Types of Disasters

5

Paul N. Severin and Phillip A. Jacobson

5.1 Unique Vulnerabilities of Children

Although pediatric patients are viewed as a unique population, they are the population: it has been estimated that more than 25% of the U.S. population fits within the pediatric age range. In all actuality, it is more appropriate to consider the pediatric patient as one with unique vulnerabilities. These vulnerabilities, especially with respect to disasters, are based on the following pediatric developmental differences: anatomy and physiology; behavior and development; and psychology and mental health (Hilmas et al. 2008; Jacobson and Severin 2012; Severin 2011).

5.1.1 Anatomy and Physiology

As depicted on pediatric burn assessment charts, the head accounts for a majority of the total body surface area, while the legs are much less. With growth, the head decreases in size to adult parameters by adolescence. The larger body surface area-to-mass ratio increases the risk of hypothermia. The decreased ability to shiver is another disadvantage to the pediatric patient as heat can be rapidly lost. Hypothermia can be a deadly combination with any trauma leading to coagulopathy and uncontrollable hemorrhage. When exposed to various toxins, the larger body surface area enhances the amount of absorption and end-organ toxicity. Their normally thin, delicate skin can add to absorption, especially in the presence of abrasions or burns. Orthopedic injuries are common in the pediatric patient due to pliability of the

P. N. Severin (✉) · P. A. Jacobson
Pediatric Critical Care Medicine, Rush Medical College and John H. Stroger, Jr. Hospital of Cook County, Chicago, IL, USA
e-mail: Paul_N_Severin@rush.edu; pseverin@cookcountyhhs.org; pjacobson@cookcountyhhs.org

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skeleton as a result of incomplete calcification and active bone growth centers. Protected organs, such as the lungs and heart, may be injured due to overlying fractures. Cervical spine injuries can also be pronounced, as in patients with head trauma. In fact, spinal cord injury may be present without any radiographic abnormalities of the spine. Finally, vital signs will vary based on the pediatric patient's age. This may be a pitfall during rapid evaluation by any nurse or HCP not accustomed to the care of children. Younger pediatric patients have higher metabolic rates and, therefore, higher respiratory rates and heart rates. This can be a distinct disadvantage versus older pediatric patients when encountering similar diseases. An example is inhaled toxins (e.g., nerve agents and lung-damaging agents). Infants and children will suffer greater toxicity since they inhale at a faster rate due to higher metabolic demands and thus, distribute the toxin more rapidly to various end-organs.

Understanding respiratory differences is essential to the management of the acutely ill pediatric patient. The most common etiology for cardiorespiratory arrest in children is respiratory pathology, typically of the upper airway. Most of the airway resistance in children occurs in the upper airway. Nasal obstruction can lead to severe respiratory distress due to infants being obligate nose breathers. Their relatively large tongue and small mouth can lead to airway obstruction quickly, especially when the neuromuscular tone is abnormal such as during sedation or encephalopathy. In infants, physiologic (i.e., copious secretions) and pathologic (i.e., edema, vomitus, blood, and foreign body) factors will exaggerate this obstruction. Securing the airway in such events can be quite challenging. Typically, the glottis is located more anterior and cephalad. Appropriate visualization during laryngoscopy can be further hampered by the prominent occiput that causes neck flexion and, therefore, reduces the alignment of visual axes. The omega or horseshoe-shaped epiglottis in young infants and children is quite susceptible to inflammation and swelling. As in epiglottitis, the glottis becomes strangulated in a circumferential manner leading to dangerous supraglottic obstruction. Children also have a natural tendency to laryngospasm and bronchospasm. Finally, due to weaker cartilage in infants, dynamic airway collapse can occur especially in states of increased resistance and high expiratory flow. Along with altered pulmonary compensation and compliance, a child may rapidly progress to respiratory failure and possibly arrest.

Cardiovascular differences are critical in the pediatric patient. Typical physiological responses tend to allow compensation with seemingly normal homeostasis. With tachycardia and elevated systemic vascular resistance, younger pediatric patients can maintain normal blood pressure despite decreased cardiac output and poor perfusion (compensated shock). Since children have less blood and volume reserve, they progress to this state quickly. In pediatric patients with multiorgan injury or severe gastrointestinal losses, these compensatory mechanisms are pushed to their limits. The unaccustomed HCP may be lulled into complacency since the blood pressure is normal. All the while, the pediatric patient's organs are being poorly perfused. Once these compensatory mechanisms are exhausted, the patient rapidly progresses to hypotension and, therefore, hypotensive shock. If not reversed expeditiously, this may lead to irreversible shock, ischemia, multiorgan dysfunction, and death.

Pediatric patients with altered mental status pose significant problems. The differential diagnosis will be very broad in the comatose patient based on development alone. For example, younger pediatric patients can present with nonconvulsive status epilepticus (NCSE) instead of generalized convulsive status epilepticus (GCSE). In fact, NCSE is more common among younger pediatric patients than GCSE, especially in those from 1 to 12 months of age. Furthermore, many of them are previously well without preexisting diseases such as epilepsy. Other disease states may include poisoning, inborn errors of metabolism, meningitis, and other etiologies of encephalopathy. Using the modified pediatric Glasgow Coma Scale (GCS) is the cornerstone when evaluating the young pediatric patient when they are preverbal. Pupillary response, external ocular movements, and gross motor response may be challenging to evaluate in a developmentally young or delayed pediatric patient.

Pediatric traumatic brain injury is extremely devastating. Whether considered accidental (motor vehicle crash) or nonaccidental (abusive head trauma), evaluation of the neurological status of the acutely injured pediatric patient can be problematic, especially the GCS. Some prefer to use the AVPU system (Alert, Responds to Verbal, Responds to Pain, and Unresponsive). Due to the disproportionately larger head and weaker neck muscles, there is more risk of acceleration–deceleration injuries (fall from a significant height, vehicular ejection, and abusive head trauma). Furthermore, the softer skull, dural structural differences, and vessel supply will place the pediatric patient at risk for brain injury and intracranial hemorrhage. Finally, due to pediatric brain composition, the risk of diffuse axonal injury and cerebral edema is much higher.

Although spinal cord injury is rare in young pediatric patients, morbidity and mortality are significant. In pediatric patients less than 9 years of age, the most commonly seen injuries are in the atlas, axis, and upper cervical vertebrae. In young pediatric patients, spinal injuries tend to be anatomically higher (cervical) versus adolescents (thoracolumbar). Furthermore, congenital abnormalities, such as atlantoaxial abnormalities (Trisomy 21), may exaggerate the process. The clinical presentation of spinal cord injury varies in young pediatric patients due to ongoing development. Laxity of ligaments, wedge-shaped vertebrae, and incomplete ossification centers contribute to specific patterns of injuries. Finally, Spinal Cord Injury without Radiographic Abnormality (SCIWORA) may result. Because of the disproportionately larger head, weaker neck muscles, and elasticity of the spine, significant distraction and flexion injury of the spinal cord may occur without apparent ligament or bony disruption (Hilmas et al. 2008; Jacobson and Severin 2012; Severin 2011).

5.1.2 Behavior and Development

Motor skills develop from birth. Gross and fine motor milestones are achieved in a predictable manner and must be assessed during each HCP encounter. Cognitive development will follow a similar pattern of maturation. The development of these skills can often predict injuries and their extent. For example, consider a house fire. A young infant, preschooler, and adolescent are sleeping upstairs in house when a fire breaks out in the middle of the night. The smoke detectors begin to alarm. Each

child is awoken by the ensuing noise and chaos. Based on the development, the adolescent will most likely make it out of the house alive. He will comprehend the threat, run down the stairs, and exit the house without delay. Smoke inhalation may be minimal. If it is a middle adolescent, an attempt may be made to jump out of the window leading to multiple blunt trauma with or without traumatic brain injury. The preschooler most likely will be too scared and not understand how to escape. Tragically, he may hide under a bed or in a closet. When the firefighters arrive and search the house, the preschooler may remain silent because of fear, especially of strangers in the house. He will most likely succumb to thermal injuries along with the effects of carbon monoxide and die. As far as the infant, he cannot walk, climb, crawl, or run. Furthermore, he cannot scream for help or know how to escape. As the smoke engulfs the room, he will most likely suffer severe smoke inhalation injury including extensive carbon monoxide toxicity along with thermal injuries and die. This example also points out another important difference in pediatric patients: their dependence on caregivers. When considering neonates, for example, their entire existence depends on the caregiver, including feeding, changing of diapers, nurturing, and environmental safety. These dynamics are essential to the pediatric patient's health and survival, especially during a disaster.

Another aspect of development is the attainment of language skills. This, too, develops over time in a predictable fashion. One of the biggest challenges in pediatrics is the lack of the patient's ability to verbally convey complaints. As described above, verbal milestones vary among the different age ranges of the pediatric patient. HCPs are often faced with a caregiver's subjective assessment of the problem. Although it can be revealing and informative, this may not be available in an acute crisis situation. It will take the astute HCP to determine, for example, if an inconsolably crying infant is in pain from a corneal abrasion or something more life-threatening such as meningitis. This can also be a challenging task in a teenager, especially during middle adolescence. An HCP will have to determine, for example, if the seemingly lethargic middle adolescent is intoxicated with illicit drugs or has diabetic ketoacidosis.

Finally, the HCP will have to address developmental variances among their pediatric patients and any comorbid features. Young pediatric patients can regress developmentally during any illness or injury. This is especially seen in patients with chronic medical conditions (cancer) or during prolonged hospitalization with rehabilitation (multisystem trauma). Furthermore, those pediatric patients with developmental and intellectual disabilities, for example, will be difficult to evaluate based on the effects of their underlying pathology. These pediatric patients typically have unique variances in their physical exams (Jacobson and Severin 2012; Severin 2011). Please refer to Chap. 7 for more detailed information on pediatric development.

5.1.3 Psychology and Mental Health

Pediatric patients will often reflect the emotional state of their caregiver. They take verbal and physical cues from their caregiver. At times, this may also occur in the presence of a nurse or HCP. The psychological impact of illness will vary greatly

with the child's development and experience. Children tend to have a greater vulnerability to post-traumatic stress disorder especially with disaster events. Furthermore, they are highly prone to becoming psychiatric casualties despite the absence of physical injury to themselves. And as any pediatric HCP can tell you, the younger pediatric patients tend to also have greater levels of anxiety, especially while preparing for invasive procedures such as phlebotomy and intravenous line placement (Hilmas et al. 2008; Jacobson and Severin 2012; Severin 2011). Please refer to Chap. 12 for more detailed content on mental health.

5.2 Pediatric Disaster Planning

The World Health Organization and the Pan American Health Organization define a *disaster* as "an event that occurs in most cases suddenly and unexpectedly, causing severe disturbances to people or objects affected by it, resulting in the loss of life and harm to the health of the population, the destruction or loss of community property, and/or severe damage to the environment. Such a situation leads to disruption in the normal pattern of life, resulting in misfortune, helplessness, and suffering, with adverse effects on the socioeconomic structure of a region or a country and/or modifications of the environment to such an extent that there is a need for assistance and immediate outside intervention" (Lynch and Berman 2009).

Types of disasters usually fall into two broad categories: natural and man-made. Natural disasters are generally associated with weather and geological events, including extremes of temperature, floods, hurricanes, earthquakes, tsunamis, volcanic eruptions, landslides, and drought. Naturally occurring epidemics, such as the 2009 H1N1, 2014 Ebola, and 2019 novel coronavirus (COVID-19) outbreaks, are often included in this category. Man-made disasters are usually associated with a criminal attack such as an active shooter incident, or a terrorist attack using weapons such as explosive, biological, or chemical agents. However, man-made disasters can also refer to human-based technological incidents, such as a building or bridge collapse, or events related to the manufacture, transportation, storage, and use of hazardous materials, such as the 1986 Chernobyl radiation leak and the 1984 Bhopal toxic gas leak. Even though disasters can be primarily placed into any of these two categories, they can often impact each other and compound the magnitude of any disaster incident (United States Department of Homeland Security, Office of Inspector General 2009). A prime example is the March 2011 Tohoku earthquake leading to a tsunami (natural) that triggered the Fukushima Daiichi nuclear disaster (man-made).

Disasters can also be characterized by the location of such an event. *Internal* disasters are those incidents that occur within the health care facility or system. Employees, physical plant, workflow and operations of the clinic, hospital, or system can be disrupted. *External* disasters are those incidents that occur outside of the health care facility or system. This impacts the community surrounding the facility, proximally or distally, but does not directly threaten the facility or its employees. As with natural and man-made disasters, internal and external disasters can impact each other. For example, an overflow of patients during a high census period may

lead to the shutdown of the hospital to any new patients (internal disaster). This will place the hospital on bypass and possibly stress other hospitals in the community beyond their means (external disaster). A terrorist event, such as the release of sarin in a subway system during a busy morning commute, can lead to massive disruption in the community (external disaster). All the victims of the attack will seek medical care at nearby hospitals, possibly overwhelming the health care staff and depleting critical resources (internal disaster). Characterization of disasters by geography (local, state, national, and international) can also be used. Again, no matter the site of the incident, a disaster in one area could easily create a disaster in another geographical region. For example, a factory and its community could be ravaged by a hurricane (local disaster). If this is the only factory in the world to produce a certain medication, this could lead to critical shortages to hospitals all around the world (international disaster).

The term "disaster preparedness" has been used over the years as a way to describe efforts to manage any disaster event. However, preparedness is only one aspect of the process. The use of the term disaster planning is more appropriate. It considers all aspects needed for an effective effort and is dependent on additional phases, not just preparedness. National preparedness efforts, including planning, are now informed by the Presidential Policy Directive (PPD) 8 that was signed by the president in March 2011 and describes the nation's approach to preparedness (United States Department of Education, Office of Elementary and Secondary Education, Office of Safe and Healthy Students 2013; United States Department of Homeland Security 2018b). A recommended method for disaster preparedness efforts is the utilization of an "all-hazards" model of emergency management (Adini et al. 2012; Waugh 2000). The four overlapping phases of the model include mitigation, preparedness, response, and recovery. The *mitigation* phase involves "activities designed to prevent or reduce losses from a disaster" (Waugh 2000). Examples include land use planning in flood plains, structural integrity measures in earthquake zones, and deployment of security cameras. The *preparedness* phase includes the "planning of how to respond in an emergency or a disaster, and developing capabilities for more effective response" (Waugh 2000). Examples include training programs for emergency responders, drills and exercises, early warning systems, contingency planning, and development of equipment and supply caches. Up to this point, all planning efforts are proactive and not reactive. Often times, a hazard analysis is conducted to delineate areas of strengths and identify potential risks. It helps in "the identification of hazards, assessment of the probability of a disaster, and the probable intensity and location; assessment of its potential impact on a community; the property, persons, and geographic areas that may be at risk; and the determination of agency priorities based on the probability level of a disaster and the potential losses" (Waugh 2000). After a disaster or emergency incident occurs, the *response* phase, or "immediate reaction to a disaster",(Waugh 2000) begins. Examples include mass evacuations, sandbagging buildings and other structures, providing emergency medical services, firefighting, and restoration of public order. In some situations, the response period may be a short (e.g., house fire), intermediate (e.g., bomb detonation), or extended (e.g., pandemic influenza) duration. After a period of time, the *recovery* phase follows. These are "activities that continue beyond the

emergency period to restore lifelines" (Waugh 2000). Examples include the provision of temporary shelter, restoration of utilities such as power, critical stress debriefing for responders, and victims, job assistance and small business loans, and debris clearance. Recovery always seems to be the most unpredictable; it may take days to months to years. As demonstrated with recent hurricanes Harvey, Irma, and Maria in 2017, the most affected regions are still in the phase of recovery and may be along a prolonged track as Hurricane Katrina in 2005.

As mentioned, the early phases of planning (mitigation and preparedness) truly hinge upon the environment or community surrounding the health care site (e.g., clinic, hospital, or long-term care facility). Identification of potential hazards and risks is a key step in disaster planning. Using a Hazard Vulnerability Assessment (HVA) or a Threat and Hazard Identification and Risk Assessment (THIRA) can provide a basis for mitigation and prevention tasks. An HVA/THIRA emphasizes which types of natural or man-made disasters are likely to occur in a community (e.g., tornado, flood, chemical release, or terrorist event). They further highlight the impact those disasters may have on the community and any capabilities that are in place that may lessen the effects of the disaster (Illinois Emergency Medical Services for Children 2018).

A basic principle of the HVA methodology is to determine the risk of such an event or attack occurring at a given hospital or hospital system. Simply, the risk is a product of the probability of an event and the severity of such an event if it occurs (risk = probability × severity). However, there are many complexities in quantifying terrorism risk (Waugh 2005; Woo 2002). It is important to note that in some circumstances, exposure may need to be included in the equation (risk = probability × severity × exposure), but usually for operational risk management applications (Mitchell and Decker 2004). At any rate, issues to consider for the probability of an event occurring include, but are not limited to, geographic location and topography, proximity to hazards, degree of accessibility, known risks, historical data, and statistics of various manufacturer/vendor products. Severity, on the other hand, is dependent on the gap between the magnitude of an event and mitigation for the given event (severity = magnitude – mitigation). Magnitude varies upon the impact of the event to humans, property, and/or business. Mitigation varies upon the development of internal and external readiness before a disaster strikes. As one can surmise, if the magnitude of the event outstrips the mitigation, the event is considered a threatening hazard. Once the HVA is completed, the health care site should immediately prioritize planning efforts for the top 5–10 hazards and develop plans accordingly. All other identified hazards must also be addressed to ensure a broad and robust disaster plan. It is important to realize that local and regional entities also perform comprehensive HVAs. A concerted analysis among a hospital and key community stakeholders is optimal for a coordinated plan.

An HVA/THIRA contains both quantitative and qualitative components. Specific tools have been developed through private and public organizations (e.g, FEMA) that can help in the analysis (United States Department of Homeland Security, Federal Emergency Management Agency 2001). Using these tools as a guide, the entity can determine what types of hazards have a high, medium, or low probability of occurring within specific geographic boundaries. Typically, these tools do not have

components specific to children or other at-risk populations. However, the tools can be adapted either directly by adding children to specific hazards or ensuring considerations specific to children are incorporated into the HVA/THIRA calculations. The HVA/THIRA should be reviewed and updated minimally on an annual basis to identify changing or external circumstances. This includes conducting a pediatric-specific disaster risk assessment to identify where children congregate and their risks (e.g., schools, popular field trip designations, summer camps, houses of worship, and juvenile justice facilities) (Illinois Emergency Medical Services for Children 2018).

Of note, HVA techniques have been utilized for pediatric-specific disaster plans. Having a separate pediatric HVA (PHVA) is crucial to a well-rounded and robust health care disaster plan. First, it demonstrates the extent of the pediatric population in the community. It is estimated that 25% of the population fits within the age range of pediatric patients. In some situations, it may be more. During the performance of a PHVA, it was demonstrated that 29% of the community was less than 19 years of age (Jacobson and Severin 2012). Second, a PHVA increases the situational awareness of those tasked to plan for disasters that involve children and adolescents. Often times, children and adolescents are excluded from local and regional disaster plans. The unique vulnerabilities of pediatric patients will demand appropriate drills, exercises, equipment, medications, and expertise. Thirdly, identifying pediatric risks in a community will help prioritize efforts of planning, especially in those hospitals not accustomed to caring for pediatric patients. Finally, a PHVA helps to develop a framework for global pediatric disaster planning. This can extend beyond a local community and actually advance city, state, regional, and national disaster planning efforts. There has been a development of web-based tools to simplify and enhance the PHVA process (Jacobson and Severin 2012).

After an HVA/THIRA has been completed, the results should be used to help direct and plan drills/exercises based on high impact and high probability threats. It is advised to conduct an HVA/THIRA on an annual basis to assess specific threats unique to your organization's physical structure as well as the surrounding geographic environment. It will also provide insight into whether there is an improvement in previous planning efforts. Completion of a population assessment that provides a demographic overview of the community with a breakdown of the childhood population is strongly recommended in conjunction with the HVA/THIRA. Collaborating with other community partners, such as local health departments and emergency management agencies, can assist an organization with the conduction of a comprehensive HVA/THIRA (Illinois Emergency Medical Services for Children 2018). Please see Chap. 13 for further information on hospital planning.

5.3 General Resuscitation, Equipment, Supplies, and Medications

Pediatric supplies, equipment, and medications will be scarce during a disaster. It will become more of an issue if the health care facility is not accustomed to caring for acutely ill pediatric patients. This will be further exacerbated by a massive surge

of acutely ill pediatric patients, a widespread or prolonged disaster, and supply line disruptions. To protect the health security of children and families during a public health emergency, the Assistant Secretary for Preparedness and Response (ASPR) manages and maintains the Strategic National Stockpile (SNS), a cache of medical countermeasures for rapid deployment and use in response to a public health emergency or disaster (Fagbuyi et al. 2016). Various pediatric-specific supplies and countermeasures are included in the SNS. Maintaining a supply of medications and medical supplies for specific health threats allows the stockpile to respond with the right product when a specific disease or agent is known. If a community experiences a large-scale public health incident in which the disease or agent is unknown, the first line of support from the stockpile is to send a broad-range of pharmaceuticals and medical supplies. The majority of stockpile assets are held in storage and kept as managed inventory. Each package contains 50 tons of emergency medical resources. The SNS is deployed along with a Federal Medical Station Strike Team who have in-depth knowledge of the stockpile and supply operations (Assistant Secretary for Preparedness and Response, United States Department of Health and Human Services 2018). The SNS, depending upon the threat, is intended to only supplement state and local supplies used for immediate care during the initial response. Contents are prepacked and configured in transport-ready containers for rapid delivery anywhere in the U.S. within 12 h of the federal decision to deploy (Assistant Secretary for Preparedness and Response, United States Department of Health and Human Services 2018). However, the federal recommendation is to always maintain a local stockpile of supplies to support patients, families, and staff independently for at least 96 h (Illinois Emergency Medical Services for Children 2018).

There are hospital guidelines for pediatric-specific medications, equipment, and supplies for pediatric emergency preparedness (American Academy of Pediatrics, Committee on Pediatric Emergency Medicine, American College of Emergency Physicians, Pediatric Committee, and Emergency Nurses Association Pediatric Committee 2009) (Tables 5.1 and 5.2). Much of the equipment is already recommended for ambulance services responding to pediatric emergencies (American College of Surgeons Committee on Trauma, American College of Emergency Physicians, National Association of EMS Physicians, Pediatric Equipment Guidelines Committee Emergency Medical Services for Children (EMSC) Partnership for Children Stakeholder Group, American Academy of Pediatrics 2009) and health care centers tasked with receiving acutely ill pediatric patients (American Academy of Pediatrics, Committee on Pediatric Emergency Medicine, American College of Emergency Physicians, Pediatric Committee, and Emergency Nurses Association Pediatric Committee 2009; Place and Martin 2012). The emergency equipment and supply lists can easily be adapted for any pediatric disaster emergency (Place and Martin 2012) or incident requiring pediatric mass critical care (Desmond et al. 2011). Age-appropriate nutrition, hygiene, bedding, and toys/distraction devices should also be available (Illinois Emergency Medical Services for Children 2013) (Tables 5.3 and 5.4).

Table 5.1 Potentially useful medications in pediatric emergencies[a]

Adenosine	Diphenhydramine	Glucagon	Lorazepam	Phenytoin
Albuterol	Dobutamine	Glucose	Magnesium sulfate	Prednisone/Prednisolone
Alprostadil (PGE1)	Dopamine	Hydrocortisone	Mannitol	Procainamide
Amiodarone	Epinephrine	Ipratropium bromide	Methylprednisolone	Propranolol
Atropine	Epinephrine, racemic	Kayexalate™ (sodium	Midazolam	Rocuronium
Bicarbonate, sodium	Fentanyl	polystyrene sulfonate)	Milrinone	Succinylcholine
Calcium chloride	Flumazenil	Ketamine	Morphine	Vecuronium
Charcoal, activated	Fosphenytoin	Levalbuterol	Naloxone	
Dexamethasone	Furosemide	Lidocaine	Nitroprusside	
Diazepam			Norepinephrine	
			Phenobarbital	

[a]References: (American Academy of Pediatrics and American College of Emergency Physicians et al. 2012; American Academy of Pediatrics, Committee on Pediatric Emergency Medicine, American College of Emergency Physicians, Pediatric Committee, and Emergency Nurses Association Pediatric Committee 2009; Hegenbarth 2008)

Table 5.2 Guidelines for pediatric-specific equipment and supplies[a]

General equipment	Patient warming device (infant warmer) Restraint device for children Weight scale for infants and children Length-based resuscitation tape Pain-scale assessment tools appropriate for age
Monitoring equipment	Blood pressure cuffs (neonatal, infant, child, adult) Doppler ultrasound devices ECG monitor/defibrillator with pediatric and adult capabilities, including pediatric-sized pads/paddles Invasive thermometer Pulse oximeter with pediatric and adult probes Continuous end-tidal carbon dioxide monitoring device
Respiratory	Endotracheal tubes • Uncuffed: 2.5 and 3.0 mm • Cuffed or uncuffed: 3.5, 4.0, 4.5, 5.0, and 5.5 mm • Cuffed: 6.0, 6.5, 7.0, 7.5, and 8.0 mm Feeding tubes (5F and 8F) Laryngoscope blades curved: 2 and 3; straight: 0, 1, 2, and 3 Laryngoscope handle Magill forceps (pediatric and adult) Nasopharyngeal airways (infant, child, and adult) Oropharyngeal airways (sizes 0–5) Stylets for endotracheal tubes (pediatric and adult) Suction catheters (infant, child, and adult) Tracheostomy tubes (sizes 2.5, 3.0, 3.5, 4.0, 4.5, 5.0, and 5.5 mm) Yankauer suction tip Bag-mask device (manual resuscitator), self-inflating (infant size: 450 mL; adult size: 1000 mL) Clear oxygen masks (standard and nonrebreathing) for an infant, child, and adult Masks to fit bag-mask device adaptor (neonatal, infant, child, and adult sizes) Nasal cannulas (infant, child, and adult) Nasogastric tubes (sump tubes): infant (8F), child (10F), and adult (14F–18F) Laryngeal mask airway[a]
Vascular access	Arm boards (infant, child, and adult sizes) Catheter over-the-needle device (14–24 gauge) Intraosseous needles or device (pediatric and adult sizes) Intravenous catheter–administration sets with calibrated chambers and extension tubing and/or infusion devices with ability to regulate rate and volume of infusate Umbilical vein catheters (3.5F and 5.0F)[b] Central venous catheters (4.0F–7.0F) Intravenous solutions to include normal saline, dextrose 5% in normal saline, and dextrose 10% in water
Fracture-management devices	Extremity splints, including femur splints (pediatric and adult sizes) Spine-stabilization method/devices appropriate for children of all ages[c]

(continued)

Table 5.2 (continued)

Pediatric trays or kits	Lumbar puncture tray, including infant (22-gauge), pediatric (22-gauge), and adult (18- to 21-gauge) Lumbar puncture needles Supplies/kit for patients with difficult airway conditions (to include but not limited to supraglottic airways of all sizes, such as the laryngeal mask airway, two-needle cricothyrotomy supplies, and surgical cricothyrotomy kit) Tube thoracostomy tray Chest tubes to include infant, child, and adult sizes (infant: 10F–12F; child, 16F–24F; adult, 28F– 40F) Newborn delivery kit (including equipment for initial resuscitation of a newborn infant: umbilical clamp, scissors, bulb syringe, and towel) Urinary catheterization kits and urinary (indwelling) catheter (6F–22F)

References: (American Academy of Pediatrics and American College of Emergency Physicians et al. 2012; American Academy of Pediatrics, Committee on Pediatric Emergency Medicine, American College of Emergency Physicians, Pediatric Committee, and Emergency Nurses Association Pediatric Committee 2009; American College of Surgeons Committee on Trauma, American College of Emergency Physicians, National Association of EMS Physicians, Pediatric Equipment Guidelines Committee Emergency Medical Services for Children (EMSC) Partnership for Children Stakeholder Group, American Academy of Pediatrics 2009; Desmond et al. 2011)

[a]Laryngeal mask airways could be shared with anesthesia but must be immediately accessible to the ED

[b]Feeding tubes (size 5F) may be used as umbilical venous catheters but are not ideal. A method for securing the umbilical catheter, such as an umbilical tie, should also be available

[c]A spinal stabilization device is one that can stabilize the neck of an infant, child, or adolescent in a neutral position

Table 5.3 Useful general care items for managing the needs of children[a]

Nutrition, hygiene, and sleeping supplies			
Nutrition	Nutrition supplies	Hygiene	Sleeping supplies
Baby formula Baby food/cereal Oral electrolyte solutions, age-appropriate	Baby bottles and nipples for bottles Plastic bowls Sippy cups Toddler feeding spoons/forks Manual breast pumps with bottles Small towels for spit up Pacifiers	Hand sanitizer Washcloths/towels Diapers (sizes 1–6) Pull ups (sizes 4 T–5 T) Diaper wipes Container for soiled diapers Diaper rash ointment Disposable changing pads Toddler toilet seat (potty chairs) Cloth diapers Infant wash (soap) Infant bathing bins Baby laundry detergent (dye and fragrance free)	Portable cribs, bassinets, play pens Laundry baskets (can be used for infant bed) Bed sheets Lightweight hypoallergenic blankets

[a]Reference: (Illinois Emergency Medical Services for Children 2013)

Table 5.4 Age-appropriate Toys/Distraction Devices[a]

For safety and infection control reasons, only stock toys for children that are washable, nontoxic, difficult to break, and without small pieces (NOTE: Toys with small parts may present a potential choking hazard). Consider including some toys/objects that could be given to the child to keep (small new stuffed animals, cars, stickers, coloring books/crayons, etc.).

