harlem Hospital Center, New York, NY

Contributors

Julie Quintero, MD
Attending Physician
Assistant Professor of Pediatrics and Emergency Medicine
Robert Wood Johnson University Hospital, New Brunswick, NJ
Former Fellow in Pediatric Emergency Medicine
Bellevue Hospital Center, New York, NY

Acknowledgments

Sherlita Amler, MD
Yaron Bar-Dayan, MD
Susan M. Briggs, MD
Lt. Gen. Paul K. Carlton, Jr., MC, USAF (Ret)
T. Forcht Dagi, MD
Timothy E. Davis, MD, MPH
Yoel Donchin, MD
Zvi Feigenberg, MD
Eric R. Frykberg, MD
Pinchas Halpern, MD
Richard D. Hunt, MD
Catherine Y. Lee, MPH

Kobi Peleg, PhD
Michael Stein, MD
John A. Weigelt, MD

Mental Health Issues

Section Editor

David Szydlo, MD, PhD
Education and Curriculum Development Director
National Center for Children Exposed to Violence
Associate Research Scientist, Child Study Center
Yale University School of Medicine, New Haven, CT
Currently Director, Centro de Estudios Psicoanaliticos A.C., Mexico

Contributors

Steven Berkowitz, MD, FAAOS, FACS, CIME
Medical Director
National Center for Children Exposed to Violence, Child Study Center
Yale University School of Medicine, New Haven, CT
Associate Director, Connecticut Trauma Response Program, New Haven, CT

Russell T. Jones, PhD
Terrorism Disaster Branch
National Center for Child Traumatic Stress, Blacksburg, VA
Professor of Clinical Psychology
Virginia Tech University, Blacksburg, VA

Betty Pfefferbaum MD, JD
Director and Professor of Psychiatry and Behavioral Sciences
Department of Psychiatry and Behavioral Sciences
University of Oklahoma Health Sciences Center, Oklahoma City, OK

Acknowledgment

Steven Marans, PhD
Director, National Center for Children Exposed to Violence, Child Study Center
Harris Associate Professor of Child Psychoanalysis
Associate Professor of Psychiatry
Yale University School of Medicine, New Haven, CT

Integrating Preparedness into Your Pediatric Practice

Section Editors

David Markenson, MD, FAAP
Director, Center for Disaster Medicine
School of Public Health, New York Medical College
Chief, Pediatric Emergency Medicine
Maria Fareri Children's Hospital
Westchester Medical Center, New York, NY

Irwin Redlener, MD, FAAP
President and Co-Founder
Children's Health Fund, New York, NY
Director, National Center for Disaster Preparedness
Associate Dean for Public Health Advocacy and Preparedness
Mailman School of Public Health
Columbia University, New York, NY

Contributor

Danielle Laraque, MD, FAAP
Debra and Leon Black Professor of Pediatrics
Chief, Division of General Pediatrics
Mount Sinai School of Medicine
Mount Sinai Medical Center, New York, NY

Working with Government Agencies and Advocacy

Section Editors and Contributors

David Markenson, MD, FAAP

Irwin Redlener, MD, FAAP

Conclusion and Lessons Learned

Section Editor

George L. Foltin, MD, FAAP, FACEP

Contributors

Steven Baldwin, MD, FAAP

Dan Kavanaugh, MSW
Director, Emergency Medical Services for Children Program
Health Resources Services Administration
Maternal & Child Health Bureau, Rockville, MD

Errol McRae, RN, MN
Executive Director, Louisiana Chapter–ACEP
Baton Rouge, LA

Gretchen Mahan
AAP Chapter Executive Director
Mississippi Chapter–AAP, Jackson, MS

Keith E. Parker, RN
Director, Bureau of Emergency Medical Services
Mississippi Department of Health, Jackson, MS

William D. Payne, MD, FAAP
President, Mississippi Chapter–AAP, Jackson, MS

Lou E. Romig MD, FAAP, FACEP
Children's Hospital Medical Director
Pediatric Emergency Medicine, Miami, FL
FL-5 Disaster Medical Assistance Team, FEMA/Department of Homeland Security

David J. Schonfeld, MD, FAAP

Michael Shannon, MD, MPH

Paul E. Sirbaugh, DO, FAAP
Director, Prehospital Medicine
Texas Children's Hospital
Assistant Professor of Pediatrics
Baylor College of Medicine, Houston, TX

Linda Hill Southward, PhD, ACSW
Research Professor
Coordinator, Family and Children Research Unit
Social Science Research Center
Mississippi State University, Mississippi State, MS

Michael Tunik, MD, FAAP
Associate Professor of Pediatrics and Emergency Medicine
NYU School of Medicine
Research Director, Center for Pediatric Emergency Medicine
Bellevue Hospital Center, New York, NY

Other Contributors

Deborah Levine, MD
Clinical Assistant Professor of Pediatrics and Emergency Medicine
New York University School of Medicine
Bellevue Hospital Center, New York, NY

Virginia M. Ferrante, MA
Medical Illustrator
Ferrante Medical Media, Darien, CT

Reviewers

Errol Alden, MD, FAAP
Executive Director, American Academy of Pediatrics

Steven Baldwin, MD, FAAP

Jay Berkelhammer, MD, FAAP
President Elect, American Academy of Pediatrics

Carol Berkowitz, MD, FAAP
Immediate Past President, American Academy of Pediatrics

Carden Johnston, MD, FAAP, FACEP, FRCP
Former President, American Academy of Pediatrics
Emeritus Professor of Pediatrics
Children's Hospital of the University of Alabama, Birmingham, AL

Kathryn Nichol, MD, FAAP
Board of Directors, American Academy of Pediatrics

Eileen Ouellette, JD, MD, FAAP
President, American Academy of Pediatrics

Committee on Pediatric Emergency Medicine

Steven E. Krug, MD, FAAP
Committee Chairman, 2004-2008
Children's Memorial Hospital, Chicago

Thomas Bojko, MD, MS, FAAP
Robert Wood Johnson Medical School

Margaret A. Dolan, MD, FAAP

Karen S. Frush MD, FAAP
Duke University Medical Center

Joan E. Shook, MD, MBA
Baylor College of Medicine

Robert E. Sapien, MD, FAAP
University of New Mexico Health Sciences Center

Kathy Shaw, MD, MSCE, FAAP
The Children's Hospital of Philadelphia

Paul Sirbaugh DO, FAAP
Texas Children's Hospital

Loren Yamamoto, MD, MPH, MBA
University of Hawaii Hospital

Liaisons

American College of Emergency Physicians
Sharon Mace, MD, FAAP, FACEP

American College of Surgeons
David W. Tuggle, MD, FAAP

EMSC National Resource Center
Jane Ball, RN, DrPH
Susan Eads Role, JD, MSLS

Maternal & Child Health Bureau
Dan Kavanaugh, MSW

National Association of EMS Physicians
Kathleen Brown, MD, FAAP

American Academy of Family Physicians
Kim Bullock, MD

American Academy of Pediatrics

Project Manager

Mary McGowan, MA

Staff

Michael Glasstetter, JD
Graham Newson, MA
Cindy Pellegrini
Susan Tellez