Infants/toddlers	Preschool/school age	Adolescents	General
Musical/light toys	Plastic animals, action figures, cars	Teen rated games	Bubbles
Pop-up toys	Building blocks	Video/electronic games	Balls
Mirrors	Books	Journal and writing supplies	Coloring books and supplies
Shape sorters	Dolls	Books and magazines	Arts and craft supplies
Stacking rings	Elementary school rated games	Activity sets	Music
Activity blocks	Foam balls	Music	Stickers
Teething rings			Sculpting clay
Board books			
Beginner toy cars			

[a]Reference: (Illinois Emergency Medical Services for Children 2013)

When a pediatric disaster victim presents acutely ill to the hospital, various emergency interventions will be needed to stabilize the patient. Evaluation of the pediatric patient should include a primary survey (ABCDE), secondary survey (focused SAMPLE history and focused physical examination), and diagnostic assessments (laboratory, radiological, and other advanced tests). This will guide further therapeutic interventions. Particular attention should be given to the identification of respiratory and/or circulatory derangements of the child, including airway obstruction, respiratory failure, shock, and cardiopulmonary failure. Interventions will be based on physiologic derangements of the pediatric patient and determined by the scope of practice and protocols, such as standard resuscitation algorithms for neonatal (American Academy of Pediatrics and American Heart Association et al. 2016) and pediatric (American Heart Association 2016) victims.

The HCP must be knowledgeable of various emergency medications (Table 5.1) used for children, the appropriate dosages and their mechanism of action, any potential side effects, and drug/drug interactions. Other medications, such as antibiotics, antidotes, or countermeasures, may be needed as well. Pharmacologic therapy should be initiated immediately based on clinical suspicion and not delayed due to pending laboratory tests (e.g., antibiotics for presumed infection/sepsis or antidotes for suspected nerve agents). Dosages should be based on the patient's weight or a length-based weight system. Utilizing current resuscitation references, such as *Handbook of Emergency Cardiovascular Care for Healthcare Providers*, (American Heart Association 2015) *Neonatal Resuscitation Program*, (American Academy of Pediatrics and American Heart Association et al. 2016) *Pediatric Advanced Life Support*, (American Heart Association 2016) or *Advanced Pediatric Life Support*, (American Academy of Pediatrics and American College of Emergency Physicians 2012) is highly recommended (American Academy of Pediatrics, Committee on Pediatric Emergency Medicine, American College of Emergency Physicians, Pediatric Committee, and Emergency Nurses Association Pediatric Committee

2009; Hegenbarth 2008). Pediatric countermeasure dosing recommendations for various chemical, biological, and radiological/nuclear exposures can be found online through the U.S. Department of Health and Human Services (United States Department of Health and Human Services, Office of the Assistant Secretary for Preparedness and Response, National Library of Medicine 2019; United States Department of Health and Human Services, Chemical Hazards Emergency Medical Management (CHEMM) 2019) or Centers for Disease Control and Prevention (Centers for Disease Control and Prevention 2018a). Pediatric antidote dosing cards (Montello et al. 2006) or hard copy countermeasure manuals may be more practical, especially during a disaster incident when computer service or internet access may be unreliable.

5.4 Natural Disasters

In 1988, the Centre for Research on the Epidemiology of Disasters (CRED) launched the Emergency Events Database (EM-DAT). EM-DAT was created with the initial support of the World Health Organization (WHO) and the Belgian Government. The main objective of the database is to serve the purposes of humanitarian action at national and international levels. The initiative aims to rationalize decision-making for disaster preparedness as well as provide an objective base for vulnerability assessment and priority setting. EM-DAT contains essential core data on the occurrence and effects of over 22,000 mass disasters in the world from 1900 to the present day. The database is compiled from various sources, including United Nation agencies, nongovernmental organizations (NGOs), insurance companies, research institutes, and press agencies (CRED 2019). As described in the CRED report entitled *Natural disasters 2017: lower mortality, higher cost*, a disaster is entered into the database if at least one of the following criteria is fulfilled: 10 or more people reported killed; 100 or more people reported affected; declaration of a state of emergency; and/or call for international assistance (CRED 2018).

In *Economic losses, poverty and disasters 1998–2017: CRED/UNISDR Report*, the CRED defines a disaster as "a situation or event which overwhelms local capacity, necessitating a request at national or international level for external assistance; an unforeseen and often sudden event that causes great damage, destruction and human suffering" (CRED 2018). The CRED EM-DAT classifies disasters according to the type of hazard that triggers them. The two main disaster groups are natural and technological disasters.

There are six natural disaster subgroups. *Geophysical* disasters originate from the solid earth and include earthquake (ground movement and tsunami), dry mass movement (rock fall and landslides), and volcanic activity (ash fall, lahar, pyroclastic flow, and lava flow). Lahar is a hot or cold mixture of earthen material flowing on the slope of a volcano either during or between volcanic eruptions. *Meteorological* disasters are caused by short-lived, micro- to meso-scale extreme weather and

atmospheric conditions that last from minutes to days and include extreme temperatures (cold wave, heat wave, and severe winter conditions such as snow/ice or frost/freeze), fog, and storms. Storms can be extra-tropical, tropical, or convective. Convective storms include derecho, hail, lightning/thunderstorm, rain, tornado, sand/dust storm, winter storm/blizzard, storm/surge, and wind. Derecho is a widespread and usually fast-moving windstorm associated with convection/convective storm and includes downburst and straight-line winds. *Hydrological* disasters are caused by the occurrence, movement, and distribution of surface/subsurface freshwater and saltwater and include floods, landslides (an avalanche of snow, debris, mudflow, and rockfall), and wave action (rogue wave and seiche). Flood types can be coastal, riverine, flash, or ice jam. *Climatological* disasters are caused by long-lived, meso- to macro-scale atmospheric processes ranging from intraseasonal to multidecadal climate variability and include drought, glacial lake outburst, and wildfire (forest fire, land fire: brush, bush, or pasture). *Biological* disasters are caused by the exposure to living organisms and their toxic substances or vector-borne diseases that they may carry and include epidemics (viral, bacterial, parasitic, fungal, and prion), insect infestation (grasshopper and locust), and animal accidents. *Extraterrestrial* disasters are caused by asteroids, meteoroids, and comets as they pass near-earth, enter Earth's atmosphere, and/or strike the earth, and by changes in the interplanetary conditions that affect the Earth's magnetosphere, ionosphere, and thermosphere. Types include impact (airbursts) and space weather (energetic particles, geomagnetic storm, and shockwave) events (CRED 2019).

There are three technological disaster subgroups. *Industrial* accidents include chemical spills, collapse, explosion, fire, gas leak, poisoning, radiation, and oil spills. A chemical spill is an accidental release occurring during the production, transportation, or handling of hazardous chemical substances. *Transport* accidents include disasters in the air (airplanes, helicopters, airships, and balloons), on the road (moving vehicles on roads or tracks), on the rail system (train), and on the water (sailing boats, ferries, cruise ships, and other boats). *Miscellaneous* accidents vary from collapse to explosions to fires. Collapse is an accident involving the collapse of a building or structure and can either involve industrial structures or domestic/nonindustrial structures (CRED 2019). Technological disasters are considered man-made, but as suggested by their subgroup, they are accidental and not intentional.

The United Nations Office for Disaster Risk Reduction (UNISDR) and CRED report, *Economic losses, poverty, and disasters 1998–2017*, reviews global natural disasters during that time period, their economic impact, and the relationship with poverty. Between 1998 and 2017, climate-related and geophysical disasters killed 1.3 million people and left a further 4.4 billion injured, homeless, displaced, or in need of emergency assistance. Although the majority of fatalities were due to geophysical events, mostly earthquakes and tsunamis, 91% of all disasters was caused by floods, storms, droughts, heatwaves, and other extreme weather events. The financial impact was staggering. In 1998–2017, disaster-hit countries reported direct economic losses valued at US$ 2908 billion, of which climate-related disasters caused US$ 2245 billion

or 77% of the total. This was up from 68% (US$ 895 billion) of losses (US$ 1313 billion) reported between 1978 and 1997. Overall, reported losses from extreme weather events rose by 151% between these two 20-year periods. In absolute monetary terms, over the last 20-years, the USA recorded the biggest losses (US$ 945 billion), reflecting high asset values as well as frequent events. China, by comparison, suffered a significantly higher number of disasters than the USA (577 vs. 482) but lower total losses (US$ 492 billion) (CRED 2018) (Figs. 5.1, 5.2, 5.3, 5.4, 5.5, 5.6, 5.7, 5.8 and 5.9).

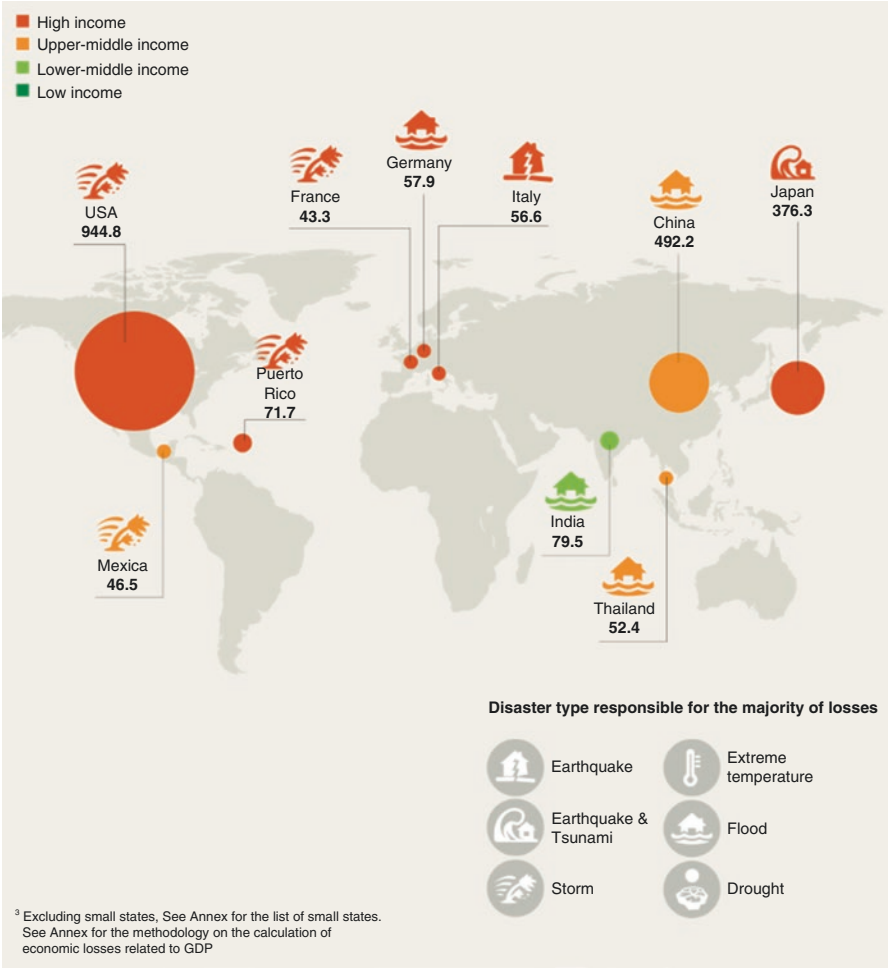


Fig. 5.1 Top 10 countries/territories in terms of absolute losses*. *Used with permission from CRED (2018)

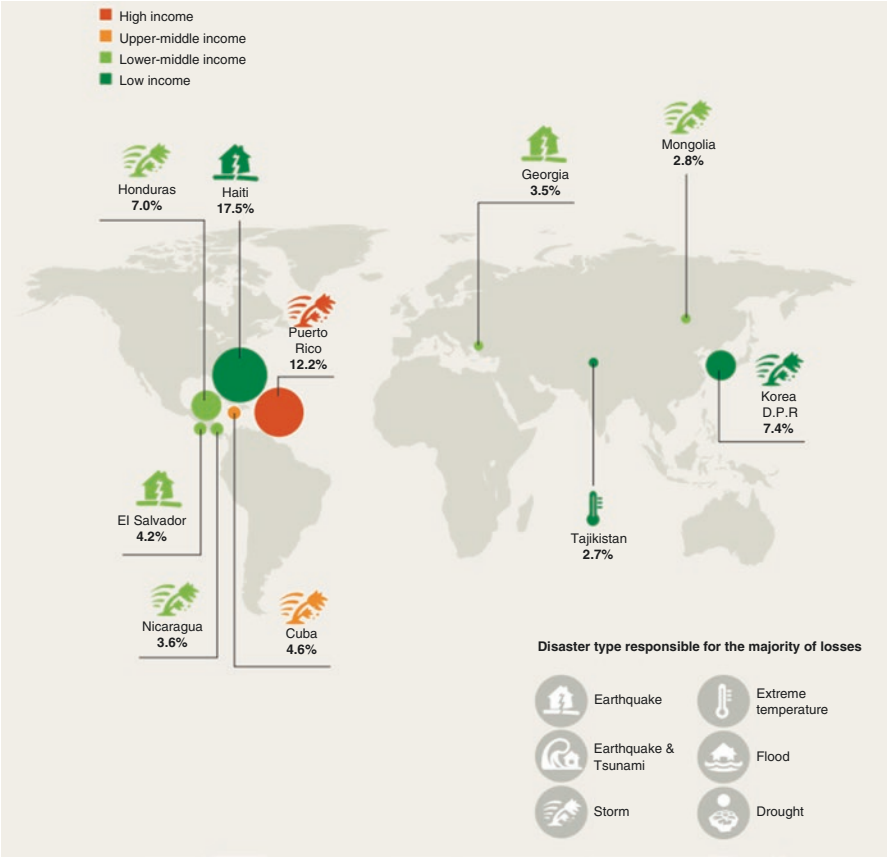


Fig. 5.2 Top 10 countries/territories in terms of average annual percentage losses relative to GDP*. *Used with permission from CRED (2018)

In 2018, 281 climate-related and geophysical incidents in the world were estimated with 10,733 deaths and over 60 million people impacted. Indonesia recorded approximately half of the deaths with India accounting for half of those impacted by disasters. Notable features of 2018 were intense seismic activity in Indonesia, a series of disasters in Japan, floods in India, and an eventful year for both volcanic activity and wildfires. However, an ongoing trend of lower death tolls from previous years continued into 2018 (Centre for Research on the Epidemiology of Disasters (CRED) and United Nations Office for Disaster Risk Reduction (UNISDR) 2019) (Tables 5.5, 5.6, 5.7, 5.8, 5.9, 5.10 and 5.11).

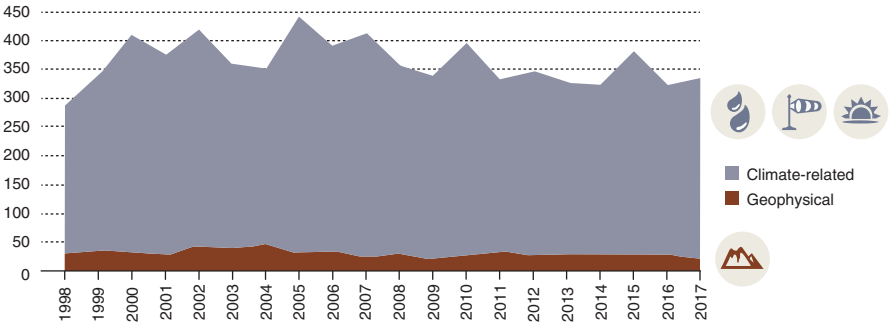


Fig. 5.3 Number of disasters by major category per year 1998–2017*. *Used with permission from CRED (2018)

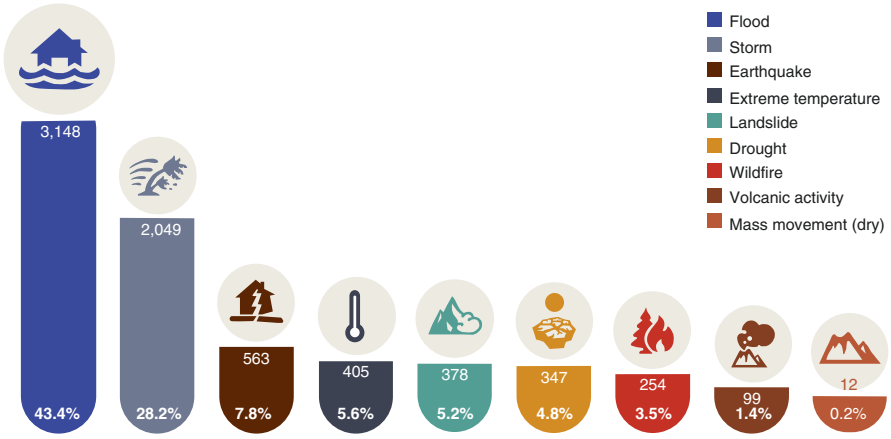


Fig. 5.4 Numbers of disasters per type 1998–2017*. *Used with permission from CRED (2018)

There are no specific deviations when medically managing children after a natural disaster. According to Sirbaugh and DiRocco (2012) "Small-scale mass casualty incidents occur daily in the United States. Few present unusual challenges to the local medical systems other than in the number of patients that must be treated at one time. Except in earthquakes, explosions, building collapses, and some types of terrorist attacks, the same holds true for large-scale disasters. Sudden violent disaster mechanisms can produce major trauma cases, including patients needing field amputations or management of crush syndrome. For the most part, medicine after a disaster is much the same as it was before the disaster, with more minor injuries, more people with exacerbations of their chronic illnesses, and number of patients

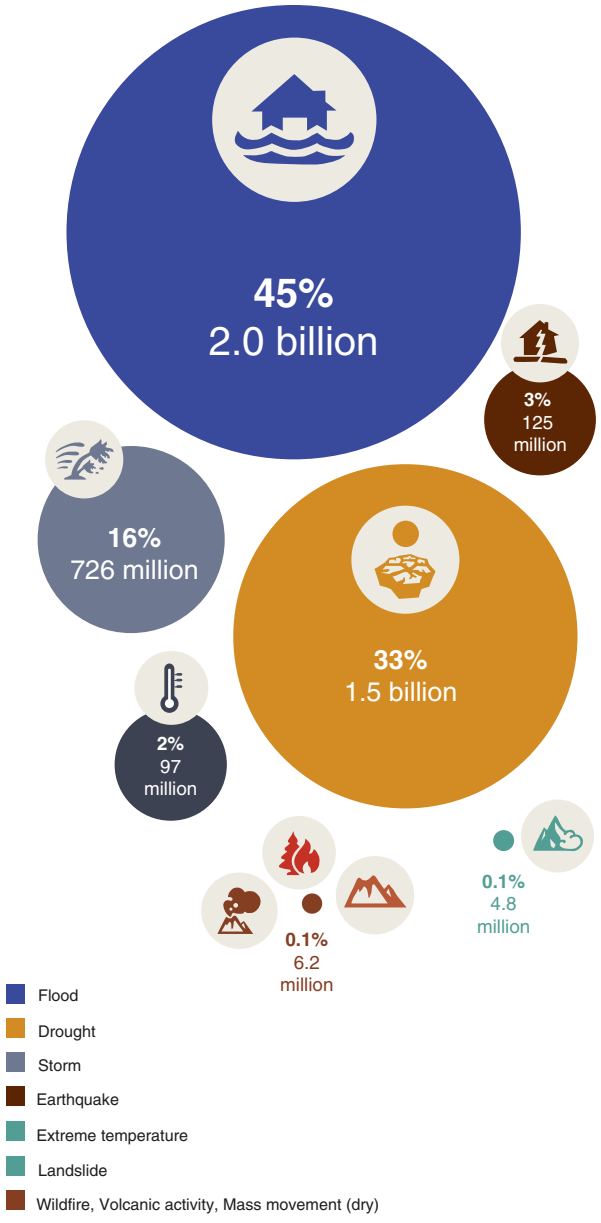


Fig. 5.5 Number of people affected per disaster type 1998–2017*. *Used with permission from CRED (2018)

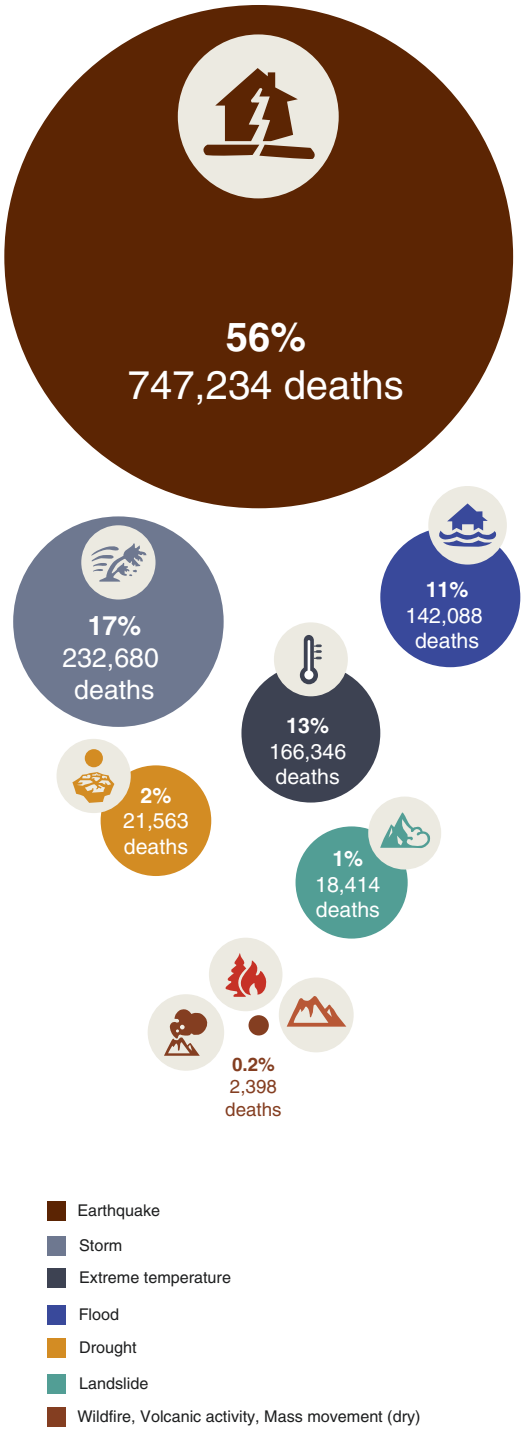


Fig. 5.6 Number of deaths per disaster type 1998–2017*. *Used with permission from CRED (2018)

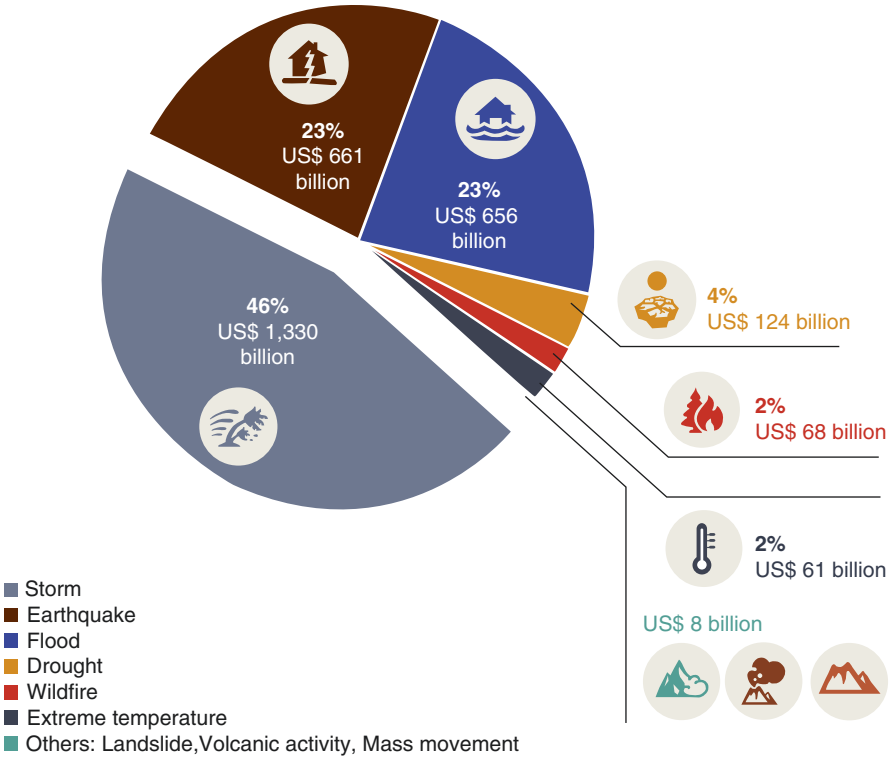


Fig. 5.7 Breakdown of recorder economic losses (US$) per disaster type 1998–2017. *Used with permission from CRED (2018)

seeking what is ordinarily considered primary care. This is true for children and adults." It should be noted, however, that children have a predisposition to illness and injury after natural disasters. The HCP must be able to identify any health problems and treat the child effectively and efficiently while utilizing standard resuscitation protocols as indicated.

Traumatic injuries may be seen after any natural disaster. The injuries can range from minor scrapes and bruises to major blunt trauma or traumatic brain injury. Children are at increased risk for injury since adults are distracted by recovery efforts and may not be able to supervise them closely. The environment may not be safe due to environmental hazards, such as collapsed buildings, sinkholes, and high water levels. Dangerous equipment used during relief efforts may be present, such as heavy earth moving equipment, chainsaws, and power generators. Hazardous chemicals, such as gasoline and other volatile hydrocarbons, may be readily accessible or taint the environment. Without suitable shelter, children are also exposed to weather, animals, and insects (Sirbaugh and DiRocco 2012).

Infectious diseases may also pose a problem to children after a natural disaster. Infectious patterns will persist during a disaster based on the season and time of

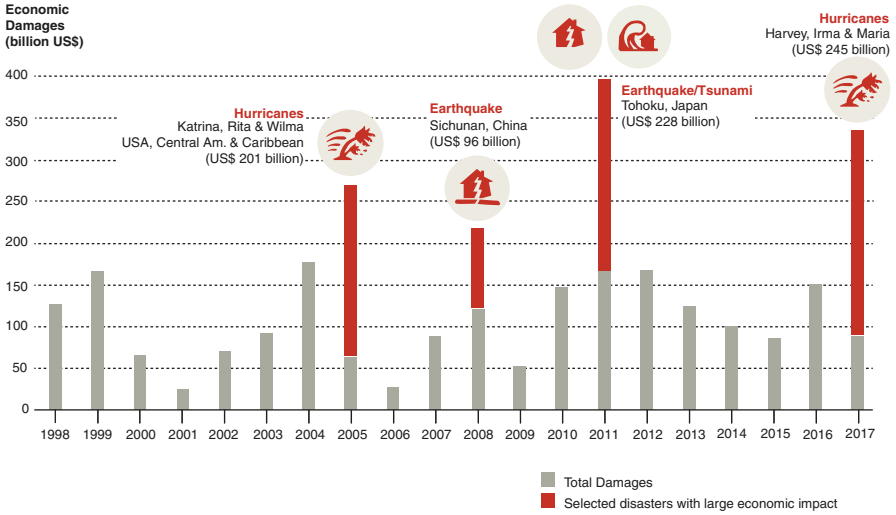


Fig. 5.8 Total reported economic losses per year with major events highlighted 1998–2017. *Used with permission from CRED (2018)

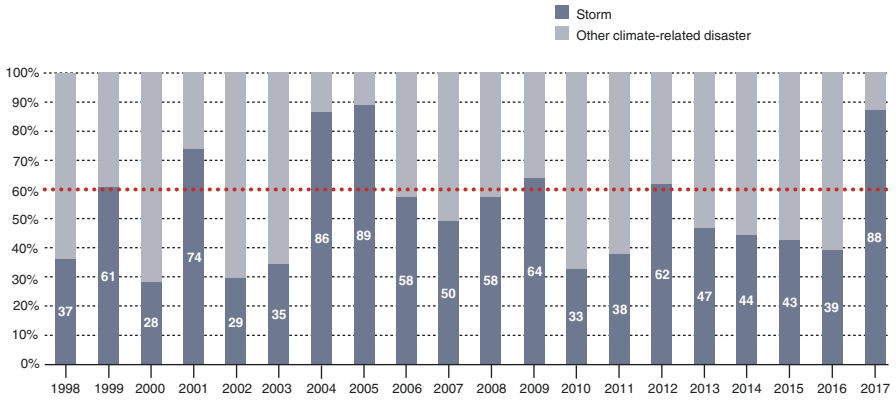


Fig. 5.9 Share of losses due to storms as a percentage of annual climate-related disaster losses 1998–2017. *Used with permission from CRED (2018)

year. There may be outbreaks or epidemics of highly contagious infections (e.g., influenza, respiratory syncytial virus, *Streptococcus pyogenes*) due to mass sheltering of children and families. Poor nutrition or decreased availability of food may lower their resistance against infections. Various water-borne or food-borne diseases may cause illnesses in children. Poor hygiene and mass shelter environments may exacerbate these illnesses. Immunized children should be protected against

Table 5.5 Summary of disaster events in 2018[a]

Natural disaster category	Events	Key details
Droughts and extreme temperature	39	Kenya: three million people affected Afghanistan: 2.2 million people affected Central America: 2.5 million affected Europe
Earthquakes and Tsunamis	20	Papua New Guinea: 181 killed, over half million affected Island of Lombok, Indonesia: 564 killed Island of Sulawesi, Indonesia: 3400 killed
Floods	108	Somalia: 700,000 affected Nigeria: 300 killed, four million affected Japan: 230 killed Kerala, India: 504 killed, over 23 million affected
Storms	84	United States: Hurricanes Florence and Michael Japan: Typhoon Jebi Asia China India Philippines
Volcanic activity	7	Volcan de Fuego, Guatemala: 400 killed, 1.7 million affected Anak Krakatau, Indonesia: resulting tsunami resulted in 400 killed on the islands of Sumatra and Java
Wildfires	9	Attica Fires, Greece: 126 killed Camp Fire, California, US: 88 killed

[a]Used with permission from CRED, 2019 (Centre for Research on the Epidemiology of Disasters (CRED) and United Nations Office for Disaster Risk Reduction (UNISDR) 2019)

Table 5.6 Top 10 countries by number of people affected (2018)[a]

Rank	Country	Total number of people affected
1	India	23,900,348
2	Philippines	6,490,216
3	China	6,415,024
4	Nigeria	3,938,204
5	Guatemala	3,291,359
6	Kenya	3,211,188
7	Afghanistan	2,206,750
8	USA	1,762,103
9	Japan	1,599,497
10	Madagascar	1,472,190

[a]Used with permission from CRED, 2019 (Centre for Research on the Epidemiology of Disasters (CRED) and United Nations Office for Disaster Risk Reduction (UNISDR) 2019)

Table 5.7 Top 10 countries by total death toll (2018)[a]

Rank	Country	Total death toll
1	Indonesia	4535
2	India	1388
3	Guatemala	427
4	Japan	419
5	China	341
6	Nigeria	300
7	USA	298
8	Pakistan	240
9	Korea DPR	237
10	Philippines	221

[a]Used with permission from CRED, 2019 (Centre for Research on the Epidemiology of Disasters (CRED) and United Nations Office for Disaster Risk Reduction (UNISDR) 2019)

Table 5.8 Top 10 deadliest disaster events (2018)[a]

Rank	Event	Country	Death toll
1	Earthquake/Tsunami	Indonesia	3400
2	Earthquake, August	Indonesia	564
3	Flood, August	India	504
4	Volcanic Activity/Tsunami, December	Indonesia	453
5	Volcanic Activity, June	Guatemala	425
6	Flood, June	Japan	220
7	Flood, September	Nigeria	199
8	Heatwave, May	Pakistan	180
9	Flood, August	Korea DPR	151
10	Earthquake, February	Papua New Guinea	145

[a]Used with permission from CRED, 2019 (Centre for Research on the Epidemiology of Disasters (CRED) and United Nations Office for Disaster Risk Reduction (UNISDR) 2019)

common preventable diseases after a natural disaster but still could be a problem in mass groups that are not completely or appropriately immunized. After the 2010 Haiti earthquake, there were increased cases of diarrhea, cholera, measles, and tetanus in children months after the earthquake despite some level of vaccination (Sirbaugh and DiRocco 2012).

Children are at risk for various environmental emergencies. Austere environments will impact children greatly. Heat exposure coupled with minimal access to drinkable water may lead to severe dehydration. Exposure to the cold may lead to frostbite or hypothermia. Children are at risk for carbon monoxide toxicity due to generator use or natural gas poisoning due to disrupted gas lines. There is always a risk for thermal injury due to the use of candles and other flame sources. Exposure

Table 5.9 Death Toll by disaster type (2018 vs. average twenty-first century)[a]

Event	2018	Average (2000–2017)
Drought	0	1361
Earthquake	4321	46,173
Extreme temperature	536	10,414
Flood	2859	5424
Landslide	282	929
Mass movement (dry)	17	20
Storm	1593	12,722
Volcanic activity	878	31
Wildfire	247	71
Total	**10,733**	**77,144**

[a]Used with permission from CRED, 2019 (Centre for Research on the Epidemiology of Disasters (CRED) and United Nations Office for Disaster Risk Reduction (UNISDR) 2019)

Table 5.10 Number of people affected by disaster type (2018 vs. average twenty-first century)[a]

Event	2018	Average (2000–2017)
Drought	9,368,345	58,734,128
Earthquake	1,517,138	6,783,729
Extreme temperature	396,798	6,368,470
Flood	35,385,178	86,696,923
Landslide	54,908	263,831
Mass movement (dry)	0	286
Storm	12,884,845	34,083,106
Volcanic activity	1,908,770	169,308
Wildfire	256,635	19,243
Total	**61,772,617**	**193,312,310**

[a]Used with permission from CRED, 2019 (Centre for Research on the Epidemiology of Disasters (CRED) and United Nations Office for Disaster Risk Reduction (UNISDR) 2019)

to animals (snakes) and insects (spiders) may increase the risk of envenomation. Submersion injury and drowning incidents may escalate. This will be due to lack of supervision of children around storm drains, newly formed bodies of water, or rushing waters of storm diversion systems (Sirbaugh and DiRocco 2012).

Mental health issues are often seen in children after natural disasters. Even though a child may not be injured, they may become "psychiatric casualties" due to the horrific sights they have seen during or after the disaster. Children and adolescents with behavioral or psychiatric problems may experience worsening symptoms and signs due to stress, trauma, disruption of routines, or availability of medications. This is often exacerbated if the parent, guardian, caregiver, or HCP is also

Table 5.11 Total death tolls by year (twenty-first century)[a]

Year	Death toll	Major events (500+ deaths)
2000	9609	
2001	30,844	Gujarat Earthquake
2002	12,124	
2003	109,827	Bam Earthquake, European Heatwave
2004	242,765	Indian Ocean Earthquake
2005	88,673	Kashmir Earthquake
2006	24,239	Java Earthquake
2007	16,960	
2008	235,256	Cyclone Nargis, Sichuan Earthquake
2009	10,672	
2010	297,140	Haiti Earthquake, Russian Heatwave, Somalia Drought
2011	51,434	Japan Earthquake
2012	10,319	
2013	21,859	North India Floods, Typhoon Haiyan
2014	7993	
2015	22,774	Nepal Earthquake
2016	8512	
2017	9734	
2018	10,733	
Total	**1,221,465**	

[a]Used with permission from CRED, 2019 (Centre for Research on the Epidemiology of Disasters (CRED) and United Nations Office for Disaster Risk Reduction (UNISDR) 2019)

having difficulty coping with the stress of the disaster. In general, the most common mental health problem in children is a post-traumatic stress disorder. However, separation anxiety, obsessive-compulsive symptoms, and severe stranger anxiety can also be seen in children after a traumatic event (Sirbaugh and DiRocco 2012). See Chap. 12 for more detailed information.

5.5 Man-Made Disasters

5.5.1 Terrorism

Terrorism impacts children and families all around the world (Tables 5.12 and 5.13). After the events of 9/11, much attention has been given to the possibility of another mass casualty act of terrorism, especially with weapons of mass destruction, that include chemical, biological, nuclear, radiological, and explosive devices (CBNRE), or other forms of violence such as active shooter incidents and mass shootings

Table 5.12 Terrorist and criminal attacks targeting children[a]

Date	Type	Location	Method and target	Child casualties		Total casualties	
				Killed	Injured	Killed	Injured
18 May 1927	Criminal	Bath, Michigan, US	Bombing of school	41	55	46	58
11 April 1956	**Terrorism**	Shafrir, Israel	Shooting attack on synagogue	3	5	4	5
15 September 1959	Criminal	Houston, Texas US	Suicide bombing of school	3	17	6	18
15 September 1963	**Terrorism**	Birmingham, Alabama US	Bombing of church	4	2+	4	23
18 March 1968	**Terrorism**	Negev desert, Israel	Landmine attack on school bus	2	28	2	28
22 May 1970	**Terrorism**	Avivim, Israel	Rocket attack on school bus	9	19	12	19
11 April 1974	**Terrorism**	Qiryat Shemona, Israel	Shooting attack on residential building	8	?	18	16
15 April 1974	**Terrorism**	Ma'alot, Israel	Hostage taking	20	66+	27	134
8 February 1975	**Terrorism**	El Arish, Sinai	Bombing	1	3	1	3
13 November 1975	**Terrorism**	Jerusalem, Israel	Bombing outside of ice cream shop	6	?	6	42
3 February 1976	**Terrorism**	Djibouti	Hostage taking of school bus	1	0	36	0
26 Jan-13 Feb 1978	**Terrorism**	Maastricht, Netherlands, and Bremen, West Germany	Poisoning of Israeli citrus products	0	5	0	13
11 March 1978	**Terrorism**	Tel Aviv, Israel	Shooting attack on beach and hostage taking on bus	13	?	51	72
20 August 1978	**Terrorism**	Abadan, Iran	Arson of movie theater	Many	0	477	10
18 November 1978	Criminal	Jonestown, Guyana	Poisoning of children during mass suicide by cult members	276	0	918	11
7 April 1980	**Terrorism**	Misgav Am Kibbutz, Israel	Hostage taking at children's dormitory	1	4	8	16
2 June 1980	**Terrorism**	Hebron, West Bank	Four bombings, including grenade attack near elementary school	0	4	0	11
2 August 1980	**Terrorism**	Bologna, Italy	Bombing at railway station	8	?	85	300
16 May 1986	**Terrorism**	Cokeville, Wyoming, US	Hostage taking and bombing of school	0	70	2	79
2 June 1988	**Terrorism**	Jerusalem, Israel	Attack in park	1	0	1	0
2 October 1988	**Terrorism**	Jerusalem, Israel	Explosion of bomb hidden in loaf of bread	0	3	0	3

(continued)

Table 5.12 (continued)

Date	Type	Location	Method and target	Child casualties		Total casualties	
				Killed	Injured	Killed	Injured
17 January 1989	Criminal	Stockton, California, US	Suicide shooting attack at school	5	29	6	30
18 April 1989	Criminal	Indianapolis, Indiana, US	Explosion of bomb hidden in toothpaste tube	0	1	0	1
17 March 1992	**Terrorism**	Buenos Aires, Argentina	Suicide car bombing at Israeli embassy	?	?	29	252
1 May 1992	Criminal	Olivehurt, California, US	Shooting at high school	3	9	4	10
19 April 1995	**Terrorism**	Oklahoma City, OK, US	Car bombing of federal office building	19	?	169	675
4 March 1996	**Terrorism**	Tel Aviv, Israel	Suicide bombing at Dizengoff Center	5	?	14	163
13 March 1996	Criminal	Dunblane, Scotland, UK	Suicide shooting attack at school	16	10	18	12
16 November 1996	**Terrorism**	Kasiysk, Dagestan, Russia	Bombing of Russian border guard housing	20	?	67	?
22 July 1997	**Terrorism**	Israel	Terrorist drove car into a group of teenagers, then exited and began attacking with a knife	?	?	1	12
24 March 1998	Criminal	Jonesboro, Arkansas, US	Shooting attack at school	4	9	5	11
6 May 1998	**Terrorism**	Jerusalem, Israel	Stabbing attack	1	0	1	0
21 May 1998	Criminal	Springfield, Oregon, US	Shooting attacks at residence and high school	2	25	4	25
29 October 1998	**Terrorism**	Gus Katif, Gaza	Attempted bombing of school bus	0	3	2	8
29 October 1998	Criminal	Goteborg, Sweden	Arson attack on dance hall	49	?	63	?
20 April 1999	Criminal	Littleton, Colorado, US	Suicide shooting attack at school	12	25	15	27
15 September 1999	Criminal	Fort Worth, Texas, US	Shooting attack at church service	4	4	8	8
20 November 2000	**Terrorism**	Gaza	Bombing of school bus	0	5	2	9
31 December 2000	**Terrorism**	Ramallah, West Bank	Shooting attack on civilian car	0	5	2	5
26 March 2001	Criminal	Machakos, Kenya	Arson of secondary school	67	19	67	19
5 June 2001	**Terrorism**	Shiloh, Israel	Stoning attack on car	1	0	1	0
8 June 2001	Criminal	Ikeda, Osaka, Japan	Knife attack on school	8	13	8	15
4 September 2001	**Terrorism**	Jerusalem, Israel	Attempted suicide bombing at school	0	?	1	20

9 September 2001	**Terrorism**	Jerusalem, Israel	Suicide bombing at pizzeria	7	5+	15	130
1 December 2001	**Terrorism**	Jerusalem, Israel	Two suicide bombings in pedestrian mall	5	?	13	188
2 March 2002	**Terrorism**	Jerusalem, Israel	Suicide bombing outside synagogue	7	1+	11	50
26 April 2002	Criminal	Erfurt, Germany	Shooting attack in high school	2	?	17	?
9 May 2002	**Terrorism**	Kaspiysk, Russia	Bombing at parade	17	31	43	151
28 May 2002	**Terrorism**	Itamar, Israel	Shooting attack on school	3	2	4	2
3 June 2002	**Terrorism**	Thailand	Shooting attack on school bus	2	15	2	15
20 June 2002	**Terrorism**	Itamar, West Bank	Shooting attack	3	2	7	4
23 September 2002	**Terrorism**	Hebron, West Bank	Shooting attack	0	3	1	3
7 October 2002	**Terrorism**	Bowie, Maryland, US	Shooting attack by Beltway sniper	0	1	0	1
29 October 2002	**Terrorism**	Hermesh, West Bank	Shooting attack	2	?	4	2
11 November 2002	Criminal	Changde, P.R. China	Poisoning of food at high school	0	Many	0	193
21 November 2002	**Terrorism**	Jerusalem, Israel	Suicide bombing on bus	4	?	12	50
25 November 2002	Criminal	Zhanjiang City, Guangdong, P.R. China	Poisoning of food at kindergarten school	0	70	0	72
26 April 2003	Criminal	Erfurt, Germany	Shooting attack in high school	2	?	17	1
5 March 2003	**Terrorism**	Haifa, Israel	Suicide bombing on bus	9	?	18	53
10 August 2003	**Terrorism**	Near Lebanon border	Antiaircraft rocket fired from across Lebanon border	1	4	1	4
19 August 2003	**Terrorism**	Jerusalem, Israel	Suicide bombing on bus	8	?	24	133
7 July 2003	**Terrorism**	Moshav Kfar Yavetz, Israel	Suicide bombing at house	0	3	2	3
2 March 2004	**Terrorism**	Karbala, Iraq	Suicide bombings at shrines	15	?	121	122
21 April 2004	**Terrorism**	Basra, Iraq	Car bombings at police stations and police academy	13	?	74	100
2 May 2004	**Terrorism**	Gush Katif, Gaza	Shooting attack on vehicle	4	0	5	3
28 June 2004	**Terrorism**	Sderot, Gaza	Rocket attack on nursery school	1	?	2	11
4 August 2004	Criminal	Beijing, P.R. China	Knife attack at kindergarten school	1	14	1	17
1–3 Sept 2004	**Terrorism**	Beslan, Russia	Hostage taking and bombing at school	156	337	366	747

(continued)

Table 5.12 (continued)

Date	Type	Location	Method and target	Child casualties		Total casualties	
				Killed	Injured	Killed	Injured
30 September 2004	**Terrorism**	Baghdad, Iraq	Car bombing at public ceremony	35	72	42	69
25 November 2004	Criminal	Ruzhou, Henan, P.R. China	Knife attack at high school dormitory	8	4	8	4
28 February 2005	**Terrorism**	Hilla, Babil, Iraq	Car bombing outside medical clinic	8	?	135	130
21 March 2005	Criminal	Red Lake, Minnesota, US	Shooting attack at high school	6	7	10	7
4 April 2005	Criminal	Zhanjiang, Guangdong, P.R. China	Knife attack at middle school	0	8	0	8
23 April 2005	**Terrorism**	Pakhapani, Rolpa, Nepal	Bombing	5	3	5	3
10 June 2005	Criminal	Hikari, Yamaguchi, Japan	Bomb attack at high school	0	56	0	58
16 June 2005	Criminal	Siem Reap, Cambodia	Hostage taking at elementary school	1	0	1	0
24 June 2005	**Terrorism**	Beit Hagai, West Bank		2	?	2	3
13 July 2005	**Terrorism**	Baghdad, Iraq	Car bombing of gathered children	24	20	27	50
29 September 2005	**Terrorism**	Balad, Salah Al-Din, Iraq	Three car bombings	25	?	95	101
12 October 2005	Criminal	Guangde, Anhui, P.R. China	Shooting at primary school	0	16	0	18
29 November 2005	**Terrorism**	Santa Cruz, California, US	Incendiary bombings of homes	0	2	0	4
15 June 2006	**Terrorism**	Kebithigollewa, Sri Lanka	Mine explosion against bus	15	?	64	80
25 June 2006	**Terrorism**	Beitar Illit, West Bank	Teenager kidnapped and killed	1	0	1	0
2 August 2006	**Terrorism**	Baghdad, Iraq	Explosion of bombs buried in soccer field	12	?	12	?
27 September 2006	Criminal	Bailey, Colorado, US	Hostage taking at school	1	?	2	?
2 October 2006	Criminal	Nickel Mines, PA, US	Hostage taking and shooting at school	5	5	6	5
6 October 2006	**Terrorism**	Basauti, Kailai, Nepal	Bombing of village development committee area	3	0	3	5
3 December 2006	**Terrorism**	Baghdad, Iraq	Mortar attack on school	0	10	0	10
28 January 2007	**Terrorism**	Baghdad, Iraq	Mortar attack on school	5	20	5	20
28 January 2007	**Terrorism**	Ramadi, Iraq	Bombing attack near school	2	10	2	10
20 February 2007	**Terrorism**	Taji, Iraq	Bombing of chlorine tanker truck near restaurant	0	52	9	150

24 February 2007	**Terrorism**	Habbaniya, Al-Anbar, Iraq	Fuel truck bombing near Sunni mosque	5	?	56	48
16 March 2007	**Terrorism**	Fallujah, Iraq	Suicide bombing with dump truck carrying chlorine tanks	0	7	6	250
2 April 2007	**Terrorism**	Kirkuk, Iraq	Truck bombing of police station next to school	2	50	12	200
6 April 2007	**Terrorism**	Ramadi, Iraq	Suicide truck bombing using chlorine tanks	?	?	35	50
12 June 2007	**Terrorism**	Logar province, Afghanistan	Shooting attack on girls leaving school	2	4	2	4
15 June 2007	**Terrorism**	Tarinkot, Afghanistan	Suicide bombing near school	11	3	11	3
6 August 2007	**Terrorism**	Qubbak, Iraq	Truck bombing of residential area	19	?	28	50
14 August 2007	**Terrorism**	Al-Qataniyah and Al-Adnaniyah, Iraq	Multiple truck bombings in villages	Many	Many	520	1500
12 October 2007	**Terrorism**	Tuz Khurmato, Iraq	Bombing on playground	2	17	2	18
6 November 2007	**Terrorism**	Baghlani-jadid, Afghanistan	Suicide bombing at public ceremony	61	93	77	100
7 November 2007	Criminal	Tuusula, Finland	Shooting attack at high school	6	10	8	10
22 January 2008	**Terrorism**	Ba'qubah, Iraq	Suicide bombing at school	0	17	1	21
6 March 2008	**Terrorism**	Jerusalem, Israel	Shooting attack	7	?	9	11
12 November 2008	**Terrorism**	Kandahar City, Afghanistan	Acid attack on schoolgirls	0	14	0	15
26 December 2008	**Terrorism**	Beit Lahiya, Gaza Strip	Rocket attack	2	3	2	3
28 December 2008	**Terrorism**	Khost, Afghanistan	Suicide car bombing at checkpoint near elementary school	14	?	16	58
13 February 2009	**Terrorism**	Iskandariya, Iraq	Suicide bombing of Shiite pilgrimage	?	28	32	76
2 April 2009	**Terrorism**	Bat Ayin, West Bank	Attack by axe-wielding terrorist	1	1	1	1
26 April 2009	**Terrorism**	Charikar, Kapisa province, Afghanistan	Gas poisoning attack on girls' school	0	40	0	45
10 May 2009	**Terrorism**	Busurungi, D.R.C.	Armed attack on village	25	?	86	24
11 May 2009	**Terrorism**	Charikar, Kapisa province, Afghanistan	Gas poisoning attack on girls' school	0	61	0	62
12 May 2009	**Terrorism**	Afghanistan	Gas poisoning attack on girls' school	0	98	0	104

(continued)

Table 5.12 (continued)

Date	Type	Location	Method and target	Child casualties		Total casualties	
				Killed	Injured	Killed	Injured
9 July 2009	**Terrorism**	Logar province, Afghanistan	Truck bombing near highway	16	?	25	5
8 September 2009	**Terrorism**	Muqdadiyah, Diyala, Iraq	Kidnapping and killing of 10-year-old son of politician	1	0	1	0
25 October 2009	**Terrorism**	Baghdad, Iraq	Twin vehicle bombing of government buildings	20	6+	155	540
28 October 2009	**Terrorism**	Peshawar, Pakistan	Car bombing at marketplace	13	?	118	213
4 December 2009	**Terrorism**	Rawalpindi, Punjab, Pakistan	Attack on mosque with guns, grenades, and suicide bombs	17	?	40	81
7 December 2009	**Terrorism**	Baghdad, Iraq	Bombing of school	6	25	8	41
23 March 2010	Criminal	Fujian, P.R. China	Attack on students at elementary school	9	?	8	?
1 April 2010	Criminal	Fujian, P.R. China	Knife attack on primary school students	0	16	0	17
21 April 2010	**Terrorism**	Kunduz, Afghanistan	Gas poisoning attack on girls' school	0	23	0	23
24 April 2010	**Terrorism**	Kunduz, Afghanistan	Gas poisoning attack on girls' school	0	48	0	51
25 April 2010	**Terrorism**	Kunduz, Afghanistan	Gas poisoning attack on girls' school	0	13	0	13
4 May 2010	**Terrorism**	Kunduz, Afghanistan	Gas poisoning attack on girls' school	0	22	0	25
11 May 2010	**Terrorism**	Kunduz, Afghanistan	Gas poisoning attack on girls' school	0	30	0	30
11 May 2010	**Terrorism**	Kunduz, Afghanistan	Gas poisoning attack on girls' school	0	6	0	6
12 May 2010	Criminal	Shaanxi, P.R. China	Knife attack on kindergarten students	7	?	9	?
13 May 2010	**Terrorism**	Israel	Shooting attack	1	0	1	0
June 2010	**Terrorism**	Afghanistan	Gas poisoning attack on girls' school	0	30	0	30
12 June 2010	**Terrorism**	Ghazni City, Afghanistan	Gas poisoning attack on girls' school	0	60	0	60
3 August 2010	Criminal	Zibo, Shandong, P.R. China	Knife attack at kindergarten school	3	?	4	20
21 August 2010	**Terrorism**	Kabul, Afghanistan	Gas poisoning attack on girls' school	0	23	0	23
25 August 2010	**Terrorism**	Kabul, Afghanistan	Gas poisoning attack on girls' school	0	60	0	74

28 August 2010	**Terrorism**	Kabul, Afghanistan	Gas poisoning attack on girls' school	0	48	0	52
31 August 2010	**Terrorism**	Kabul, Afghanistan	Gas poisoning attack on girls' school	0	74	0	74
22 October 2010	Criminal	Zamboanga City, Philippines	Knife attack at elementary school	1	4	2	6
2010	**Terrorism**	Afghanistan	Gas poisoning attack on girls' school	0	?	0	?
2010	**Terrorism**	Afghanistan	Gas poisoning attack on girls' school	0	?	0	?
2010	**Terrorism**	Afghanistan	Gas poisoning attack on girls' school	0	?	0	?
2010	**Terrorism**	Afghanistan	Gas poisoning attack on girls' school	0	?	0	?
2010	**Terrorism**	Afghanistan	Gas poisoning attack on girls' school	0	?	0	?
11 March 2011	**Terrorism**	Itamar, West Bank	Knife attack on residence	3	0	5	0
11 March 2011	Criminal	Panjwayi district, Afghanistan	Shooting attack on villages by U.S. soldier	9	0	16	0
7 April 2011	Criminal	Rio de Janeiro, Brazil	Shooting attack at elementary school	12	12	13	12
7 April 2011	**Terrorism**	Sa'ad, Israel	Antitank missile attack on school bus	1	0	1	1
17 June 2011	**Terrorism**	Sirkanay, Kunar, Afghanistan	Strike of rocket fired across border from Pakistan	4	?	4	?
19 June 2011	**Terrorism**	Kapisa province, Afghanistan	Rocket strike on school during gunbattle	0	5	1	7
3 July 2011	**Terrorism**	Faryab province, Afghanistan	Grenade attack on high school	0	17	0	25
22 July 2011	**Terrorism**	Utoya and Oslo, Norway	Shooting attack and bombing	50	40?	77	151
13 September 2011	**Terrorism**	Peshawar, Khyber-Pakhtunkhwa, Pakistan	Attack on school bus	4	12	5	18
28 November 2011	Criminal	Kunduz, Afghanistan	Acid attack on family in home	0	3	0	4
31 March 2012	**Terrorism**	Esfandi area, Ghazni province, Afghanistan	Acid attack on children	2	0	2	0
17 April 2012	**Terrorism**	Rustaq district, Takhar province, Afghanistan	Water poisoning attack on girls' school	0	150	0	171

(continued)

Table 5.12 (continued)

Date	Type	Location	Method and target	Child casualties		Total casualties	
				Killed	Injured	Killed	Injured
22 May 2012	**Terrorism**	Taloqan, Takhar province, Afghanistan	Water poisoning attack on girls' school	0	80	0	84
26 May 2012	**Terrorism**	Taloqan, Takhar province, Afghanistan	Poisoning attack on girls' school	0	40	0	40
28 May 2012	**Terrorism**	Takhar province, Afghanistan	Poisoning attack on girls' school	0	121	0	124
2 June 2012	**Terrorism**	Taloqan, Takhar province, Afghanistan	Poisoning attack on girls' school	0	20	0	20
3 June 2012	**Terrorism**	Farkhar district, Takhar province, Afghanistan	Poisoning attack on girls' school	0	65	0	65
5 June 2012	**Terrorism**	Rustaq district, Takhar province, Afghanistan	Poisoning attack on girls' school	0	60	0	60
June 2012	**Terrorism**	Shirin Hazara, Bamyan province, Afghanistan	Gas poisoning attack on girls' school	0	2+	0	2+
19 June 2012	**Terrorism**	Bamyan province, Afghanistan	Gas poisoning attack on girls' school	0	47	0	47
22 June 2012	**Terrorism**	Sar-e-Pul province, Afghanistan	Poisoning attack on girls' high school	0	118	0	118
23 June 2012	**Terrorism**	Sar-e-Pul, Sar-e-Pul province, Afghanistan	Poisoning attack on girls' school	0	94	0	94
25 June 2012	**Terrorism**	Sar-e-Pul province, Afghanistan	Poisoning attack on girls' high school	0	90	0	90
30 June 2012	**Terrorism**	Sar-e-Pul, Sar-e-Pul province, Afghanistan	Poisoning attack on girls' school	0	53	0	53
2 July 2012	**Terrorism**	Sheberghan province, Afghanistan	Poisoning attack on girls' school	0	255	0	255

July 2012	**Terrorism**	Jawzjan province, Afghanistan	Poisoning attack on girls' school	0	100	0	100
7 July 2012	**Terrorism**	Jawzjan province, Afghanistan	Poisoning attack on girls' school	0	60	0	60
9 October 2012	**Terrorism**	Mingora, Pakistan	Shooting attack on school bus	0	3	0	3
26 October 2012	**Terrorism**	Maimana, Faryab, Afghanistan	Suicide bombing by 15-year-old	6	4	40	59
15 November 2012	**Terrorism**	Kiryat Malachi, Israel	Rocket attack from Gaza on apartment building	0	4	3	4
14 December 2012	Criminal	Chengping, Henan, P.R. China	Knife attack on children outside primary school	0	22	0	23
14 December 2012	Criminal	Newtown, Connecticut, US	Shooting attack on primary school	20	0	28	3
18 March 2013	**Terrorism**	Maiduguri, Borno, Nigeria	Armed attack on three schools	0	3	3	3
15 April 2013	**Terrorism**	Boston, Massachusetts, US	Two bombings at Boston Marathon	1	14	3	264
18 April 2013	**Terrorism**	Taloqan, Takhar, Afghanistan	Poisoning attack on girls' school	0	18	0	18
21 April 2013	**Terrorism**	Taloqan, Takhar, Afghanistan	Poisoning attack on girls' school	0	74	0	74
1 May 2013	**Terrorism**	Kabul, Afghanistan	Poisoning attack on girls' school	0	150	0	150
1 May 2013	**Terrorism**	Makhachkala, Dagestan, Russia	Bombing at shopping mall	2	0	2	2
14 May 2013	**Terrorism**	Balkh, Afghanistan	Poisoning attack on girls' school	0	150	0	150
15 May 2013	**Terrorism**	Faryab, Afghanistan	Poisoning attack on girls' school	0	80	0	80
1 June 2013	**Terrorism**	Behsud, Afghanistan	Poisoning attack on girls' school	0	22	0	22
1 June 2013	**Terrorism**	Maimana, Faryab, Afghanistan	Poisoning attack on girls' school	0	77	0	77
3 June 2013	**Terrorism**	Paktika, Afghanistan	Suicide bombing targeting school children on lunch break	10	16	14	26
16 June 2013	**Terrorism**	Damaturu, Yobe, Nigeria	Attack on school	7	?	9	?

(continued)

Table 5.12 (continued)

Date	Type	Location	Method and target	Child casualties		Total casualties	
				Killed	Injured	Killed	Injured
17 June 2013	**Terrorism**	Maiduguri, Borno, Nigeria	Attack on school	9	?	9	?
30 June 2013	**Terrorism**	Baghdad, Iraq	Bombing at soccer game	10	?	12	?
6 July 2013	**Terrorism**	Mamudo, Yobe, Nigeria	Attack on boarding school	41	?	42	?
9 July 2013	**Terrorism**	Herat, Afghanistan	Roadside bombing	4	?	17	?
19 November 2013	**Terrorism**	Khairkot, Paktika, Afghanistan	Roadside bombing	7	3	7	3
20 January 2014	**Terrorism**	Rawalpindi, Pakistan	Suicide bombing at market	3	?	14	29
2 March 2014	**Terrorism**	Maiduguri, Borno, Nigeria	Truck bombing in market	Many	?	50	?
8 April 2014	**Terrorism**	Sibi, Pakistan	Bombing at market	5	?	17	46
14 April 2014	**Terrorism**	Chibok, Borno, Nigeria	Attack and kidnapping at girls' boarding school	?	276	1	276
28 April 2014	**Terrorism**	Karachi, Sindh, Pakistan	Grenade attack on seminary	3	11	3	11
12 June 2014	**Terrorism**	West bank	Kidnapping and killing of teenagers	3	0	3	0
2 July 2014	**Terrorism**	West Bank	Kidnapping and killing of teenager	1	0	1	0
6 September 2014	Criminal	Mediterranean Sea off Malta	Ramming and sinking of refugee ship	100	1	450	8
22 October 2014	**Terrorism**	Jerusalem, Israel	Vehicle driven into crowd at rail station	1	?	3	13
16 December 2014	**Terrorism**	Peshawar, Pakistan	Shooting/bomb attack on school	132	121	148	124
16 December 2014	**Terrorism**	Radaa, Bayda, Yemen	Two bombings, once striking a school bus	20	?	31	12
3–4 January 2015	**Terrorism**	Baga, Borno, Nigeria	Armed attack and arson destroying most of Baga	?	?	700	300
4 February 2015	**Terrorism**	Fotokol, Cameroon	Armed attack on town	?	?	90	500
24 March 2015	Criminal	Prads-Haute-Bleone, Alpes-de-Haute-Provence, France	Intentional crash of airliner by copilot	17	0	150	0
13 July 2015	**Terrorism**	Camp Chapman, Khost, Afghanistan	Suicide car bombing at checkpoint near Camp Chapman	12	?	33	10

2 October 2015	**Terrorism**	Maiduguri, Borno, Nigeria	Five suicide bombings at a mosque and home of local leader; the five bombers were girls aged 9–15; three bombers attacked the mosque, one ran into a nearby bush, and one attacked the home	5	?	15	36
10 October 2015	**Terrorism**	N'Djamena, Chad	Five suicide bombings, multiple each at a marketplace where 16 were killed and at a refugee camp where 22 were killed	?	14	41	50
30 January 2016	**Terrorism**	Dalori, Borno, Nigeria	Attack on village including suicide bombings by three females, shootings, and firebombing of huts	Many	?	86	62
31 January 2016	**Terrorism**	Sayyida, Zeinab, Syria	Bombings at a bus station and a military headquarters	5	?	71	99
27 March 2016	**Terrorism**	Lahore, Pakistan	Suicide bombing attack on amusement park	29	?	72	200
14 July 2016	**Terrorism**	Nice, France	Vehicular attack on crowds celebrating Bastille Day; truck was driven down a 2 km stretch of crowded boardwalk	10	54	87	434
20 August 2016	**Terrorism**	Gaziantep, Turkey	ISIS suicide bombing of wedding	34	?	57	90
16 February 2017	**Terrorism**	Sehwan, Pakistan	Suicide bombing at Lal Shahbaz Qalandar, a Sufi shrine	9	?	88	343
11 March 2017	**Terrorism**	Damascus, Syria	Bombings targeting Shia pilgrims	8	?	74	120
15 April 2017	**Terrorism**	Rashindin, Syria	Suicide car bombing targeting buses carrying refugees	80	?	126	100
25 April 2017	**Terrorism**	Godar district, Parachinar, Pakistan	Passenger van struck by landmine explosion	4	?	14	9
2 May 2017	**Terrorism**	Al-Lataminah, Hama, Syria	?	5	?	6	?

(continued)

Table 5.12 (continued)

Date	Type	Location	Method and target	Child casualties		Total casualties	
				Killed	Injured	Killed	Injured
22 May 2017	**Terrorism**	Manchester, England, UK	Suicide bombing outside concert at Manchester Arena	7	16	23	119
5 October 2017	**Terrorism**	Jhal Magsi district, Pakistan	Suicide bombing at Shiite shrine	5	?	25	20
31 October 2017	**Terrorism**	New York City, New York, US	Vehicular attack on pedestrians, bikers, and a school bus	0	4	8	12
4 November 2017	**Terrorism**	Deir ez-Zor, Syria	Suicide car bombing at refugee center; most victims were women and children	Many	Many	100	140
5 November 2017	Criminal	Sutherland Springs, Texas, US	Shooting attack on church during worship service	9	4	27	30
17 November 2017	**Terrorism**	Deir ez-Zor, Syria	Car bombing at rally	12	?	26	30
23 November 2017	**Terrorism**	Bir al-Abed, Sinai, Egypt	Attack on mosque with bombs followed by shooting attack on fleeing survivors	27	?	305	128

[a]The table describes terrorist and criminal acts targeting children from May 1927 to December 2017. It includes the following types of incidents: terrorist attacks in which the targets were preferentially children; attempted terrorist attacks preferentially targeting children; terrorist attacks which produced very high casualties among children; and nonterrorist criminal acts which are relevant in terms of methodology and child victims. The full table, description of incidents and data resources, can be viewed at http://www.johnstonsarchive.net/terrorism/wrjp39ch.html. Used with permission from Wm. Robert Johnston PhD (2017)

Table 5.13 Summary of historical attacks using chemical or biological weapons[a]

Date	Location	Attacker	Agent	Affected population	Casualties	Description
21–27 October 2016	Near Mosul, Iraq	Islamic State militants	Sulfur	Civilians, soldiers	**2 killed**, 1500 injured	Sulfur mine set on fire, producing widespread sulfur dioxide plumes
8 March 2016	Taza, Kirkuk, Iraq	Islamic State	Blistering agent	Civilians	**1 killed**, 600 injured	Attack on town; fatality was 3-year-old child
23 January 2015	Between Mosul, Iraq, and Syrian Border	Islamic State militants	Chlorine	Kurdish soldiers	Approximately 30 injured	Truck bomb with chlorine-filled tanks against troops
September–October 2014	Duluiya and Balad, Iraq	Islamic State militants	Chlorine, possibly mustard gas	Iraqi and Shiite soldiers	40 injured	Bombs filled with chlorine-filled cylinders used against defending troops
27 March–22 April 2014	Syria--Damascus, Kafr Zita in Hama, and Talmenes in Idlib	Syrian military suspected	Chlorine, others	Civilians	**104 killed**, 200 injured	Chlorine bombs used on civilians in two towns
21 August 2013	Damascus suburbs, Syria	Syrian military	Sarin nerve gas?	Civilian in urban areas	**1429 killed (including 426 children)**, 2200 injured	Rockets with chemical agents fired at about 12 areas in suburbs south and east of Damascus, targeting rebel-held areas
19 March–13 April 2013	Syria--Damascus, Al-Otaybeh, Khan al-Assal, Adra, Aleppo, Sheikh Maqsoud, and Saraqeb	Syrian military?	Multiple chemical agents?	rebel soldiers and civilians	**At least 44 killed**, 76 injured	Multiple attacks, mostly blamed on Syrian government; Syrian government accuses rebels of the attacks

(continued)

Table 5.13 (continued)

Date	Location	Attacker	Agent	Affected population	Casualties	Description
April 2012–June 2013	Afghanistan—Takhar province (American College of Surgeons Committee on Trauma, American College of Emergency Physicians, National Association of EMS Physicians, Pediatric Equipment Guidelines Committee Emergency Medical Services for Children (EMSC) Partnership for Children Stakeholder Group, American Academy of Pediatrics 2009), Sar-e-Pul province (American Academy of Pediatrics (AAP) 2003), others	Islamist terrorists	Pesticides?	Schoolchildren	1952 injured (including 1924 children)	23 poison attacks on girls' schools, some cases of water poisoning
March 2012–April 2013	Afghanistan	Islamist terrorists	Rat poison?	Police, other civilians	**53 killed**, 40 injured	9 attacks involving poisoning of food at police stations/ academies
April–August 2010	Afghanistan—Kabul (American Academy of Pediatrics and American Heart Association et al. 2016), Kunduz (American Academy of Pediatrics (AAP) 2003), others	Islamist terrorists	Pesticides?	Schoolchildren	672 injured (including 636 children)	20 gas attacks on girls' schools
11 March 2007	Iraq	Islamist terrorists	Mustard gas	U.S. soldiers	2 injured	Failed improvised explosive device using chemical weapon artillery shells

October 2006–June 2007	Iraq cities--Ramadi (American Academy of Pediatrics and American Heart Association et al. 2016), Baghdad (Advanced Hazmat Life Support (AHLS) 2003), Falluja (Advanced Hazmat Life Support (AHLS) 2003), others	Islamist terrorists	Chlorine	Civilian targets	**115 killed**, 854 injured (including 85 children)	15 car/truck bombings with chlorine tanks used; most fatalities were from the explosions, most injuries from the chemical releases
8 October 2006	Numaniyah, Iraq	Islamist terrorists	Poison	Policemen	**7 killed**, 700 injured	Poisoning of food at meal on police base (unconfirmed)
25 September 2006	Baghdad, Iraq	Islamist terrorists	Mustard gas	U.S. soldiers	2 injured	Improvised explosive device using chemical weapon artillery shells
15 May 2004	Baghdad, Iraq	Islamist terrorists	Sarin nerve gas	U.S. soldiers	2 injured	Failed improvised explosive device using chemical weapon artillery shell near Baghdad airport
24 June–July 2003	Near Mosul, Iraq	Islamist terrorists	Sulfur	Civilians, soldiers	At least 41 soldiers injured	Sulfur stockpiles at mine set on fire, producing widespread sulfur dioxide plumes
11 November 2002	Changde, PR China	Criminal	Poison	Schoolchildren	193 injured (mostly children)	Poisoning of food at high school

(continued)

Table 5.13 (continued)

Date	Location	Attacker	Agent	Affected population	Casualties	Description
26 October 2002	Moscow, Russia	Russian soldiers	Fentanyl incapacitating agent	Terrorists and civilian hostages	**124 killed**, 501 injured	Chechen terrorists took 800 hostages at Moscow theater, 23 Oct; Russian forces used fentanyl when storming the theater and killing all the terrorists on 26 Oct, but many hostages were killed or injured by the gas
18 September–9 October 2001	United States--Washington, DC, New York City, NY, others	Bruce Ivins?	Anthrax	Government and civilian media individuals; postal employees and customer	**5 killed**, 17 injured	Anthrax-laced letters mailed to federal officials in Washington DC and new media offices in multiple locations; many casualties among postal workers
20 March 1995	Tokyo, Japan	Aum Shinrikyo cult	Sarin nerve gas	Tokyo subway	**12 killed**, 5511 injured	Nerve gas releases in subway; many permanent injuries
28 June 1994	Matsumoto, Japan	Aum Shinrikyo cult	Sarin nerve gas	Civilians	**7 killed**, 270 injured	Overnight release of nerve gas in city
21 January 1994	Ormancik, Turkey	Terrorists	Chemical agent	Civilians	**16 killed**	Attack on village using chemical grenades

16 March 1988	Halabja, Iraq	Iraqi military	Cyanide, mustard gas, nerve agents	Iraqi Kurdish civilians	**5000 killed**, 8000 injured	Use of chemical agents against civilians in village; additional use of agents by Iranian military possible
6 September 1987	Zamboanga City, Philippines	Terrorists	Poison	Policemen	**19 killed**, 140 injured	Water poisoning with pesticide at constabulary
1987–August 1988	Iraq–Iran	Iranian military	Mustard gas, cyanide	Iraqi soldiers	**?,?**	Some use
2–3 December 1984	Bhopal, India	Accidental	Methyl isocynate gas	Civilians	**3787 killed**, 558,125 injured (including 200,000 children)	Accidental release from pesticide plant with gas plume blown across city of Bhopal
9–19 September 1984	The Dalles, Oregon, United States	Bhadwan Shree Rajneesh cult	Salmonella	Civilian restaurants	751 injured	Food poisoning in several restaurants; was experiment in preparation to interfere with upcoming election
August 1983–July 1988	Iraq–Iran	Iraqi military	Chemical agents	Iranian soldiers and civilians	**21,000 killed**, 92,000 injured	Extensive use against soldiers and civilians

(continued)

Table 5.13 (continued)

Date	Location	Attacker	Agent	Affected population	Casualties	Description
June 1979-mid 1981	Afghanistan	Soviet and Afghan militaries	Multiple chemical agents	Civilians and rebel soldiers	**3042 killed**	Used in at least 47 instances in the invasion of Afghanistan
April 1979	suburbs southeast of Sverdlovsk, USSR	Accidental	Anthrax	Civilians	**68 killed**, 300 injured	Accidental release from bioweapons production facility caused anthrax outbreak in Sverdlovsk; cause of outbreak was denied by Soviet government

[a]The table summarizes known historical instances of the use of chemical or biological weapons, in reverse chronological order, 1979–2016. A full list of all chemical or biological events since 1900 (with references and sources of data) can be found at http://www.johnstonsarchive.net/terrorism/chembioattacks.html. Although there were some earlier instances of chemical/biological warfare prior to 1900, these instances were generally of very limited effectiveness. Note that some incidents are disputed, and casualty figures in some cases are very uncertain. The following events are included: **use in warfare:** multiple attacks within a war are grouped together; **use by terrorists:** includes attacks with larger numbers of casualties; and **other:** several criminal incidents and accidental chemical releases are included because of their significance. Used with permission from Wm. Robert Johnston PhD (2017)

(Jacobson and Severin 2012). Since then, other incidents, both foreign and domestic, have involved children and complicates the concept of and the response to terrorism. Johnston (2017) said it best in his review of *Terrorist and Criminal Attacks Targeting Children*: "One of the more accepted defining characteristics of terrorism is that it targets noncombatants including men, women, and children. However, terrorist attacks specifically targeting children over other noncombatants are uncommon. This is for the same reason that most terrorists have historically avoided mass casualty terrorism: the shock value is so great that such attacks erode support for the terrorists' political objectives. The 9/11 attacks represent an increasing trend in mass casualty terrorism. At the same time, policymakers are examining this evolving threat, they must increasingly consider the threat of terrorist attacks targeting children." Based on historical events, it is clear infants, toddlers, children, and adolescents have been victims of terrorism. This global trend of terrorists targeting children seems to be escalating (Johnston 2017). Therefore, it is imperative to understand terrorism and ways it impacts the children and families served by the health care community.

Combs (2018) defines *terrorism* as "an act of violence perpetrated on innocent civilian noncombatants in order to evoke fear in an audience". However, she goes on to argue that to become an operational definition, there must also be the addition of a "political purpose" of the violent act. Therefore, "terrorism, then, is an act composed of at least four crucial elements: 1) it is an act of violence, 2) it has a political motive or goal, 3) it is perpetrated against civilian noncombatants, and 4) it is staged to be played before an audience whose reaction of fear and terror is the desired result."(Combs 2018).

There are different typologies of terrorism. At least five types of terror violence have been suggested by Feliks Gross: "*Mass terror* is terror by a state, where the regime coerces the opposition in the population, whether organized or unorganized, sometimes in an institutionalized manner. *Dynastic assassination* is an attack on a head of state or a ruling elite. *Random terror* involves the placing of explosives where people gather (such as post offices, railroads, and cafes) to destroy whoever happens to be there. *Focused random terror* restricts the placing of explosives, for example to where significant agents of oppression are likely to gather. Finally, *tactical terror* is directed solely against the ruling government as a part of a 'broad revolutionary strategic plan'" (Combs 2018). An additional typology offered is "*lone wolf terror* which involves someone who commits violent acts in support of some group, movement, or ideology, but who does stand alone, outside of any command structure and without material assistance from any group" (Combs 2018).

Martin (2017) reviews eight different terrorism typologies in the ever shifting, multifaceted world of modern terrorism. *The New Terrorism* "is characterized by the threat of mass casualty attacks from dissident terrorist organizations, new and creative configurations, transnational religious solidarity, and redefined moral justifications for political violence" (Martin 2017). *State Terrorism* is "committed by governments against perceived enemies and can be directed externally against adversaries in the international domain or internally against domestic enemies" (Martin 2017). *Dissident Terrorism* is "committed by nonstate movements and

groups against governments, ethno-national groups, religious groups, and other perceived enemies" (Martin 2017). *Religious Terrorism* is "motivated by an absolute belief that an otherworldly power has sanctioned and commanded the application of terrorist violence for the greater glory of the faith…[it] is usually conducted in defense of what believers consider to be the one true faith" (Martin 2017). *Ideological Terrorism* is "motivated by political systems of belief (ideologies), which champion the self-perceived inherent rights of a particular group or interest in opposition to another group or interest. The system of belief incorporates theoretical and philosophical justifications for violently asserting the rights of the championed group or interest" (Martin 2017). *International Terrorism* "spills over onto the world's stage. Targets are selected because of their value as symbols of international interests, either within the home country or across state boundaries" (Martin 2017). *Criminal Dissident Terrorism* "is solely profit-driven, and can be some combination of profit and politics. For instance, traditional organized criminals accrue profits to fund their criminal activity and for personal interests, while criminal-political enterprises acquire profits to sustain their movement" (Martin 2017). *Gender-Selective Terrorism* "is directed against an enemy population's men or women because of their gender. Systematic violence is directed against men because of the perceived threat posed by males as potential soldiers or sources of opposition. Systematic violence is directed against women to destroy an enemy group's cultural identity or terrorize the group into submission" (Martin 2017).

The all-hazards National Planning Scenarios are an integral component of DHS's capabilities-based approach to implementing Homeland Security Presidential Directive 8: National Preparedness (HSPD-8). The National Planning Scenarios are planning tools and are representative of the range of potential terrorist and natural disasters and the related impacts that face the nation. The federal interagency community has developed 15 all-hazards planning scenarios for use in national, federal, state, and local homeland security preparedness activities. The objective was to develop a minimum number of credible scenarios to establish the range of response requirements to facilitate disaster planning (DHS 2006) (Table 5.14).

Twelve of the scenarios represent terrorist attacks while three represent natural disasters or naturally occurring epidemics. This ratio reflects the fact that the nation has recurring experience with natural disasters but faces newfound dangers, including the increasing potential for use of weapons of mass destruction by terrorists. The scenarios form the basis for coordinated federal planning, training, exercises, and grant investments needed to prepare for all hazards. DHS employed the scenarios as the basis for a rigorous task analysis of prevention, protection, response, and recovery missions and identification of key tasks that supported the development of essential all-hazards capabilities (United States Department of Homeland Security, Federal Emergency Management Agency 2019) (Table 5.15).

Each of the 15 scenarios follows the same outline to include a detailed scenario description, planning considerations, and implications. For each of the 12 terrorism-related scenarios, FEMA National Preparedness Directorate (NPD) partnered with DHS Office of Intelligence and Analysis (I&A) and other intelligence community and law enforcement experts to develop and validate prevention prequels. The

Table 5.14 DHS national planning scenarios[a]

	Category	Scenario description
Scenario 1	Nuclear Detonation	10-kiloton Improvised Nuclear Device
Scenario 2	Biological Attack	Aerosol Anthrax
Scenario 3	Biological Disease Outbreak	Pandemic Influenza
Scenario 4	Biological Attack	Plague
Scenario 5	Chemical Attack	Blister Agent
Scenario 6	Chemical Attack	Toxic Industrial Chemicals
Scenario 7	Chemical Attack	Nerve Agent
Scenario 8	Chemical Attack	Chlorine Tank Explosion
Scenario 9	Natural Disaster	Major Earthquake
Scenario 10	Natural Disaster	Major Hurricane
Scenario 11	Radiological Attack	Radiological Dispersal Devices
Scenario 12	Explosives Attack	Bombing Using Improvised Explosive Devices
Scenario 13	Biological Attack	Food Contamination
Scenario 14	Biological Attack	Foreign Animal Disease (Foot-and-Mouth Disease)
Scenario 15	Cyber Attack	Cyber Attack Against Critical Internet Related Infrastructures

[a]Reference: (United States Department of Homeland Security 2006)

Table 5.15 DHS FEMA key scenarios and corresponding national planning scenarios[a]

Key scenarios	National planning scenarios
1. Explosives Attack-Bombing Using Improvised Explosive Device	Scenario 12: Explosives Attack-Bombing Using Improvised Explosive Device
2. Nuclear Attack	Scenario 1: Nuclear Detonation-Improvised Nuclear Device
3. Radiological Attack-Radiological Dispersal Device	Scenario 11: Radiological Attack-Radiological Dispersal Device
4. Biological Attack-With annexes for different pathogens	Scenario 2: Biological Attack-Aerosol Anthrax Scenario 4: Biological Attack-Plague Scenario 13: Biological Attack-Food Contamination Scenario 14: Biological Attack-Foreign Animal Disease
5. Chemical Attack-With annexes for different agents	Scenario 5: Chemical Attack-Blister Agent Scenario 6: Chemical Attack-Toxic Industrial Chemicals Scenario 7: Chemical Attack-Nerve Agent Scenario 8: Chemical Attack: Chlorine Tank Explosion
6. Natural Disaster-With annexes for different disasters	Scenario 9: Natural Disaster-Major Earthquake Scenario 10: Natural Disaster-Major Hurricane
7. Cyber Attack	Scenario 15: Cyber Attack
8. Pandemic Influenza	Scenario 3: Biological Disease Outbreak-Pandemic Influenza

[a]References: United States Department of Homeland Security, Federal Emergency Management Agency 2019; United States Department of Homeland Security 2006)

prequels provide an understanding of terrorists' motivation, capability, intent, tactics, techniques and procedures, and technical weapons data. The prequels also provide a credible adversary based on known threats to test the homeland security community's ability to understand and respond to indications and warnings of possible terrorist attacks (United States Department of Homeland Security, Federal Emergency Management Agency 2019).

5.6 Chemical Agents

A chemical agent of terrorism is defined as any chemical substance intended for use in military operations to kill, seriously injure, or incapacitate humans (or animals) through its toxicological effects. Chemicals excluded from this list are riot-control agents, chemical herbicides, and smoke/flame materials. Chemical agents are classified as toxic agents (producing injury or death) or incapacitating agents (producing temporary effects). Toxic agents are further described as nerve agents (anticholinesterases), blood agents (cyanogens), blister agents (vesicants), and lung-damaging agents (choking agents). Incapacitating agents include stimulants, depressants, psychedelics, and deliriants (Banks 2014; Departments of the Army, the Navy, and the Air Force, and Commandant, Marine Corps 1995).

5.6.1 Nerve Agents

Nerve agents are organophosphate anticholinesterase compounds. They are used in various insecticide, industrial, and military applications. Military-grade agents include tabun (GA), sarin (GB), soman (GD), cyclosarin (GF), Venom X (VX), and the Novichok series. These are all major military threats. The only known battlefield use of nerve agents was the Iraq–Iran war. However, other nerve agent incidents, such as the 1995 Tokyo subway attack (sarin), the chemical attacks in Syria (chlorine, sarin, mustard), and the attempted assassination of Sergei Skripal in Salisbury, UK (Novichok), support that civilian threats also exist.

Nerve agents are volatile chemicals and can be released in liquid or vapor form. However, the liquid form can become vapor depending upon its level of volatility (e.g, G-agents are more volatile than VX). The level of toxicity depends on the agent, concentration of the agent, physical form, route and length of exposure, and environmental factors (temperature and wind) (Tables 5.16 and 5.17).

Nerve agents exert their effects by the inhibition of esterase enzymes. Acetylcholinesterase inhibition prevents the hydrolysis of acetylcholine. The clinical result is a cholinergic crisis and subsequent overstimulation of muscarinic and nicotinic receptors throughout the body including the central nervous system. Clinical muscarinic responses include SLUDGE (salivation, lacrimation, urination, defecation, gastrointestinal distress, and emesis) and DUMBELS (diarrhea, urinary incontinence, miosis/muscle fasciculation, bronchorrhea/bronchospasm/bradycardia, emesis, lacrimation, and salivation). Nicotinic responses vary by site. Preganglionic sympathetic nerve stimulation produces mydriasis, tachycardia,

Table 5.16 Comparative nerve agent vapor toxicity[a]

Agent	LCt_{50}	ICt_{50}	MCt_{50}
GA	400	300	2–3
GB	100	75	3
GD	70	Unknown	<1
GF	Unknown	Unknown	<1
VX	50	35	0.04

[a]For this table, one concentration of VX = 50, and one concentration of GB = 100, meaning it would take 2 times more GB to have the same median lethal dose as one concentration of VX (LCt_{50}:median lethal concentration/time; ICt_{50}:median incapacitation concentration/time; MCt_{50}: median first noticeable effect (of miosis) concentration/time) (Banks 2014)

Table 5.17 Comparative median lethal dose values on skin (liquid)[a]

Agent	Amount
GA	100
GB	170
GD	5
GF	3
VX	1

[a]For this table, one dose of VX = 1, and 170 doses of GB = 170, meaning it would take 170 times more GB to have the same median lethal dose as one dose of VX (Banks 2014)

hypertension, and pallor. However, stimulation at the neuromuscular junction leads to muscular fasciculation and cramping, weakness, paralysis, and diaphragmatic weakness. Central nervous system presentations range from anxiety and restlessness to seizures, coma, and death (Banks 2014; Rotenberg and Newmark 2003; Rotenberg 2003b).

Pediatric manifestations (Table 5.19) may vary from the classic clinical responses due to their unique vulnerabilities (Hilmas et al. 2008):

- Children may manifest symptoms earliest and possibly more severe presentations.
- Could be hospitalized for similarly related illnesses and diseases.
- Smaller mass.
- Lower baseline cholinesterase activity.
- Tendency to bronchospasm.
- Pediatric airway and respiratory differences.
- Altered pulmonary compensation.
- Lower reserves of cardiovascular system and fluids.
- Isolated central nervous system signs (stupor, coma).
- Less miosis.
- Vulnerability to seizures and neurotransmitter imbalances (excitability).
- Immature metabolic systems.

Differential diagnoses include upper or lower airway obstruction, bronchiolitis, status asthmaticus, cardiogenic shock, acute gastroenteritis, seizures, and poisonings (carbon monoxide, organophosphates, and cyanide). Diagnostic tests include acetylcholinesterase levels, red blood cell cholinesterase levels, and an arterial blood gas.

Treatment (Tables 5.20 and 5.21) includes decontamination (Reactive Skin Decontamination Lotion® [potassium 2,3-butanedione monoximate], soap and water, and 0.5% hypochlorite solution), supportive care, and administration of nerve agent antidotes (atropine, pralidoxime chloride, and diazepam). Atropine is a competitive antagonist of acetylcholine muscarinic receptors and reverses peripheral muscarinic symptoms. It does not restore function at the neuromuscular junction nicotinic receptors. It does, however, treat early phases of convulsions. Pralidoxime chloride separates the nerve agent from acetylcholinesterase and restores enzymatic function. It also binds free nerve agent. The major goal is to prevent "aging" of the enzyme (e.g., GD). Diazepam provides treatment of nerve agent-induced seizures and prevents secondary neurologic injury. Typically, associated seizures are refractory to other antiepileptic drugs. The antiseizure effect of diazepam is enhanced by atropine (Banks 2014; Cieslak and Henretig 2016; Messele et al. 2018). Potential medical countermeasures include trimedoxime (TMB4), HI-6 (an H-series oxime), obidoxime, "bioscavengers" (butyrylcholinesterase, carboxylesterase, organophosphorus acid anhydride hydrolase, and human serum paraoxonase), novel anticonvulsant drugs, *N*-methyl-D-aspartate (NMDA) receptor antagonists (ketamine, dexanabinol), and common immunosuppressants such as cyclosporine A (Jokanovic 2015; Merrill et al. 2015; National Institutes of Health 2007; United States Department of Health and Human Services 2017). All patients should be observed closely for electroencephalographic changes and neuropsychiatric pathologies. Polyneuropathy, reported after organophosphate insecticide poisoning, has not been reported in humans exposed to nerve agents and has been produced in animals only at unsurvivable doses. The intermediate syndrome has not been reported in humans after nerve agent exposure, nor has it been produced in animals. Muscular necrosis has occurred in animals after high-dose nerve agent exposure but reversed within weeks; it has not been reported in humans (Banks 2014).

5.6.2 Novichok Series

On March 4, 2018, Sergei Skripal, a former Russian double agent, and his daughter, Yulia Skripal, were found unresponsive on a park bench in Salisbury, UK. They were brought to a nearby hospital and treated for signs consistent with a cholinergic crisis due to a nerve agent exposure. Analysis of the Skripals found the presence of a secret nerve agent called Novichok. Further testing found high concentrations of the agent on the front-door handle of his home. One of the investigating police officers, Detective Sergeant Nick Bailey, unknowingly touched the door-handle and also became ill. All three survived due to rapid recognition of the nerve agent exposure by hospital personnel. Four months later, two other people, Dawn Sturgess and

Charlie Rowley, became ill with identical symptoms in the town of Amesbury, 7 miles from Salisbury. They were later confirmed to have high concentrations of Novichok on their hands from a perfume bottle found in a recycling bin. Both were immediately treated, but Dawn Sturgess later died. Charlie Rowley survived. It was believed the discarded perfume bottle contained Novichok and was discarded by the assailants after the attempt on Sergei Skripal. On September 5, 2018, the UK government revealed that their investigation uncovered two suspects from closed circuit television (CCTV) footage near the Skripal's home. The suspects entered the UK on Russian passports using the names Alexander Petrov and Ruslan Boshirov, stayed in a London hotel for 2 days, visited Salisbury briefly, and then returned to Moscow. Minute traces of Novichok were also found in the London hotel where they had stayed. The UK Prime Minister, Teresa May, said that the suspects are thought to be officers from Russia's military intelligence service the Glavnoye Razvedyvatel'noye Upravleniye (GRU), and that this showed that the poisoning was "not a rogue operation" and was "almost certainly" approved at a senior level of the Russian state. The two suspects later appeared on Russian TV denying the accusations and saying they were just "tourists" who had traveled all the way from Moscow to Salisbury just to see the "famous cathedral". However, CCTV of the cathedral area found no evidence of the two men visiting the cathedral, although they were captured on CCTV near the Skripal's home. In a development in September 2018, one of the men was revealed as actually being a Russian intelligence officer named Colonel Anatoliy Chepiga and was a decorated veteran of Russian campaigns in Chechnya and Ukraine. And later in October, the second man was named as Dr. Alexander Mishkin, a naval medical doctor allegedly recruited by the GRU (Chai et al. 2018; May 2018).

Novichok (Новичóк: Russian for "newcomer") is a highly potent nerve agent developed from the Russian classified nerve agent program known as FOLIANT. Almost everything known about these agents is due to a Russian defector, Vil Mirzayanov (2009) who was an analytical chemist at the Russian State Research Institute of Organic Chemistry and Technology (GosNIIOKhT). He has described the details of the Novichok program in his book "*State Secrets: An Insider's Chronicle of the Russian Chemical Weapons Program*". The first three nerve agents of the Novichok series developed in the program were Substance-33, A-230, and A-232 (Table 5.18). They were synthesized as unitary agents, like VX, tabun, soman, and sarin. Unitary means that the chemical structure was produced at its maximum potency. However, the Novichok agents were developed as binary agents: maximum potency when two inert substances are combined together prior to deployment to create the active nerve agent (Cieslak and Henretig 2003). Very little is known about the chemistry of these weaponized organophosphate agents. However, they appear to be more potent than current nerve agents. For example, the LD_{50} of Novichok agents is reported 0.22 μg/kg similar to 2-(dimethylamino)ethyl *N,N*-dimethylphosphoramidofluoridate (VG), a novel fourth generation nerve agent. Furthermore, Novichok-5 is 8× more effective than VX and Novichock-7 is 10× more effective than soman (Cieslak and Henretig 2003; Hoenig 2007).

Clinically, they behave like other organophosphates by binding to acetylcholinesterase preventing the breakdown of acetylcholine thereby leading to a cholinergic

Table 5.18 A list of known Novichok agents attributed to the GosNIIOKhT research program and their status[a]

Agent	Type	Current status
Substance-33	Unitary	Estimated 15,000 tons produced Designated as chemical weapon
A-230	Unitary	Experimental quantities produced Designated as chemical weapon (1990)
A-232	Unitary	Experimental agent Not designated, or officially approved
A-234	Unitary analog of A-232	Unknown
Novichok-5	Binary analog of A-232 8× more effective than VX	Experimental agent Designated as chemical weapon (1989)
Novichok-7	Binary analog of A-234 10× more effective than soman	Experimental agent Not designated
Novichok-#	Binary analog of Substance-33	Adopted as chemical weapon (1990)

[a]VX = Venom X (Cieslak and Henretig 2003)

crisis. There appears to be a similar "aging" process as seen with other nerve agents. In addition, the Novichok agents binding to peripheral sensory nerves distinguishes this class of organophosphates. Prolonged or high-dose exposure results in debilitating peripheral neuropathy. Exposure to these agents is fatal unless aggressively managed (Cieslak and Henretig 2003). Decontamination is essential to prevent ongoing exposure to the patient and medical personnel. Clothing should be removed and quickly placed in a sealed bag (prevents ongoing exposure to the emission of vapors) followed by thorough washing with soap and water. Application of dry bleach powder should be avoided as it may hydrolyze nerve agents into toxic metabolites that can produce ongoing cholinergic effects. Supportive care is essential. Antidote therapy should be given as usual for nerve agents, including atropine, diazepam, and pralidoxime chloride (United States Department of Health and Human Services, Office of the Assistant Secretary for Preparedness and Response, National Library of Medicine 2019; United States Department of Health and Human Services, Chemical Hazards Emergency Medical Management (CHEMM) 2019). Of note, the toxicity of the Novichok agents may not rely on anticholinesterase inhibition. Some have suggested that reactive oximes like potassium 2,3-butanedione monoximate are preferred oximes for antidotal therapy (Cieslak and Henretig 2003).

5.6.3 Blood Agents

Cyanide is a naturally occurring chemical. It can be found in plants and seeds. It is also used in many industrial applications and is a common product of combustion of synthetic materials. Typical cyanogens include hydrogen cyanide (AC) and cyanogen chloride (CK). Low levels of cyanide are detoxified by a natural reaction in the human body using the rhodanese system. There is reversible metabolism with Vitamin B12a to Vitamin B12 (cyanocobalamin). An irreversible reaction occurs with sulfanes to produce thiocyanates and sulfates. The former is excreted via the urinary tract. When cyanide overwhelms this natural process, cyanide binds to

cytochrome oxidase within the mitochondria and disrupts cellular respiration. Cyanide has an affinity for Fe+3 in the cytochrome a3 complex and oxidative phosphorylation is interrupted. Cells can no longer use oxygen to produce ATP and lactic acidosis ensues from resultant anaerobic metabolism.

When inhaled, cyanide produces rapid onset of clinical signs. Findings include transient tachypnea and Kussmaul breathing (from hypoxia of carotid and aortic bodies), hypertension and tachycardia (from hypoxia of aortic body), and neurologic findings such as seizures, muscle rigidity (trismus), opisthotonus, and decerebrate posturing. Other findings include cherry red flush, acute respiratory failure/arrest, bradycardia, dissociative shock, and cardiac arrest. Venous blood samples exhibit a bright red color. Arterial blood gas may demonstrate a metabolic acidosis with an increased anion gap due to lactic acid (Banks 2014; Cieslak and Henretig 2016; Rotenberg 2003a).

Pediatric manifestations (Table 5.19) may vary from the classic clinical responses due to their unique vulnerabilities (Hilmas et al. 2008):

- Thinner integument leading to shorter time from exposure to symptom development.
- Higher vapor density (CK) and concentration accumulation in living zone of children,
- Higher minute ventilation and metabolism.
- Abdominal pain, nausea, restlessness, and giddiness are common early findings.
- Cyanosis mostly noted other than classic cherry red flushing of the skin.
- Resilient with recovery even when just using supportive measures alone.

Differential diagnoses include meningitis, encephalitis, gastroenteritis, ischemic stroke, methemoglobinemia, and poisonings (nerve agents, organophosphates, methanol, hydrogen sulfide, and carbon monoxide). Diagnostic tests include arterial blood gas, lactic acid, and thiocyanate levels.

Treatment (Tables 5.20 and 5.21) includes decontamination, supportive care, and administration of cyanide antidote kit (nitrites and thiosulfate). The nitrites facilitate the production of methemoglobinemia (Fe+3) which attracts cyanide molecules forming cyanmethemoglobin. Amyl nitrite pearls are crushed into gauze and placed over the mouth/nose or in a mask used for bag/mask ventilation. Sodium nitrite is given parenterally and dosed according to the patient's estimated hemoglobin so as to prevent severe methemoglobinemia. Since the formation of cyanmethemoglobin is a reversible reaction, and sodium thiosulfate is given to extract the cyanide. Dosing is also dependent upon estimated hemoglobin. Along with the naturally occurring rhodanese enzymatic system, the irreversible reaction forms thiocyanate. Thiocyanate is water soluble and is excreted harmlessly via the kidneys (Banks 2014; Cieslak and Henretig 2016). Potential medical countermeasures (National Institutes of Health 2007; United States Army Medical Research Institute of Infectious Diseases (USAMRIID) 2014) include hydroxocobalamin, cobinamide (a cobalamin precursor), dicobalt edetate, cyanohydrin-forming compounds (alpha-ketoglutarate and pyruvate), S-substituted crystallized rhodanese, sulfur-containing drugs (*N*-acetylcysteine), and methemoglobin inducers (4-dimethylaminophenol and others).

Table 5.19 Pediatric vulnerabilities and implications for clinical management

	Unique vulnerability in children	Implications and impact from chemical toxicity
Body composition	Larger BSA compared to body mass Lower total body lipid/fat content	Greater dermal absorption Less partitioning of lipid-soluble components
Volume status	More prone to dehydration Chemical agents lead to diarrhea and vomiting	Can be more symptomatic and show signs of severe dehydration
Respiratory	Increased basal metabolic rate compared to greater minute volume	Enhance toxicity via inhalational route
Blood	Limited serum protein binding capacity Greater cutaneous blood flow	Potential for greater amount of free toxicant and greater distribution Greater percutaneous absorption
Skin	Thinner epidermis in preterm infants Greater cutaneous blood flow	Increased toxicity from percutaneous absorption of chemical agents
Organ size and enzymatic function	Larger brain mass Immature renal function Immature hepatic enzymes	Greater CNS exposure Slower elimination of renally cleared toxins, chemicals, and metabolites Decreased metabolic clearance by hepatic phase I and II reactions
Anatomical considerations	Shorter stature means breathing occurs closer to the ground where aerosolized chemical agents settle Smaller airway Greater deposition of fine particles in the upper airway Higher proportion of rapidly growing tissues	Exposure to chemicals can have significant impact on bone marrow and developing CNS Increased airway narrowing from chemical agent-induced secretions Mustard significantly affects rapidly growing tissue
Central nervous system	Higher BBB permeability Rapidly growing CNS	Increased risk of CNS damage
Miscellaneous	Immature cognitive function Unable to flee emergency Immature coping mechanisms	Inability to discern threat, follow directions, and protect self High risk for developing PTSD

BBB blood-brain barrier, *BSA* body surface area, *CNS* central nervous system, *PTSD* post-traumatic stress disorder (Hilmas et al. 2008)

5.6.4 Blister Agents

Blistering agents, or vesicants, promote the production of blisters. Typical examples include sulfur mustard (HD), nitrogen mustard (HN), and Lewisite (L). These agents, especially sulfur mustard, are considered capable chemical weapons since illness may not occur until hours or days later. Vesicants are alkylating agents that affect rapidly reproducing and poorly differentiated cells in the body. However, they can also produce cellular oxidative stress, deplete glutathione stores, and promote

intense inflammatory responses. Clinical findings are initially cutaneous (erythema, pruritus, yellow blisters, ulcers, and sloughing), respiratory (hoarseness, cough, voice changes, pneumonia, respiratory failure, acute lung injury, and acute respiratory distress syndrome), and ophthalmologic (pain, irritation, blepharospasm, photophobia, conjunctivitis, corneal ulceration, and globe perforation) in nature. After exposure through these primary portals of entry, other sites are affected, including the gastrointestinal tract (nausea, vomiting, and mucosal injury), the hematopoietic system (bone marrow suppression), the cardiovascular system (L), reproductive system (HD, HN), and the central nervous system (lethargy, headache, malaise, and depression) (Banks 2014; Yu et al. 2003).

Pediatric manifestations (Table 5.19) may vary from the classic clinical responses due to their unique vulnerabilities (Hilmas et al. 2008):

- Thinner integument leading to shorter time from exposure to symptom development.
- Higher vapor density and concentration accumulation in the living zone.
- Higher minute ventilation and metabolism.
- Greater pulmonary injury.
- Ocular findings more frequent (less self-protection and more hand/eye contact).
- Gastrointestinal manifestations more prominent.
- Unable to escape and decontaminate.
- Unable to verbalize complaints (i.e., pain).

Treatment (Tables 5.20 and 5.21) includes decontamination and supportive care. Currently, there are no antidotes for mustard toxicity (Cieslak and Henretig 2016). Agents under investigation include antioxidants (Vitamin E), anti-inflammatory drugs (corticosteroids), mustard scavengers (glutathione, *N*-acetylcysteine), and nitric oxide synthase inhibitors (L-nitroarginine methyl ester). Other therapeutics under investigation include the use of British Anti-Lewisite (BAL), reactive skin protectants, and ocular therapies (National Institutes of Health 2007; USAMRIID 2014).

5.6.5 Lung-Damaging Agents

Lung-damaging agents are toxic inhalants and potentially can affect the entire respiratory tract. Typical examples include chlorine (Cl_2), phosgene (carbonyl chloride), oxides of nitrogen, organofluoride polymers, hydrogen fluoride, and zinc oxide. Since many of these chemicals are readily available and have multiple industrial applications, they are considered terrorist weapons of opportunity. Toxicity is dependent upon agent particle size, solubility, and method of release. Large particles produce injury in the nasopharynx (sneezing, pain, and erythema). Midsize particles affect the central airways (painful swelling, cough, stridor, wheezing, and rhonchi). Small particles cause injury at the level of the alveoli (dyspnea, chest tightness, and rales). Highly soluble agents, such as chlorine, dissolve with mucosal moisture and immediately produce strong upper airway reactions. Less soluble

Table 5.20 Recommended treatment and management of chemical agents used in terrorism

Agent	Toxicity	Clinical findings	Onset	Decontamination[a]	Management
Nerve agents					
Tabun, Sarin, Soman, VX	Anticholinesterase: muscarinic, nicotinic, and CNS effects	Vapor: miosis, rhinorrhea, dyspnea	Vapor: seconds	Vapor: fresh air, remove clothes, wash hair	ABCs Atropine[b,c,d]: 0.05 mg/k IV, IM (min 0.1 mg, max 5 mg), repeat q2–5 min prn for marked secretions, bronchospasm Pralidoxime[e]: 25 mg/kg IV, IM (max 1 g IV; 2 g IM), may repeat within 30–60 min prn, then again q1 h 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 seizures, or severe exposure
		Liquid: Diaphoresis, emesis Both: coma, paralysis, seizures, apnea	Liquid: minutes to hours	Liquid: remove clothes, copious washing of skin and hair with soap and water, ocular irrigation	
Blistering (Vesicant) agents					
Mustard	Alkylation	Skin: erythema, vesicles Eye: inflammation Respiratory tract: inflammation, respiratory distress, acute respiratory distress syndrome	Hours	Skin: soap and water Eyes: irrigation with water (major impact only if done within minutes of exposure)	Symptomatic care
Lewisite	Arsenical		Immediate pain		Possibly British Anti-Lewisite (BAL) 3 mg/kg IM q4–6 h for systemic effects of lewisite in severe cases
Pulmonary agents					

Chlorine, phosgene	Liberate HCl, alkylation	Eyes, nose, throat, irritation (especially chlorine)	Minutes	Fresh air Skin: water	Symptomatic care
		Bronchospasm, pulmonary edema (especially phosgene)	Bronchospasm: minutes Pulmonary edema: hours		
Blood agents					
Cyanide	Cytochrome oxidase inhibition: cellular anoxia, lactic acidosis	Tachypnea, coma, seizures, apnea	Seconds	Fresh air Skin: soap and water	Airway, breathing, circulatory support, 100% oxygen Sodium bicarbonate prn for metabolic acidosis Sodium nitrite (3%): Dose (mL/kg)—Est. Hb (g/dL) 0.27—10 0.33—12 0.39—14 Max 10 mL Sodium thiosulfate (25%) 1.65 mL/kg (max 50 mL) Need to consider hydroxocobalamin, which may be very useful especially during a terrorist incident (Cyanokit)

Used with permission from Society of Critical Care Medicine

References: (Markenson et al. 2006; Berger and Burns 2012; Cieslak and Henretig 2016; Jacobson and Severin 2012; Markenson and Redlener 2007)

[a]Decontamination, especially for patients with significant nerve agent or vesicant exposure, should be performed by nurses or HCPs garbed in adequate personal protective equipment. For Emergency Department staff, this consists of nonencapsulated, chemically resistant body suit, boots, and gloves with a full-face air purifier mask/hood

[b]Intraosseous route is likely equivalent to intravenous

[c]Atropine might have some benefit via endotracheal tube or inhalation, as might aerosolized ipratropium

[d]As of September 2004, the FDA has approved pediatric autoinjectors of atropine in 0.25, 0.5, and 1 mg sizes. Recommendations are:

Approximate age	Approximate weight (kg)	Approximate weight (lb)	Autoinjector size
Less than 6 months	Less than 7.5 kg	Less than 15 lb	0.25 mg
6 months–4 years	7.5–18 kg	15–40 lb	0.5 mg
5–10 years	18–30 kg	41–90 lb	1 mg
Greater than 10 years	Greater than 30 kg	Greater than 90 lb	2 mg (adult size)

[e]Pralidoxime is reconstituted to 50 mg/mL (1 g in 20 mL water) for IV administration, and the total dose infused over 30 min, or may be given by continuous infusion (loading dose 25 mg/kg over 30 min, then 10 mg/kg/h). For IM use, it might be diluted to a concentration of 300 mg/mL (1 g added to 3 mL water—by analogy to the US Army's Mark-1 autoinjector concentration) to effect a reasonable volume for injection. Pediatric autoinjectors of pralidoxime are not FDA approved or available; however, for mass casualty situations, consider the following:

Approximate age (yr)	Approximate weight (kg)	Number of autoinjectors	Pralidoxime dose (mg/kg)
3–7	13–25	1	24–46 mg/kg
8–14	26–50	2	24–46 mg/kg
Older than 14	Over 50 kg	3	Less than 35 kg

agents, such as phosgene, travel to the lower airway before dissolving and subsequently causing toxicity. It is important, however, to realize that very few lung-damaging agents affect only the upper or lower airway (e.g., Cl_2). If the agent is aerosolized, solid or liquid droplets suspend in the air and distribute by size. If it is a gas or vapor release, there is uniform distribution throughout the lungs and toxicity will be based on solubility and reactivity of the agent (Banks 2014; Burklow et al. 2003; Cieslak and Henretig 2016).

Pediatric manifestations (Table 5.19) may vary from the classic clinical responses due to their unique vulnerabilities (Hilmas et al. 2008):

- Pediatric airway and respiratory tract issues (obligate nose breathers, relatively small mouth/large tongue, copious secretions, anterior/cephalad vocal cords, Omega or horseshoe-shaped epiglottis, tendency of laryngospasm and bronchospasm, and anatomically small, "floppy" airways).
- High vapor density and concentration accumulation in the living zone.
- Unable to verbalize or localize physical complaints.
- Rapid dehydration and shock secondary to pulmonary edema.
- Increased minute ventilation and metabolism.

Differential diagnoses include smoke inhalation injury, cardiogenic shock, heart failure, traumatic injury, asthma, bronchiolitis, and poisoning (cyanide). Treatment (Tables 5.20 and 5.21) includes decontamination and supportive care. Currently, there are no antidotes for lung-damaging agent toxicity (Cieslak and Henretig 2016). Potential countermeasures include novel positive-pressure devices, drugs to prevent lung inflammation, and treatments for chemically induced pulmonary edema (beta agonists, dopamine, insulin, allopurinol, and ibuprofen). In addition, drugs are being investigated that act at complex molecular pathways of the lung

Table 5.21 Decontamination for patients exposed to chemical agents of terrorism

Agent	Decontamination[a]
Nerve agents (tabun, sarin, soman, VX)	Vapor: fresh air, remove clothes, wash hair Liquid: remove clothes, wash skin, hair with copious soap and water, ocular irrigation
Vesicants (mustard, Lewisite)	Skin: soap and water Eyes: water (effective only if done within minutes of exposure)
Pulmonary agents (chlorine, phosgene)	Fresh air Skin: water
Cyanide	Fresh air Skin: soap and water

[a]Decontamination, especially for patients with significant nerve agent or vesicant exposure, should be performed by nurses or HCPs garbed in adequate personal protective equipment. For Emergency Department staff, this equipment consists of a nonencapsulated, chemically resistant body suit, boots, and gloves with a full-face air-purifier mask/hood (Cieslak and Henretig 2016)

(i.e., modulate the activity of ion channels that control fluid transport across lung membranes or supports surfactant) (National Institutes of Health 2007; USAMRIID 2014).

5.7 Biological Agents

The Centers for Disease Control and Prevention (CDC) has delineated bioterrorism agents and diseases into three categories based on priority. Category A agents include organisms with the highest risk because the ease of dissemination or transmission from person-to-person, result in high mortality rates, have the potential for major public health impact, promote public panic and social disruption, and require special action of public health preparedness. These agents/diseases include smallpox (*Variola major*), anthrax (*Bacillus anthracis*), plague (*Yersinia pestis*), viral hemorrhagic fevers (filoviruses [Ebola, Marburg] and arenaviruses [Lassa, Macupo]), botulinum toxin (from *Clostridium botulinum*), and tularemia (*Francisella tularensis*). Category B agents, the second highest priority, include those that are moderately easy to disseminate, result in moderate morbidity and low mortality rates, and require specific enhancements of diagnostic capacity and enhanced disease surveillance. These agents/diseases include ricin toxin (*Ricinus communis*), brucellosis (*Brucella* species), epsilon toxin of *Clostridium perfringens*, food safety threats (*Salmonella* species, *Escherichia coli* O157:H7, *Shigella*), glanders (*Burkholderia mallei)*, melioidosis (*Burkholderia pseudomallei*), psitticosis (*Chlamydia psittaci*), typhus fever (*Rickettsia prowazekii*), Q fever (*Coxiella burnetii*), Staphylococcal enterotoxin B, trichothecenes mycotoxin, viral encephalitis (alphaviruses, such as eastern equine encephalitis, Venezuelan equine encephalitis, and western equine encephalitis), and water safety threats (*Vibrio cholera*, *Cryptosporidium parvum*). Category C agents have the next priority and include emerging pathogens that could be engineered for mass dissemination because of availability, ease of production and dissemination, and have the potential for high morbidity and mortality rates and major health impact.

These agents include Nipah virus, hantavirus, yellow fever virus, drug resistant tuberculosis, and tick-borne encephalitis (Markenson et al. 2006; Centers for Disease Control and Prevention 2019a; Cieslak 2018). Based on biological threats to national security, the National Institute of Allergy and Infectious Disease has developed a strategic plan that outlines priority areas of biodefense research including infrastructure, basic research, and medical countermeasure development for these agents/diseases (National Institutes of Health 2005). The strategic efforts have since been expanded by a collaborative effort among the CDC, Food and Drug Administration (FDA), Biomedical Advanced Research and Development Authority (BARDA), Public Health Emergency Medical Countermeasure Enterprise (PHEMCE), National Institutes of Health (NIH), and other key stakeholders (Fagbuyi et al. 2016; U.S. Department of Health and Human Services, Assistant Secretary for Preparedness and Response Biomedical Advanced Research and Development Authority 2016; U.S. Department of Health and Human Services, Assistant Secretary for Preparedness and Response 2017; USAMRIID 2014).

Recognition of a biologic attack is essential. There are various epidemiologic clues to consider when determining whether the outbreak is natural or man-made (Markenson et al. 2006; Cieslak 2018; USAMRIID 2014):

- The appearance of a large outbreak of cases of a similar disease or syndrome, or especially in a discrete population.
- Many cases of unexplained diseases or deaths.
- More severe disease than is usually expected for a specific pathogen or failure to respond to standard therapy.
- Unusual routes of exposure for a pathogen, such as the inhalational route for disease that normally occur through other exposures.
- A disease case or cases that are unusual for a given geographic area or transmission season.
- Disease normally transmitted by a vector that is not present in the local area.
- Multiple simultaneous or serial epidemics of different diseases in the same population.
- A single case of disease by an uncommon agent (smallpox, some viral hemorrhagic fevers, inhalational anthrax, pneumonic plague).
- A disease that is unusual for an age group.
- Unusual strains or variants of organisms or antimicrobial resistance patterns different from those known to be circulating.
- A similar or identical genetic type among agents isolated from distinct sources at different times and/or locations.
- Higher attack rates among those exposed in certain areas, such as inside a building if released indoors, or lower rates in those inside a sealed building if released outside.
- Outbreaks of the same disease occurring in noncontiguous areas.
- Zoonotic disease outbreaks.
- Intelligence of a potential attack, claims by a terrorist or aggressor of a release, and discovery of munitions, tampering, or other potential vehicle of spread (spray device, contaminated letter).

One should know the cellular, physiological, and clinical manifestations of each biologic agent. Furthermore, knowledge of distinct presentation patterns of children will be helpful to diagnosis. In any event, the ten steps in the management of biologic attack victims, pediatric, or otherwise, should be applied (Cieslak and Henretig 2003; Cieslak 2018; USAMRIID 2014):

1. Maintain an index of suspicion.
2. Protect yourself.
3. Assess the patient.
4. Decontaminate as appropriate.
5. Establish a diagnosis.
6. Render prompt treatment.
7. Practice good infection control.
8. Alert the proper authorities.
9. Assist in the epidemiologic investigation and manage the psychological consequences.
10. Maintain proficiency and spread the word.

5.7.1 Smallpox (Variola major)

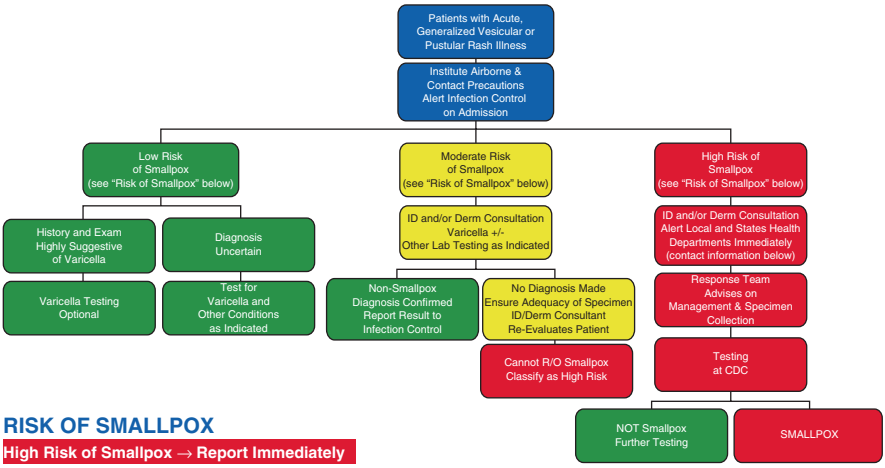
Smallpox is caused by the Orthopoxvirus variola and was declared globally eradicated in 1980. The disease is highly communicable from person-to-person and remains a threat due to its potential for weaponization. The only stockpiles are at the CDC and at the Russian State Centre for Research on Virology and Biotechnology. However, clandestine stockpiles in other parts of the world are unknown. Since the cessation of smallpox vaccination, the general population has little or no immunity.

The three clinical forms of smallpox include ordinary, flat, and hemorrhagic. Another form, modified type, occurred in those previously vaccinated who were no longer protected. The asymptomatic incubation period is from 7 to 17 days (average 12 days) after exposure. A prodrome follows that lasts for 2–4 days and is marked by fever, malaise, and myalgia. Lesions start on the buccal and pharyngeal mucosa. The rash then spreads in a centrifugal fashion, and the lesions are synchronous. Initially, there are macules followed by papules, pustules, and scabs in 1–2 weeks. Other clinical features include extensive fluid loss and hypovolemic shock, nausea, vomiting, diarrhea, bacterial superinfections, viral bronchitis and pneumonitis, corneal ulceration with or without keratitis, and encephalitis. Death, if it occurs, is typically during the second week of clinical disease. *Variola minor* caused a mortality of 1% in unvaccinated individuals. However, the *Variola major* type caused death in 3% and 30% in those vaccinated and unvaccinated, respectively. Flat (mostly children) and hemorrhagic (pregnant women and immunocompromised) types caused severe mortality in those populations infected.

The differential diagnoses for smallpox include chickenpox (varicella), herpes, erythema multiforme with bullae, or allergic contact dermatitis. Varicella typically has a longer incubation period (14–21 days) and minimal or no prodrome. Furthermore, the rash distributes in a centripetal fashion and the progression is asynchronous (Images 5.1 and 5.2). Diagnosis of smallpox is mostly clinical

EVALUATING PATIENTS FOR SMALLPOX

ACUTE, GENERALIZED VESICULAR OR PUSTULAR RASH ILLNESS PROTOCOL



RISK OF SMALLPOX

High Risk of Smallpox → Report Immediately

1. Febrile prodrome (defined below) **AND**
2. Classic smallpox lesion (defined below & photo at top right) **AND**
3. Lesions in same stage of development (defined below)

Moderate Risk of Smallpox → Urgent Evaluation

1. Febrile prodrome (defined below) **AND**
2. One other **MAJOR** smallpox criterion (defined below)

OR

1. Febrile prodrome (defined below) **AND**
2. ≥4 **MINOR** smallpox criteria (defined below)

Low Risk of Smallpox → Manage as Clinically Indicated

1. No febrile prodrome

OR

1. Febrile prodrome **AND**
2. <4 **MINOR** smallpox criteria (defined below)

There have been no naturally occuring cases of smallpox anywhere in the world since 1977. A high risk case of smallpox is a public health and medical emergency.

Report all HIGH RISK CASES immediately (without waiting for lab result) to:

1. Hospital Infection Control _____ ( ) ____ - _____
   _____ ( ) ____ - _____
2. _____ health department ( ) ____ - _____
   ( ) ____ - _____
3. _____ health department ( ) ____ - _____
   ( ) ____ - _____

MAJOR SMALLPOX CRITERIA

- **FEBRILE PRODROME:** occurring 1-4 days before rash onset: fever ≥101°F **and** at least one of the following: prostration, headache, backache, chills, vomiting or severe abdominal pain
- **CLASSIC SMALLPOX LESIONS:** deep-seated, firm/hard, round well-circumscribed vesicles or pustules; as they evolve, lesions may become umbilicated or confluent
- **LESIONS IN SAME STAGE OF DEVELOPMENT:** on any one part of the body (e.g., the face, or arm) all the lesions are in same stage of development (i.e., all are vesicles, or all are pustules)

MINOR SMALLPOX CRITERIA

- Centrifugal distribution: greatest concentration of lesions on face and distal extremities
- First lesions on the oral mucosa/palate, face, or forearms
- Patient appears toxic or moribund
- Slow evolution: lesions evolve from macules to papules→ pustules over days (each stage lasts 1-2 days)
- Lesions on the palms and soles

For more information, please go to the CDC website www.cdc.gov/smallpox

Centers for Disease Control and Prevention
National Center for Emerging and Zoonotic Infectious Diseases

CS205116-A

Image 5.1 CDC Evaluating Patients for Smallpox Algorithm. (Used with permission from Centers for Disease Control and Prevention; Source: https://www.cdc.gov/smallpox/pdfs/smallpox-diagnostic-algorithm-poster-2-pages.pdf)

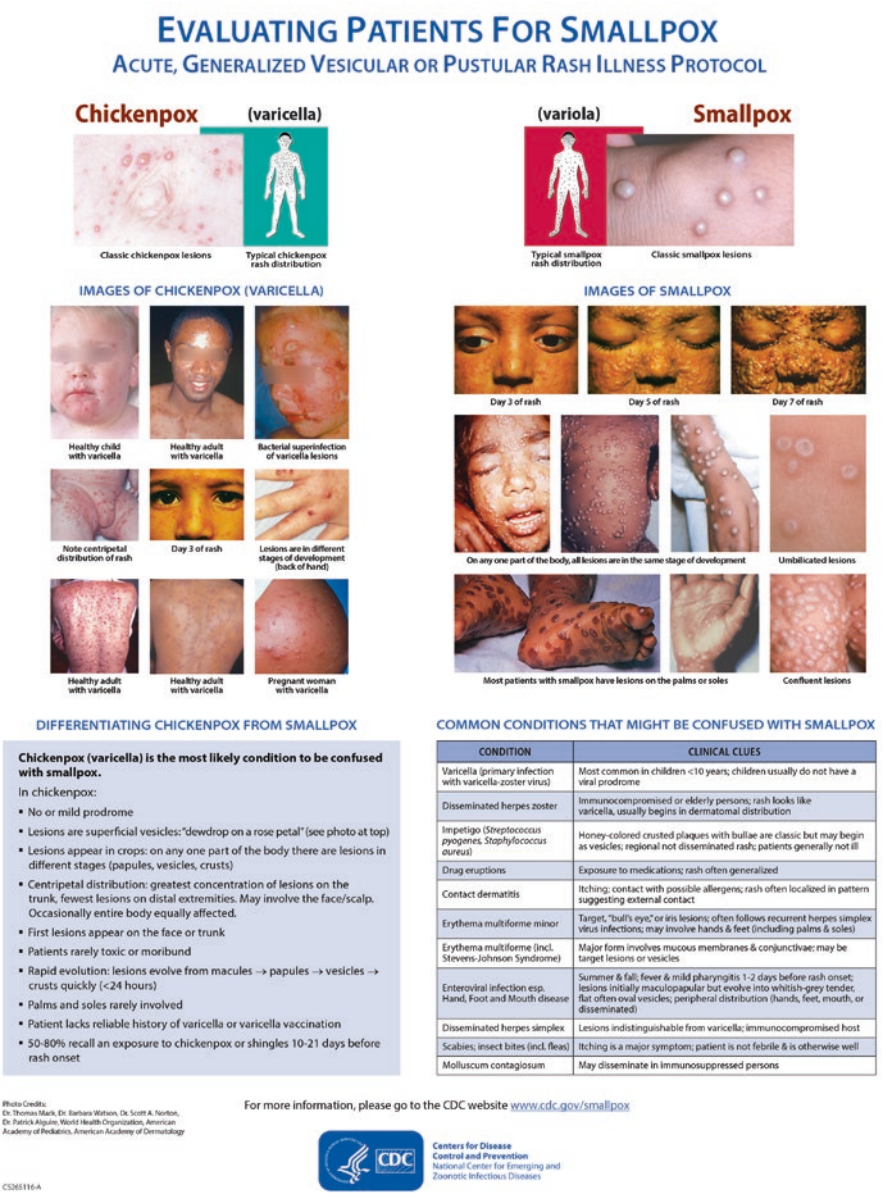


Image 5.2 CDC Evaluating Patients for Smallpox Algorithm. (Used with permission from Centers for Disease Control and Prevention; Photo Credits: Dr. Thomas Mack, Dr. Barbara Watson, Dr. Scott A. Norton, Dr. Patrick Alguire, World Health Organization, Philadelphia Department of Public Health, American Academy of Pediatrics, American Academy of Dermatology, American College of Physicians; Source: https://www.cdc.gov/smallpox/pdfs/smallpox-diagnostic-algorithm-poster-2-pages.pdf)

(Centers for Disease Control and Prevention 2019a). If considered, contact public health immediately. Laboratory confirmation (CDC or WHO) can be done by DNA sequencing, polymerase chain reaction (PCR), restriction fragment-length polymorphism (RFLP), real-time PCR, and microarrays. These are more sensitive and specific than the conventional virological and immunological approaches (Goff et al. 2018).

Generally, treatment is largely supportive (Table 5.23). Fluid losses and hypovolemic shock must be addressed. Also, due to electrolyte and protein loss, replacement therapy will be required. Bacterial superinfections must be aggressively treated with appropriate antibiotics. Biologic countermeasures and antivirals against smallpox are under investigation, including cidofovir, brincidovir (CMX-001), and tecovirimat (ST-246). These agents have shown efficacy in orthopoxvirus animal models and have been used to treat disseminated vaccinia infection under emergency use. Cidofovir has activity against poxviruses in animal studies (in vitro and in vivo) and some humans (eczema vaccinatum and molluscum contagiosum). Brincidovir is an oral formulation of cidofovir with less nephrotoxicity and has recently been announced as an addition to the Strategic National Stockpile (SNS) for patients with smallpox. Tecovirimat is a potent and specific inhibitor of orthopoxvirus replication. A recent study found that treatment with tecovirimat resulted in 100% survival of cynomolgus macaques challenged with intravenous variola virus. The disease was milder in tecovirimat-treated survivors and viral shedding was reduced compared to placebo-treated survivors. Prophylaxis comes in the form of the smallpox vaccine (vaccinia virus), ACAM2000®, which replaced Wyeth Dryvax™ in 2007. Safety profile of the two vaccines appears to be similar. Side effects of vaccination range from low-grade fever and axillary lymphadenopathy to inadvertent inoculation of the virus to other body sites to generalized vaccinia and cardiac events (myopericarditis). Rare, but typically fatal complications include progressive vaccinia, eczema vaccinatum, postvaccination encephalomyelitis, and fetal vaccinia. Modified Vaccinia Ankara (MVA) Smallpox Vaccine (Bavarian Nordic's IMVAMUNE®) is a live, highly attenuated, viral vaccine that is under development as a future nonreplicating smallpox vaccine (Greenberg et al. 2016; Kennedy and Greenberg 2009). Passive immunoprophylaxis exists in the form of vaccinia immune globulin (VIG) and is used for primarily treating complications from smallpox vaccine. Limited information suggests that VIG may be of use in postexposure prophylaxis of smallpox if given the first week after exposure and with vaccination. Monoclonal antibodies may represent another form of immunoprophylaxis. Postexposure administration of human monoclonal antibodies has protected rabbits from a lethal dose of an orthopoxvirus. As mentioned, smallpox is highly communicable person-to-person (Table 5.25). Contact precautions with full personal protective equipment (PPE) are required. Airborne isolation with the use of an N-95 mask is needed for baseline protection. An N-95 mask or powered air-purifying respirator (PAPR) is recommended for protection during high risk procedures (Beigel and Sandrock 2009; Goff et al. 2018; Rotz et al. 2005; Pittman et al. 2018; USAMRIID 2014).

5.7.2 Anthrax (*Bacillus anthracis*)

Anthrax is caused by the aerobic, spore-forming, nonmotile, encapsulated gram-positive rod *Bacillus anthracis*. It is a naturally occurring disease in herbivores. Humans contract the illness by handling contaminated portions of infected animals, especially hides and wool. Infection is introduced by scratches or abrasions on the skin. There is concern for potential aerosol dispersal leading to intentional infection through inhalation: it is fairly easy to obtain, capable of large quantity production, stable in aerosol form, and highly lethal.

Anthrax spores enter the body via skin, ingestion, or inhalation. The spores germinate inside macrophages and become vegetative bacteria. The vegetative form is released, replicates in the lymphatic system, and produces intense bacteremia. The production of virulence factors leads to overwhelming sepsis. The main virulence factors are encoded on two plasmids. One produces an antiphagocytic polypeptide capsule. The other contains genes for the synthesis of three proteins it secretes: protective antigen, edema factor, and lethal factor. The combination of protective antigen with lethal factor or edema factor forms binary cytotoxins, lethal toxin, and edema toxin. The anthrax capsule, lethal toxin, and edema toxin act in concert to drive the disease. Three clinical syndromes occur with anthrax: cutaneous, gastrointestinal, and inhalational.

Cutaneous anthrax is the most common naturally occurring form. After an individual is exposed to infected material or the agent itself, there is a 1–12 day (average 7 days) incubation period. A painless or pruritic papule forms at the site of exposure. The papule enlarges and forms a central vesicle, which is followed by erosion into a coal-black but painless eschar. Edema surrounds the area and regional lymphadenopathy may occur.

Gastrointestinal anthrax is rare. Typically, it develops after ingestion of viable vegetative organisms found in undercooked meats of infected animals. The two forms of gastrointestinal anthrax, oropharyngeal and intestinal, have incubation periods of 1–6 days. The oropharyngeal form is marked by fever and severe pharyngitis followed by ulcers and pseudomembrane formation. Other findings include dysphagia, regional lymphadenopathy, unilateral neck swelling, airway compromise, and sepsis. The intestinal form begins with fever, nausea, vomiting, and abdominal pain. Bowel edema develops which leads to mesenteric lymphadenitis with necrosis, shock, and death.

Endemic inhalational anthrax (Woolsorters' disease) is also extremely rare and is due to inhaling spores. Therefore, any case of inhalational anthrax should be assumed to be due to intentional exposure until proven otherwise. The incubation period is 1–5 days but can be up to 43 days. There is a prodrome of 1–2 days consisting of fever, malaise, and cough. Within 24 h, the disease rapidly progresses to respiratory failure, hemorrhagic mediastinitis (wide mediastinum), septic shock, multiorgan failure, and death. Patients with inhalational anthrax may also have hemorrhagic meningitis. Mortality is greater than 80% in 24–36 h despite aggressive treatment of inhalational anthrax.

The differential diagnoses of ulceroglandular lesions include antiphospholipid antibody syndrome, brown recluse spider bite, coumadin/heparin necrosis, cutaneous leishmaniasis, cutaneous tuberculosis, ecthyma gangrenosum, glanders, leprosy, mucormycosis, orf, plague, rat bite fever, rickettsial pox, staphylococcal/streptococcal ecthyma, tropical ulcer, tularemia, and typhus. The differential diagnoses of ulceroglandular syndromes include cat scratch fever, chancroid, glanders, herpes, lymphogranuloma venereum, melioidosis, plague, staphylococcal and streptococcal adenitis, tuberculosis, and tularemia. The differential diagnoses for inhalational anthrax include influenza and influenza-like illnesses from other causes. The differential diagnoses of mediastinal widening include normal variant, aneurysm, histoplasmosis, sarcoidosis, tuberculosis, and lymphoma.

The diagnosis of anthrax is by culture and Gram stain of the blood, sputum, pleural fluid, cerebrospinal fluid, or skin. Specimens must be handled carefully, especially by lab personnel and those performing autopsies. ELISA and PCR are available at some reference laboratories. The chest radiograph of inhalational anthrax shows the classic widening of the mediastinum. Additional findings include hemorrhagic pleural effusions, air bronchograms, and/or consolidation (Purcell et al. 2018).

Supportive treatment is indicated, including mechanical ventilation, pleural effusion drainage, fluid and electrolyte support, and vasopressor administration. For inhalational anthrax, antibiotic treatment is unlikely to be effective unless started before respiratory symptoms develop. Treatment (Table 5.22) includes ciprofloxacin (or levofloxacin or doxycycline), clindamycin, and penicillin G. Raxibacumab, a monoclonal antibody, was approved by the FDA in 2012 for the treatment of inhalational anthrax in combination with recommended antibiotic regimens and prophylaxis for inhalational anthrax when other therapies are unavailable or inappropriate. It works by inhibiting anthrax antigen binding to cells and, therefore, prevents toxins from entering cells (Kummerfeldt 2014). The adult dose is 40 mg/kg given IV over 2 h and 15 min. The dose for children is weight based; ≤15 kg: 80 mg/kg; >15–50 kg: 60 mg/kg; >50 kg: 40 mg/kg. Premedication with diphenhydramine IV or PO is recommended 1 h before the infusion. It can also be used as postexposure prophylaxis in high risk spore exposure cases (Cieslak and Henretig 2016; Migone et al. 2009; The Medical Letter 2013). Obiltoxaximab (Anthim) is a recently approved monoclonal antibody treatment for inhalational anthrax in combination with recommended antibiotic regimens and prophylaxis for inhalational anthrax when other therapies are unavailable or inappropriate. Adults and children >40 kg should receive a single obiltoxaximab dose of 16 mg/kg. The recommended dose is 24 mg/kg for children >15–40 kg and 32 mg/kg for those weighing ≤15 kg. Premedication with diphenhydramine is recommended to reduce risk of hypersensitivity reactions (The Medical Letter 2018). In patients with inhalational anthrax, intravenous anthrax immune globulin (Anthrasil) should be considered in addition to appropriate antibiotic therapy (Mytle et al. 2013; The Medical Letter 2016; USAMRIID 2014). Postexposure prophylaxis includes ciprofloxacin (or levofloxacin or doxycycline) for 60 days plus administration of vaccine; since spores can persist in human

Table 5.22 Recommended therapy and prophylaxis of anthrax in children

Form of anthrax	Category of treatment (therapy or prophylaxis)	Agent and dosage
Inhalation	Therapy[a,b]	Ciprofloxacin[c] 10–15 mg/kg IV q12 h (max 400 mg/dose) **or** Doxycycline 2.2 mg/kg IV (max 100 mg) q12 h ***and*** Clindamycin[d] 10–15 mg/kg IV q8 h ***and*** Penicillin G[e] 400–600 k μ/kg/day IV divided q4 h ***plus*** raxibacumab Patients who are clinically stable after 14 days can be switched to a single oral agent (ciprofloxacin or doxycycline) to complete a 60-day course[f] of therapy
Inhalation	Postexposure prophylaxis (60-day course[f])	Ciprofloxacin[g] 10–15 mg/kg PO (max 500 mg/dose) q12 h **or** Doxcycline 2.2 mg/kg (max 100 mg) PO q12 h
Cutaneous, endemic	Therapy[h]	Penicillin V 40–80 mg/kg/day PO divided q6 h **or** Amoxicillin 40–80 mg/kg/d PO divided q8h **or** Ciprofloxacin 10–15 mg/kg PO (max 1 g/day) q12 h **or** Levofloxacin 10–15 mg/kg IV q24 h **or** Doxycycline 2.2 mg/kg PO (max 100 mg) q12 h
Cutaneous (in setting of terrorism)	Therapy[a]	Ciprofloxacin 10–15 mg/kg PO (max 1 g/day) q12 h **or** Levofloxacin 10–15 mg/kg IV q24 h Doxycycline 2.2 mg/kg PO (max 100 mg) q12 h
Gastrointestinal	Therapy[a]	Same as inhalational

Used with permission from Society of Critical Care Medicine

References: (Berger and Burns 2012; Cieslak and Henretig 2016; Markenson and Redlener 2007; Pittman et al. 2018; USAMRIID 2014)

[a]In a mass casualty setting, in which resources are severely limited, oral therapy may need to be substituted for the preferred parenteral option

[b]In addition to appropriate antibiotic regimen, monoclonal antibody therapy (see text for dosing) and intravenous anthrax immune globulin should be administered for inhalational anthrax

[c]Levofloxacin or ofloxacin may be an acceptable alternative to ciprofloxacin

[d]Rifampin or clarithromycin may be acceptable alternatives to clindamycin as a drug that targets bacterial protein synthesis. If ciprofloxacin or another quinolone is employed, doxycycline may be used as a second agent because it also targets protein synthesis

[e]Ampicillin, imipenem, meropenem, or chloramphenicol may be acceptable alternatives to penicillin as drugs with good CNS penetration

[f]Assuming the organism is sensitive, children may be switched to oral amoxicillin (40–80 mg/kg/d divided q8 h) to complete a 60-day course. The first 14 days of therapy of postexposure prophylaxis, however, should include ciprofloxacin or levofloxacin and/or doxycycline regardless of age. Vaccination should also be provided; if not, antibiotic course will need to be longer

[g]According to most experts, ciprofloxacin is the preferred agent for oral prophylaxis

[h]Ten days of therapy may be adequate for endemic cutaneous disease. A full 60-day course is recommended in the setting of terrorism, however, because of the possibility of concomitant inhalational exposure

tissues for a long time, antibiotics must be given for a longer period if vaccine is not also given. The Anthrax Vaccine Adsorbed (AVA BioThrax™) is derived from sterile culture fluid supernatant taken from an attenuated strain of *Bacillus anthracis* and does not contain any live or dead organisms. The vaccine is given 0.5 mL intramuscularly at 0 and 4 weeks then at 6, 12, and 18 months followed by yearly boosters (Pittman et al. 2018; USAMRIID 2014). Consult with CDC for current pediatric recommendations.

Anthrax is not contagious in the vegetative form during clinical illness (Table 5.25). Contact with infected animals increases likelihood of spread. Therefore, contact should be limited and the use of appropriate PPE in endemic areas is indicated (Beigel and Sandrock 2009; Carbone 2005; Purcell et al. 2018; USAMRIID 2014).

5.7.3 Plague (*Yersinia pestis*)

Plague is caused by *Yersinia pestis*, a nonmotile, nonsporulating gram-negative bacterium. It is a zoonotic disease of rodents. It is typically found worldwide and is endemic in western and southwestern states. Humans develop the disease after contact with infected rodents, or being bitten by their fleas. After a rodent population dies off, the fleas search for other sources of blood, namely humans. This is when large outbreaks of human plague occur. Pneumonic plague is a very rare disease and when it is present in a patient, it may be highly suspicious for intentional dispersal of this deadly agent. Three clinical syndromes occur with plague: bubonic plague (85%), septicemic plague (13%), and primary pneumonic plague (1–2%).

Bubonic plague occurs after an infected flea bites a human. After an incubation period of 2–8 days, there is onset of high fever, severe malaise, headache, myalgias, and nausea with vomiting. Almost 50% have abdominal pain. Around the same time, a characteristic bubo forms which is tender, erythematous, and edematous without fluctuation. Buboes typically form in the femoral or inguinal lymph nodes, but other areas can be involved as well (axillary, intraabdominal). The spleen and liver can be tender and palpable. The disease disseminates without therapy. Severe complications can ensue, including pneumonia, meningitis, sepsis, and multiorgan failure. Pneumonia is particularly concerning since these patients are extremely contagious. Mortality of untreated bubonic plague is 60%, but 5% with efficient and effective treatment.

Septicemic plague is characterized by acute fever followed by sepsis without bubo formation. The clinical syndrome is very similar to other forms of gram-negative sepsis: chills, malaise, tachycardia, tachypnea, hypotension, nausea, vomiting, and diarrhea. In addition to sepsis, disseminated intravascular coagulation can ensue leading to thrombosis, necrosis, gangrene, and the formation of black appendages. Multiorgan failure can quickly follow. Untreated septicemic plague is almost 100% fatal versus 30–50% in those treated.

Pneumonic plague is very rare and should be considered due to an intentional aerosol release until proven otherwise. The incubation period is relatively short at

1–3 days. Sudden fever, cough, and respiratory failure quickly follow. This form produces a fulminant pneumonia with watery sputum that usually progresses to bloody. Within a short period of time, septic shock and disseminated intravascular coagulation develop. ARDS and death may occur. Mortality rate of pneumonic plague is very high but may respond to early treatment.

Plague meningitis is a rare complication of plague. It can occur in 6% of patients with septicemia and pneumonic forms and is more common in children. Usually occurring a few weeks into the illness, it affects those receiving subtherapeutic doses of antibiotics or bacteriostatic antibiotics that do not cross the blood-brain barrier (tetracyclines). Fever, meningismus, and other meningeal signs occur. Plague meningitis is virtually indistinguishable from meningococcemia.

The differential diagnoses of bubonic plague include tularemia, cat scratch fever, lymphogranuloma venereum, chancroid, scrub typhus, and other staphylococcal and streptococcal infections. The differential diagnoses of septicemic plague should include meningococcemia, other forms of gram-negative sepsis, and rickettsial diseases. The differential diagnosis of pneumonic plague is very broad. However, sudden appearance of previously healthy individuals with rapidly progressive gram-negative pneumonia with hemoptysis should strongly suggest pneumonic plague due to intentional release.

Diagnosis can be made clinically as previously described. Demonstration of *Yersinia pestis* in blood or sputum is paramount. Methylene blue or Wright's stain of exudates may reveal the classic safety-pin appearance of *Yersinia pestis*. Culture on sheep blood or MacConkey agar demonstrates beaten-copper colonies (48 h) followed by fried-egg colonies (72 h). Detection of *Yersinia pestis* F1-antigen by specific immunoassay is available, but the result is available retrospectively. Chest radiograph of patients will demonstrate patchy infiltrates (Centers for Disease Control and Prevention 2018a; Worsham et al. 2018).

Treatment includes mechanical ventilation strategies for ARDS, hemodynamic support (fluid and vasopressor administration), and antimicrobial agents (Table 5.23). Gentamicin or streptomycin is the preferred antimicrobial treatment. Alternatives include doxycycline or ciprofloxacin or levofloxacin or chloramphenicol. In cases of meningitis, chloramphenicol is recommended due to its ability to effectively cross the blood-brain barrier. Streptomycin is in limited supply and is available for compassionate use. It should be avoided in pregnant women. Postexposure prophylaxis includes doxycycline or ciprofloxacin. No licensed plague vaccine is currently in production. A previous licensed vaccine was used in the past. It only offered protection against bubonic plague but not aerosolized *Yersinia pestis*. The plague bacterium secretes several virulence factors (Fraction 1 (F1) and V (virulence) proteins) that as subunit proteins are immunogenic and possess protective properties. Recently, an F1-V antigen (fusion protein) vaccine developed by USAMRIID provided 100% protection in monkeys against high-dose aerosol challenge. There is no passive immunoprophylaxis (i.e., immune globulin) available for pre- or postexposure of plague (USAMRIID 2014).

Use of standard precautions for patients with bubonic and septicemic plague is indicated. Suspected pneumonic plague will require strict isolation with respiratory

Table 5.23 Recommended therapy and prophylaxis in children for diseases associated with bioterrorism

Disease	Therapy or prophylaxis	Treatment[a], agent and dosage
Smallpox	Therapy	Supportive care
	Prophylaxis	Vaccination may be effective if given with the first several days after exposure
Plague	Therapy	Gentamicin 2.5 mg/kg IV q8 h ***or*** Streptomycin 15 mg/kg IM q12 h (max 2 g/day, although only available for compassionate usage and in limited supply) ***or*** Doxycycline 2.2 mg/kg IV q12 h (max 200 mg/day) ***or*** Ciprofloxacin[b] 15 mg/kg IV q12 h ***or*** Levofloxacin 10–15 mg/kg IV q24 h ***or*** Chloramphenicol[c] 25 mg/kg q6 h (max 4 g/day)
	Prophylaxis	Doxycycline 2.2 mg/kg PO q12 h ***or*** Ciprofloxacin[b] 20 mg/kg PO q12 h
Tularemia	Therapy	Same as for plague
	Prophylaxis	Same as for plague
Botulism	Therapy	Supportive care, antitoxin and/or botulism immune globulin may halt progression of symptoms but are unlikely to reverse them
	Prophylaxis	None
Viral hemorrhagic fevers	Therapy	Supportive care, ribavirin may be beneficial in select cases[d]
	Prophylaxis	None

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References: (Berger and Burns 2012; Cieslak and Henretig 2016; Markenson and Redlener 2007; Pittman et al. 2018; USAMRIID 2014)
[a]In a mass casualty setting, parenteral therapy might not be possible. In such cases, oral therapy (with analogous agents) may need to be used
[b]Ofloxacin (and possibly other quinolones) may be acceptable alternatives to ciprofloxacin or levofloxacin; however, they are not approved for use in children
[c]Concentration should be maintained between 5 and 20 μg/mL. Some experts have recommended that chloramphenicol be used to treat patients with plague meningitis, because chloramphenicol penetrates the blood-brain barrier. Use in children younger than 2 may be associated with adverse reactions but might be warranted for serious infections
[d]Ribavirin is recommended for arenavirus or bunyavirus infections and may be indicated for a viral hemorrhagic fever of an unknown etiology although not FDA approved for these indications. For intravenous therapy use a loading dose: 30 kg IV once (max dose, 2 g), then 16 mg/kg IV q6 h for 4 days (max dose, 1 g), and then 8 mg/kg IV q8 h for 6 days (max dose, 500 mg). In a mass casualty setting, it may be necessary to use oral therapy. For oral therapy, use a loading dose of 30 mg/kg PO once, then 15 mg/kg/day PO in 2 divided doses for 10 days

droplet precautions for at least 48 h after initiation of effective antimicrobial therapy, or until sputum cultures are negative in confirmed cases. An N-95 respirator should be used for baseline protection (Table 5.25). It is also recommended to use an N-95 respirator or PAPR for high risk procedures (Beigel and Sandrock 2009; Carbone 2005; Centers for Disease Control and Prevention 2017; Centers for Disease Control and Prevention 2018b; Pittman et al. 2018; USAMRIID 2014).

5.7.4 Viral Hemorrhagic Fevers

Viral hemorrhagic fever has a variety of causative agents. However, the syndromes they produce are characterized by fever and bleeding diathesis. The etiologies include RNA viruses from four distinct families: Arenaviridae, Bunyaviridae, Filoviridae, and Flaviviridae. The Filoviridae (includes Ebola and Marburg) and Arenaviridae (includes Lassa fever and New World viruses) are Category A agents. Based on multiple identified characteristics, there is strong concern for the weaponization potential of the viral hemorrhagic fevers. Specifically, there has been demonstration of high contagiousness in aerosolized primate models.

There are five identified Ebola species, but only four are known to cause disease in humans. The natural reservoir host of Ebola virus remains unknown. However, on the basis of evidence and the nature of similar viruses, researchers believe that the virus is animal-borne and that bats are the most likely reservoir. Four of the five virus strains occur in an animal host native to Africa. Marburg virus has a single species. Geographic distribution of Ebola and Marburg is Africa (Fitzgerald et al. 2016). Both diseases are very similar clinically. Incubation period is typically 5–10 days with a range of 2–16 days. Symptoms may include fever, chills, headache, myalgia, nausea, and vomiting. There is rapid progression to prostration, stupor, and hypotension. The onset of a maculopapular rash on the arms and trunk is classic. Disseminated intravascular coagulation and thrombocytopenia develops with conjunctival injection, petechiae, hemorrhage, and soft tissue bleeding. There is a possible central nervous system and hepatic involvement. Bleeding, uncompensated shock, and multiorgan failure are seen. High viral load early in course is associated with poor prognosis. Death usually occurs during the second week of illness. Mortality rate of Marburg is 25–85% and for Ebola 50–90%.

In a retrospective cohort study of children during the 2014/2105 Ebola outbreak in Liberia and Sierra Leone (all less than 18 years with a median age of 7 years with one-third less than 5 years of age), the most common features upon presentation were fever, weakness, anorexia, and diarrhea. About 20% were initially afebrile. Bleeding was rare upon initial presentation. The overall case fatality rate was 57%. Factors associated with death included children less than 5 years of age, bleeding at any time during hospitalization, and high viral load (Smit et al. 2017). In another retrospective cohort study of children at two Ebola centers in Sierra Leone in 2014 (all less than 5 years of age), presenting symptoms included weakness, fever, anorexia, diarrhea, and cough. About 25% were afebrile on presentation. The case fatality rate was higher in children less than 2 years (76%) versus 2–5 years of age (46%) and 9 times more likely to die if child had a higher viral load. Signs associated with death included fever, emesis, and diarrhea. Interestingly, hiccups, bleeding, and confusion were only observed in children who died (Shah et al. 2016).

Lassa virus and New World viruses (Junin, Machupo, Sabia, and Guanarito) are transmitted from person-to-person. The vector in nature is the rodent. The incubation period is from 5 to 16 days. The geographical distribution is West Africa and South America, respectively. The South American hemorrhagic fevers are quite similar but differ from Lassa fever. The onset of the South American viruses is

insidious and results in high fever and constitutional symptoms. Petechiae or vesicular enanthem with conjunctival injection is common. These fevers are associated with neurologic disease (hyporeflexia, gait abnormalities, and cerebellar dysfunction). Seizures portend a poor prognosis. Mortality ranges from 15% to over 30%. On the contrary, Lassa viruses are mild. Less than 10% of infections result in severe disease. Signs include chest pain, sore throat, and proteinuria. Hemorrhagic disease is uncommon. Other features include neurologic disease such as encephalitis, meningitis, cerebellar disease, and cranial nerve VIII deafness (common feature). Mortality can be as high as 25%.

Differential diagnoses include malaria, meningococcemia, hemolytic uremic syndrome, thrombotic thrombocytopenic purpura, and typhoid fever. Diagnosis is through detection of the viral antigen testing by ELISA or viral isolation by culture at the CDC. No specific therapy is present and generally involves supportive care, especially mechanical ventilation strategies for ARDS, hemodynamic support, and renal replacement therapy. For the Arenaviridae and Bunyaviridae groups, ribavirin may be indicated (Table 5.24). For Ebola, there has been anecdotal success with immune serum transfusion. Additional potential therapies under investigation include favipiravir (Ebola), BCX4430 (Ebola, Marburg, and other viral hemorrhagic fevers), interferons (IFN α-2b or IFN-β for viral hemorrhagic fevers), and small molecule therapeutics (antisense phosphorodiamidate morpholino oligomers [AVI-6002, AVI-6003] and lipid nanoparticle/small interfering RNA [TKM-Ebola]) (Pittman et al. 2018).

There is no current vaccine for Ebola that is licensed by the FDA. An experimental vaccine called rVSV-ZEBOV was found to be highly protective against Ebola virus in a trial conducted by the World Health Organization (WHO) and other international partners in Guinea in 2015. FDA licensure for the vaccine is expected in 2019. Until then, 300,000 doses have been committed for an emergency use stockpile under the appropriate regulatory mechanism in the event and an outbreak occurs before FDA approval is received (Centers for Disease Control and Prevention 2019b; Henao-Restrepo et al. 2015). Another Ebola vaccine candidate, the recombinant adenovirus type-5 Ebola vaccine, was evaluated in a phase 2 trial in Sierra Leone in 2015. An immune response was stimulated by this vaccine within 28 days of vaccination and

Table 5.24 Recommended ribavirin dosing for treatment of viral hemorrhagic fevers[a]

		Intravenous	Oral
Adults	Loading dose	30 mg/kg IV (max 2 g once)	2000 mg PO once
	Maintenance dose	Day 1–4: 16 mg/kg IV (max 1 g) q6 h for 4 days Day 5–7: 8 mg/kg IV (max 500 mg) q8 for 6 days	Weight > 75 kg: 600 mg PO BID for 10 days Weight < 75 kg: 400 mg PO qAM, 600 mg PO in PM for 10 days
Children	Loading dose	Same as adult	30 mg/kg PO once
	Maintenance dose	Same as adult	7.5 mg/kg PO BID for 10 days

[a]For confirmed or suspected arenavirus or bunyvirus or viral hemorrhagic fever of unknown etiology (USAMRIID 2014)

Table 5.25 Isolation precautions for critical biologic agents of terrorism

Disease	Isolation precautions
Anthrax (inhalational)	Standard
Plague (pneumonic)	Droplet (for first 3 days of therapy)
Tularemia	Standard
Smallpox	Airborne plus contact
Botulism	Standard
Viral hemorrhagic fevers	Contact (consider airborne in cases of massive hemorrhage)

References: (Centers for Disease Control and Prevention 2019a; Cieslak and Henretig 2016)

the response decreased over 6 months after injection. Research on this vaccine is ongoing (Centers for Disease Control and Prevention 2019b; Zhu et al. 2017).

Strict contact precautions (hand hygiene, double gloves, gowns, shoe and leg coverings, and face shield or goggles) and droplet precautions (private room or cohorting, surgical mask within 3 ft) are mandatory for viral hemorrhagic fevers. Airborne precautions (negative-pressure isolation room with 6–12 air exchanges per h) should also be instituted to the maximum extent possible and especially for procedures that induce aerosols (e.g., bronchoscopy). At a minimum, a fit-tested, HEPA filter-equipped respirator (e.g., an N-95 mask) should be used, but a battery-powered PAPR or a positive pressure-supplied air respirator should be considered for personnel sharing an enclosed space with, or coming within 6 ft of, the patient. Multiple patients should be cohorted in a separate ward or building with a dedicated air-handling system when feasible (Table 5.25). Environmental decontamination is accomplished with hypochlorite or phenolic disinfectants (Beigel and Sandrock 2009; Radoshitzky et al. 2018; USAMRIID 2014; Won and Carbone 2005).

5.7.5 Tularemia (*Francisella tularensis*)

Francisella tularensis, a small aerobic, nonmotile gram-negative coccobacillus, causes tularemia (rabbit fever). Clinical disease is caused by two isolates, Biovars Jellison Type A and B. This organism can be stabilized for weaponization and delivered in a wet or dry form. The incubation period is usually 3–6 days (range 1–21 days). Initial symptoms are nonspecific and mimic the flu-like symptoms or other upper respiratory tract infections. There is acute onset of fever with chills, myalgias, cough, fatigue, and sore throat. The two clinical forms of tularemia are typhoidal and ulceroglandular diseases.

Typhoidal tularemia (5–15%) occurs after inhalational exposure and sometimes intradermal or gastrointestinal exposures. There is abrupt onset of fever, headache, malaise, myalgias, and prostration. It presents without lymphadenopathy. Nausea, vomiting, and abdominal pain are sometimes present. Untreated, there is a 35% mortality rate in naturally acquired cases (vs. 1–3% in those treated). It is higher if pneumonia is present. This form would be most likely seen during an aerosol release of the agent.

Ulceroglandular tularemia (75–85%) occurs through skin or mucus membrane inoculation. There is abrupt onset of fever, chills, headache, cough, and myalgias along with a painful papule at the site of exposure. The papule becomes a painful ulcer with tender regional lymph nodes. Skin ulcers have heaped up edges. In 5–10%, there is focal lymphadenopathy without an apparent ulcer. Lymph nodes may become fluctuant and drain when receiving antibiotics. Without treatment, they may persist for months or even years.

In some cases (1–2%), the primary entry port is the eye leading to oculoglandular tularemia. Patients have unilateral, painful, and purulent conjunctivitis with local lymphadenopathy. Chemosis, periorbital edema, and small nodular granulomatous lesions or ulceration may be found. Oropharyngeal tularemia with pharyngitis may occur in 25% of patients. Findings include exudative pharyngitis/tonsillitis, ulceration, and painful cervical lymphadenopathy. The differential diagnosis is antibiotic unresponsive pharyngitis, infectious mononucleosis, and viral pharyngitis. Pulmonary involvement (47–94%) is seen in naturally occurring disease. It ranges from mild to fulminant. Various processes include pneumonia, bronchiolitis, cavitary lesions, bronchopleural fistulas, and chronic granulomatous diseases. Left untreated, 60% will die.

Differential diagnoses include those for typhoidal (typhoid fever, rickettsia, and malaria) or pneumonic (plague, mycoplasma, influenza, Q-fever, and staphylococcal enterotoxin B) tularemia. Diagnosis should be considered when there is a cluster of nonspecific, febrile, systemically ill patients who rapidly progress to fulminant pneumonitis. Tularemia can be diagnosed by recovering the organism from sputum (PCR or DFA) or serology at a state health laboratory. Chest radiograph is nonspecific with possible hilar adenopathy. Treatment is streptomycin or gentamicin (Table 5.23). Alternatives include doxycycline, ciprofloxacin, or chloramphenicol. A live-attenuated vaccine (NDBR 101) exists and typically used for laboratory personnel working with *Francisella tularensis*. There is no passive immunoprophylaxis. Ciprofloxacin or doxycycline can be given as pre- and postexposure prophylaxis (Beigel and Sandrock 2009; Hepburn et al. 2018; Pittman et al. 2018; USAMRIID 2014).

5.7.6 Botulinum Toxin (*Clostridium botulinum*)

Botulinum neurotoxins (BoNT) are produced from the spore-forming, Gram-positive, obligate anaerobe *Clostridium botulinum*. It is the most potent toxin known to man. A lethal dose is 1 ng per kilogram. It is 100,000 times more toxic than sarin (GB). There are seven serotypes of botulinum toxin (A through G). A new serotype (H) has been tentatively identified in a case of infant botulism but has not been fully investigated. Most common are serotypes A, B, and E. The toxin acts on the presynaptic nerve terminal of the neuromuscular junction and cholinergic autonomic synapses. This disrupts neurotransmission and leads to clinical findings. There are three forms of botulism: foodborne, wound, and intestinal (infant or adult intestinal). Botulinum toxin can also be released as an act of bioterrorism via ingestion or aerosol forms.

Incubation can be from 12 h after exposure to several days later. Clinical findings of botulism include cranial nerve palsies such as ptosis, diplopia, and dysphagia. This is followed by symmetric descending flaccid paralysis. However, the victim remains afebrile, alert, and oriented. Death is typically due to respiratory failure. Prolonged respiratory support is often required (1–3 months).

Differential diagnoses include Guillain-Barre syndrome, myasthenia gravis, tick paralysis, stroke, other intoxications (nerve gas, organophosphates), inflammatory myopathy, congenital and hereditary myopathies, and hypothyroidism. Diagnosis is mostly clinical. Laboratory confirmation can be obtained by bioassay of patient's serum. Other assays include immunoassays for bacterial antigen, PCR for bacterial DNA, and reverse transcriptase-PCR for mRNA to detect active synthesis of toxin. Cerebrospinal fluid demonstrates normal protein (unlike Guillain-Barre syndrome). EMG reveals augmentation of muscle action potential with repetitive nerve stimulation at 20–30 Hz.

Treatment (Table 5.23) is mainly supportive including intubation and ventilator support. Tracheostomy may be required due to prolonged respiratory weakness and failure. Antibiotics do not play a role in treatment. Botulism Antitoxin Heptavalent [A, B, C, D, E, F, G]-Equine (BAT) was approved by the FDA in 2013. BAT was developed at USAMRIID as one of two equine-derived heptavalent BoNT antitoxins. BAT is approved to treat individuals with symptoms of botulism following a known or suspected exposure. It has the potential to cause hypersensitivity reactions in those sensitive to equine proteins. The safety of BAT in pregnant and lactating women is unknown. Evidence regarding safety and efficacy in the pediatric population is limited. In 2003, the FDA approved Botulinum Immune Globulin Intravenous (BabyBIG), a human botulism immune globulin derived from pooled plasma of adults immunized with pentavalent botulinum toxoid. It is indicated for the treatment of infants with botulism from toxin serotypes A and B. Immediately after clinical diagnosis of botulism, adults (including pregnant women) and children should receive a single intravenous infusion of antitoxin (BAT or, for infants with botulism from serotypes A or B, BabyBIG) to prevent further disease progression. The administration of antitoxin should not be delayed for laboratory testing to confirm the diagnosis. The pentavalent toxoid vaccine (previously for protection against A, B, C, D, and E; but not F or G) is no longer available as of 2011. No replacement vaccine is currently available. Standard isolation precautions (Table 5.25) should be followed (Beigel and Sandrock 2009; Dembek et al. 2018; Pittman et al. 2018; Timmons and Carbone 2005; USAMRIID 2014).

5.7.7 Ricin (from *Ricinus communis*)

Ricin is a potent cytotoxin derived from the castor bean plant *Ricinus communis*. It is related in structure and function to Shiga toxins and Shiga-like toxin of *Shigella dysenteriae* and *Escherichia coli*, respectively. It consists of two glycoprotein subunits, A and B, connected by a disulfide bond. The B-chain allows the toxin to bind to cell receptors and gain entrance into the cell. Once ricin enters the

cell, the disulfide chemical linkage is broken. The free A chain then acts as an enzyme and inactivates ribosomes thereby disrupting normal cell function. Cells are incapable of survival and soon die. Ricin has a high terrorist potential due to it characteristics: readily available, ease of extraction, and notoriety (Maman and Yehezkelli 2005).

Three modes of exposure exist: oral, inhalation, and injection. Four to eight hours after inhalation exposure, the victim develops fever, chest tightness, cough, dyspnea, nausea, and arthralgias. Airway necrosis and pulmonary capillary leak ensues within 18–24 h. This is followed quickly by severe respiratory distress, ARDS, and death due to hypoxemia within 36–72 h. Injection may cause minimal pulmonary vascular leak. Pain at the site and local lymphadenopathy may occur. However, it may be followed by nausea, vomiting, and gastrointestinal hemorrhage. Ingestion leads to necrosis of the gastrointestinal mucosa, hemorrhage, and organ necrosis (spleen, liver, and kidney).

Diagnosis is suspected when multiple cases of acute lung injury occur in a geographic cluster. Serum and respiratory secretions can be checked for antigen using ELISA. Pulmonary intoxication is managed by mechanical ventilation. Gastrointestinal toxicity is managed by gastric lavage and use of cathartics. Activated charcoal has little value due to the size of ricin molecules. Supportive care is indicated for injection exposure. In general, treatment is largely supportive, especially for pulmonary edema that can result from the capillary leak. There is no vaccine available or prophylactic antitoxin for human use. However, there are two ricin vaccines in the development that focus on the ricin toxin A (RTA) chain subunit. A mutant recombinant RTA chain vaccine, RiVax, has been shown to be safe and immunogenic in humans. The other vaccine is another recombinant RTA chain vaccine, RVEc (RTA 1–33/44–198). It has shown effectiveness in animal models by producing protective immunity against aerosol challenge with ricin in animal models. Standard precautions are advised for health care workers (Pittman et al. 2018; Roxas-Duncan et al. 2018; Traub 2005; USAMRIID 2014).

5.8 Radiological/Nuclear

Recent events which include the nuclear reactor meltdown at Fukushima and international tension between nuclear powers, spark concern over potential devastation from nuclear catastrophes. There are numerous examples of radiation disasters in history. Sixty-six thousand people were killed in Hiroshima and thirty-nine thousand people were killed in Nagasaki from nuclear bombs detonated over these cities in 1945 (Avalon Project—Documents in Law, History and Diplomacy n.d.). Many other people suffered from long-term consequences of radiation poisoning. In 1986, 21,000 square kilometers of land in Russia, Ukraine, and Belarus were contaminated with radiation from a meltdown at a nuclear power plant in Chernobyl, Ukraine. One hundred and thirty-five thousand people were permanently evacuated from their homes (Likhtarev et al. 2002). Long-term health consequences included many children who developed thyroid cancer several years later. Many of these children died.

A tsunami pummeled the east coast of Japan in March of 2011. The power outage that ensued at the Fukushima power plant led to a failure of the cooling system of the fuel rods, leading to a meltdown of four of the reactors at the plant. A massive quantity of radiation was released into the atmosphere, forcing people to evacuate their homes indefinitely. Creative thinking and heroic actions by the Tokyo fire department prevented entire populations of cities from being poisoned with radiation.

Terrorism experts are concerned that terrorist organizations will produce and detonate a radiological dispersion device (RDD), sometimes referred to as a dirty bomb. This is a conventional explosive, loaded with radioactive material which would be dispersed upon detonation. This would likely involve only one radioisotope. Fewer people would be exposed and a smaller area would be contaminated than what would transpire with the detonation of a nuclear weapon. Spreading fear and panic would be the primary purpose of such a device (Mettler Jr and Voelz 2002).

5.8.1 Principles of Ionizing radiation

Radiation is the emission and propagation of energy through space or through a medium in the form of waves. Radiation can be ionizing or nonionizing depending on the amount of energy released. Most radiation that people encounter is low energy and, therefore, nonionizing with no biological effects. Ionizing radiation emits enough energy to strip electrons from an atom, which provokes cellular changes and thereby, results in biological effects. Radiation emitted from nuclear decay is always ionizing (Radiation Emergency Assistance Center/Training Site (REACT/S-CDC) 2006).

Atomic nuclei are held together by a very powerful binding energy despite positively charged protons repelling each other. This energy is released from unstable nuclei in the form of electromagnetic waves or particles. When ionizing radiation reaches biological tissue, chemical bonds are disrupted, free radicals are produced, and DNA is broken.

Electromagnetic waves are of two types, X-rays and gamma rays. X-rays are relatively low energy and less penetrating. Gamma rays have a shorter wavelength and contain relatively higher energy, making them more penetrating of biological tissue. Ionizing radiation in the form of particles consists of alpha particles, beta particles, and neutrons. Alpha particles are the largest of the forms of particulate radiation. They are composed of two neutrons and two protons. They do not easily penetrate solid surfaces, including clothes and skin. However, they can cause severe damage to an organism if internalized. In 2006, in the United Kingdom, Alexander Litvienko, an ex KGB agent was poisoned with a radioactive element called polonium (McPhee and Leikin 2009). A small amount of polonium was sprinkled into his food. Polonium releases alpha particles when it decays. It was relatively safe for the assassin to carry this element with him because of the relatively poor ability of alpha particles to penetrate clothing and skin. Once it is ingested, however, alpha particles have profound biological effects. Mr. Litvienko became very ill, and ultimately died.

Beta particles are high energy electrons discharged from the nucleus and are highly penetrating. Neutrons emitted from a nucleus are also highly penetrating. In general, neutrons are only released by the detonation of a nuclear weapon.

Ionizing radiation of any form cannot be detected by our senses. It is not smelled, felt by touch, tasted, or seen. It is possible to be exposed to a lethal dose of radiation without realizing it. In Goiania, Brazil, in 1987, children found a canister of radioactive Cesium (^{137}Cs) that had been looted from a medical center and left in the street. The children liked the appearance of the substance but were not able to sense any abnormalities or danger with it. They began to rub it on their bodies because they liked the way it made them glow in the dark. The children all became ill. Ultimately, 250 people were exposed to this radioisotope. It took 10 days before physicians recognized that the people had radiation poisoning. Four people died of acute radiation syndrome.

Four factors determine the severity of exposure to ionizing radiation: time, distance, dose, and shielding. Time is the time of exposure to the radiation source. Distance is the distance from the radiation source. Based on the inverse square law, exposure is reduced exponentially with increasing distance from the radiation source. Dose is measured by the amount of energy released by the source and is numerically described by how many disintegrations per second occur, in Curies (Ci) or Becquerels (Bq). Shielding is the efficacy of the barrier to the radioactive source. Lead is well-known to be a very effective shield to X-rays. In a radiation exposure, injury to skin from trauma or burns may cause a greater degree of contamination because of loss of the shielding of the skin.

There are four important principles for the nurse or HCP to understand with regard to exposure to ionizing radiation: external exposure, external contamination, internal contamination, and incorporation. External contamination occurs when radioactive material is carried on a person after exposure. This person can then contaminate others. Removing contaminated clothing eliminates 90% of the toxin. Others are then less vulnerable to exposure. Internal contamination is when a radioactive substance enters the body through inhalation, ingestion, or translocation through open skin. Incorporation is internalization of the toxin into body organs. Incorporation is dependent on the chemical and not the radiological properties of the radioactive toxin. Radioactive iodine, ^{131}I, is taken up by the thyroid gland because iodine enters the gland as part of normal physiology (Advanced Hazmat Life Support (AHLS) 2003).

5.8.2 Biological and Clinical Effects

Ionizing radiation can damage chromosomes directly and indirectly, causing ravaging biological effects. Indirect damage comes from the production of H^+ and OH^-. Free radical formation upsets biochemical processes and causes inflammation. These effects can take anywhere from seconds to hours to be expressed. Clinical changes can take from hours to years to be realized (Zajtchuk et al. 1989).

Immediately after a major radiation exposure, the clinical matters of most concern are those related to trauma from blast and thermal injuries. These injuries may be life-threatening and must be addressed first.

After thermal and traumatic injuries are addressed, attention should be paid to the severity of radiation exposure. Severe exposure can cause acute radiation syndrome. "The acute radiation syndrome is a broad term used to describe a range of signs and symptoms that reflect severe damage to specific organ systems and that can lead to death within hours or up to several months after exposure" (National Council on Radiation Protection (NCRP) and Measurements 2001; National Council on Radiation Protection (NCRP) and Measurements 2009). The mechanism of cell death from toxic radiation exposure is related to the inhibition of mitosis. Organs with the most rapidly dividing cells are the most susceptible. The gastrointestinal and the hematopoietic are the organ systems most notably affected. The organs of pediatric patient have a higher mitotic index, in general, to those of adults and are more vulnerable to injury from radiation poisoning. The time of onset and the severity of acute radiation syndrome are controlled by the total radiation dose, the dose rate, percent of total body exposed, and associated thermal and traumatic injuries. There is a 50% death rate (LD_{50}) within 60 days for people exposed to a dose of radiation of 2.5–4.0 Gy. The LD_{50} is lower for the pediatric population.

The acute radiation syndrome is composed of four phases: prodromal, latent, manifest illness, and death or recovery. Inflammatory mediator release during the prodromal phase causes damage to cell membranes. This phase occurs during the first 48 h after exposure to radiation. Nausea and vomiting and fever can occur during this time. If these symptoms occur during the first 2 h after exposure, there is a poor prognosis.

The onset of the latent phase is usually in the first 4 days post exposure but can ensue anytime during the first 21 days thereafter. All cell lines of the hematopoietic system are affected. Lymphocytes and platelets, the most rapidly dividing cells of the bone marrow, are most severely affected.

The illness phase manifests after 30 days since radiation exposure. Infection, impaired wound healing, anemia, and bleeding occur during this time of illness. The hematopoietic, gastrointestinal, central nervous, and integumentary are the organ systems affected. There is a marked reduction of cells from all cell lines of the bone marrow. There is a direct correlation with the drop in absolute lymphocyte count with the dose of radiation received. The absolute lymphocyte count is commonly used to estimate the dose of radiation received.

The gastrointestinal (GI) epithelial lining, one of the most rapidly dividing cell lines of the body is the second most vulnerable to radiation poisoning. The radiation dose required to affect the GI system is 8 Gy. Vomiting, diarrhea, and a capillary leak syndrome for GI tract are common manifestations. Hypovolemia and electrolyte instability ensue. Translocation of bacteria into the bloodstream, combined with the diminished immunity caused by the decimation of the hematopoietic system, place victims at high risk for septic shock.

Another organ system affected by the acute radiation syndrome is the central nervous system. This requires a large dose of at least 30 Gy. Manifestations include

cerebral edema, disorientation, hyperthermia, seizures, and coma. Acute radiation syndrome that involves the central nervous system is always fatal.

The integumentary system is frequently affected by the acute radiation syndrome, especially if the skin is in direct contact with a radioisotope. Epilation, erythema, dry desquamation, wet desquamation, and necrosis occur respectively with increasing severity associated with increasing doses of radiation. Radiation burns can be distinguished from thermal or chemical burns by their delayed onset. It can take days to weeks for radiation burns to affect victims. Thermal and chemical burns cause signs and symptoms more acutely.

5.8.3 Immediate Clinical Management

Hospitals that anticipate victims of radiation should prepare areas of triage with decontamination supplies and techniques ready to be deployed. An Emergency Department (ED) should be divided into "clean" and "dirty" areas. The dirty area is created for the purpose of decontamination to prevent the spread of radioisotopes.

All health care personnel should wear PPE including surgical scrubs and gowns, face shields, shoe covers, caps, and two pairs of gloves. The inner pair of gloves is taped to the sleeves of the gown. Each health care worker should be monitored for the exposure of the radiation and its dose with a dosimeter worn underneath the gown. The radiation safety officer of the hospital should take a leadership role in health care worker protection and decontamination procedures. Consultation from the Radiation Emergency, Assistance Center (REACT/TS) is imperative. REACT/TS is a subsidiary of the U.S. Department of Energy. Its contact information is as follows:

Phone number during business hours is 865-576-3131. The phone number is 865-576-1005 after business hours. The REACT/TS website is http://orise.orau.gov/reac/ts/.

As victims arrive, triage protocols of mass casualty scenarios should be implemented. It should be noted that radiation exposure is not "immediately" life-threatening. Initial clinical management should focus on the ABCDE (Airway, Breathing, Circulation, Disability, and Exposure) of basic trauma protocol. The "D" in the above acronym can also be a symbol for decontamination. After airway, breathing, and circulation are addressed, initial phase of decontamination entails careful removal of potentially contaminated clothing. Caution should be exercised to remove the clothing gently, while rolling garments outward to prevent the release of dust of radioactive material that could contaminate people in the treatment area. Further decontamination procedures take place after initial stabilization.

Skin decontamination procedures are identical to those of toxic chemical exposure with the following exceptions:

- PPE are slightly different as described above.
- Gentle skin rubbing is done to prevent provocation of an inflammatory response and further absorption of the radioactive toxin.

- Only soap and water are used. Rubbing alcohol and bleach should be avoided. It is advisable to shampoo the hair first, because it is usually the site of the highest level of contamination of the body, and runoff onto the body can then be cleansed during skin decontamination (Radiation Event Medical Management (REMM) of the U.S. Dept. of Health and Human Services n.d.).

It should be noted that health care workers are not at risk for contamination if they wear proper PPE during the resuscitation and decontamination process. The lack of knowledge of this point may lead to reluctance to treat patients and increase morbidity and mortality for victims. "No HCP has ever received a significant dose of radiation from handling, treating, and managing patients with radiation injuries and/or contamination."(REACT/S-CDC 2006).

5.8.4 Ongoing Clinical Management

When initial resuscitation and decontamination have been completed, attention should be paid to ongoing support of ventilation, oxygenation, the management of fluid and electrolytes, and treatment of traumatic and burn injuries. Infection control procedures are important due to the impending immunocompromised state of the victims.

It is important to ascertain the details of the catastrophic event. Data on the nature and size of the exposure and the types of radioactive agents involved are vital for ongoing management and decontamination.

After the details of the nature of the exposure are uncovered, diagnostic tests should be done, including serial CBC and cytogenetic analysis of lymphocytes, otherwise known as cytogenic dosimetry (REACT/S-CDC 2006). Measurements of change in lymphocyte counts and cytogenetic dosimetry are sensitive markers for the dose of radiation received by a victim. Measurements of internal decontamination are done by the sampling and analysis of nasal and throat swabs, stool, and 24 h urine. Wound samples and irrigation fluid should also be sampled.

5.8.5 Treatment of Internal Contamination

After initial stabilization, external decontamination, and diagnostic testing, internal decontamination is performed. External decontamination involves removal of clothes and cleaning the skin and hair. Internal decontamination removes radioisotopes that are internalized via inhalation, ingestion, and entry into open wounds. Because ionizing radiation is being released inside the body, internal decontamination must be performed promptly after initial resuscitation. Since radioisotopes behave identically to their nonradioactive counterparts, antidotes are chosen based on the chemical, and not the radiological properties of the element. Basic strategies of internal decontamination include chelation, competitive inhibition, enhanced gastrointestinal elimination, and enhanced renal elimination.

Specific agents are used for chelation of different radioisotopes. DTPA (diethyenetriaminepentaacetic acid) is administered for the elimination of heavy metals such as americium, californium, curium, and plutonium. DTPA comes in two forms, Calcium DTPA (Ca-DTPA) and Zinc-DTPA (Zn-DTPA). Ca-DTPA is ten times more effective than Zn-DTPA. For adults and adolescents, administration is as follows:

- 1 g of Ca-DTPA IV initially in the first 24 h, followed by 1 g Zn-DTPA IV daily for maintenance.
- For children less than 12 years of age administer:
- Fourteen mg/kg Ca-DTPA IV initially, followed by fourteen mg/kg of Zn-DTPA IV daily thereafter (National Council on Radiation Protection (NCRP) and Measurements 2009).
- The initial dose of DTPA may be administered via inhalation to adolescents and adults if the contamination occurred via inhalation. This method of administration is not approved for pediatric use.

Chelation with dimercaprol (BAL) is used to eliminate polonium. BAL is a highly toxic drug and should be administered with caution.

The dose is 2.5 mg per kg IM four times a day for 2 days, then twice a day on the third day and once a day for 5–10 days, thereafter (National Council on Radiation Protection (NCRP) and Measurements 2009). Alkalinization of the urine is renal protective during administration. A less toxic alternative to BAL, Dimercaptosuccinic acid (DMSA), otherwise known as Chemet® is also available. The dose of DMSA is ten mg per kg po every 8 h for 5 days. The same dose is given every 12 h for 14 days, thereafter (National Council on Radiation Protection (NCRP) and Measurements 2009).

Another mechanism for internal decontamination is competitive inhibition. The radioisotope, ^{131}I, is released during a meltdown of a reactor at a nuclear power plant. Potassium iodide (KI) is widely recognized as a competitive inhibitor to its radioactive counterpart, ^{131}I, from being incorporated into the thyroid gland. KI blocks 90% of ^{131}I uptake into the thyroid gland if KI is given within the first hour of exposure. It will block 50% of incorporation if given within 5 h of exposure. Its protective effect lasts for 24 h. With administration of this drug, thyroid function should be monitored closely. Dosing guidelines (Table 5.26) are included in the table below (U.S. Food and Drug Administration n.d.).

Gastrointestinal elimination is another mechanism of internal decontamination (Table 5.27). An ion exchanger, Prussian Blue, (ferric ferrocyanide), binds elements that circulate through the enterohepatic cycle. Since it is not absorbed through the gastrointestinal tract, Prussian Blue carries the toxins into the stool. It is highly effective in the elimination of ^{137}Cs or thallium and was used during the ^{137}Cs incident in Goiania, Brazil. The dosing of Prussian Blue is as follows:

- Infants: 0.2–0.3 mg per kg po three times a day (not FDA approved).
- Children 2–12 years of age: 1 g po three times a day.
- Children ≥12 years of age: 3 g po three times a day.

Table 5.26 Potassium Iodide (KI) dosing for ^{131}Iodine contamination

	Predicted thyroid exposure, Gy	KI dose, mg	No. of 130-mg tablets	No. of 65-mg tablets
Adults >40 years	≥5	130	1	2
Adults >18 to 40 years	≥0.1	130	1	2
Pregnant/lactating women	≥0.05	130	1	2
Adolescents >12 to 18 years	≥0.05	65	½	1
Children >3 to 12 years	≥0.05	65	½	1
Children >1 month to 3 years	≥0.05	32	¼	½
Children birth to 1 month	≥0.05	16	1/8	¼

Reproduced with permission from the US Department of Health and Human Services, Food and Drug Administration, Center for Drug Evaluation and Research (U.S. Food and Drug Administration n.d.)

Table 5.27 Table of most common radionuclides and decontamination strategies

Radionuclides	Organ of incorporation	Decontamination strategy	Antidote
Heavy metals: Americium Californium Curium Plutonium	Bone	Chelation	IV Ca-DTPA Or IV Zn-DTPA
Polonium	Total body	Chelation	BAL with IV alkalinization Or IM DMSA
^{131}Iodine	Thyroid	Competitive inhibition	PO Potassium Iodide
^{137}Cesium	Total body	Enhanced GI elimination	PO Prussian Blue
Tritium	Total body	Enhanced urinary elimination	IV fluids
Uranium	Kidneys, bone	Alkalinization of urine	Sodium Bicarbonate

Doses are included in the text (Jacobson and Severin 2012)

- Prussian Blue is administered for at least 30 days, and can be adjusted based on the degree of poisoning (National Council on Radiation Protection (NCRP) and Measurements 2009).

Urinary elimination is another useful method of internal decontamination. Tritium can be eliminated with excess fluid administration. Uranium is eliminated by alkalinizing the urine to a pH of 8–9. Sodium bicarbonate is given at a dose of 1 mEq/kg IV every 4–6 h and is titrated to effect. If renal injury occurs, dialysis may be required.

5.8.6 Treating Acute Radiation Syndrome

The basic approach to treating acute radiation syndrome is supportive therapy. GI losses from gastrointestinal difficulties are treated with IV fluids and electrolyte replacement. 5-HT3 antagonists can be used to suppress vomiting and benzodiazepines for anxiety. A patient suffering from acute radiation syndrome may be severely immunocompromised and requires a room with positive pressure isolation. Colony stimulating agents for granulocytes and erythrocytes can be used for bone anemia and leukopenia. Bone marrow transplant may be required for severe cases.

5.8.7 Treatment of Local Skin Contamination

A patient with skin contamination with radiation should be decontaminated with soap and water. A Geiger counter can be helpful to identify areas of contamination. Scrubbing is performed in a concentric matter, beginning at the outer layers of contamination and moving into the center since the area of greatest contamination is in the center. In this way, the area of contamination remains contained. Attention should be paid to good nutrition and pain control. Burn and plastic surgery service should also be consulted. More details on decontamination can be found in Chap. 9.

5.8.8 Psychosocial Implications

The psychological impact of a radiation catastrophe on the pediatric victims is likely to be devastating (American Academy of Pediatrics (AAP) 2003). Sleep disturbances, social withdrawal, altered play, chronic fear and anxiety, and developmental regression can occur. A correlation between the parent's psychological response and that of the child would occur as with other types of disaster. Mental health professionals should be consulted in the event of this type of situation. Please refer to Chap. 12 for more information.

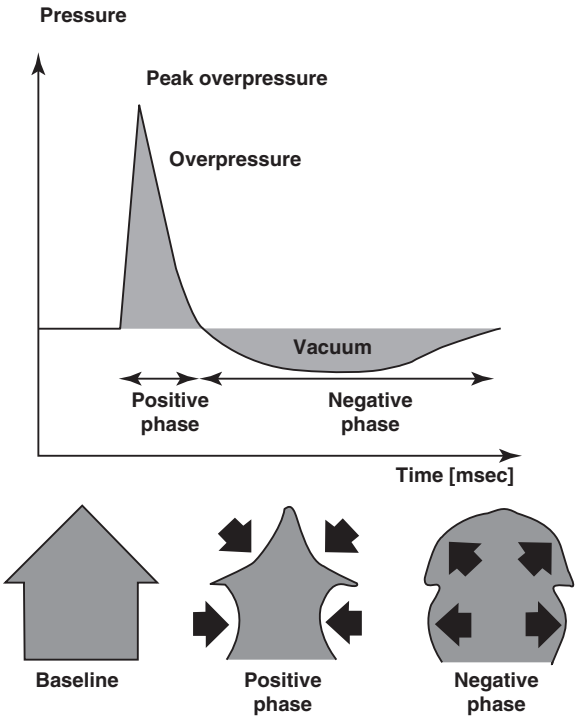
5.9 Explosives

A lot of concern has been expressed over the possibility of terrorist attacks involving explosive devices in recent years (DePalma et al. 2005). Explosive devices are relatively simple to manufacture and easy to detonate. They can injure and kill many people and spread fear over large populations. Victims of bomb blasts sustain more body regions injured, have more body injury severity scores, and require more surgeries than victims of nonexplosive trauma incidents. Victims of explosives also have a higher mortality (Kluger et al. 2004). These observations are also true of pediatric victims (Daniel-Aharonson et al. 2003).

Many factors influence the number of people injured and the severity of the injuries in an explosion. The magnitude of the explosion and its proximity to people and the number of people in the area affect the severity and number of injuries. Other factors include the collapse of building or structure from the blast, promptness of the rescue operation, and the caliber and proximity of medical resources in the vicinity. Victims who experience explosions in closed spaces are especially vulnerable to more severe injuries. Twenty-nine case reports of injuries from terrorist bombings were reviewed (Arnold et al. 2004a). The investigators compared the injury severity of victims of explosions who sustained injuries from structural collapse, closed space explosions without structural collapse, and open space explosions. The mortality rate for these victims was 25%, 8%, and 4%, respectively. Hospitalization rates were 25%, 36%, and 15%, respectively. ED visits were 48%, 36%, and 15%, respectively. Victims of closed space explosions without structural collapse experienced greater hospitalizations rates than those involved in a structural collapse, because many of the victims involved in the structural collapse experienced immediate death.

5.9.1 Basics of Explosions

An explosion is defined as a rapid chemical conversion of a liquid or solid into a gas with energy release. Substances that are chemically predisposed to explosion, called explosives, are characterized as low or high order, depending on the speed and magnitude of energy release. Low-order explosives release energy at a relatively slow pace and explosions from these substances tend not to produce large air pressure changes or a "blast." The energy release is caused by combustion, producing heat. The involved material "goes up in flames." Gunpowder, liquid fuel, and Molotov cocktails are examples of low-order explosives (Centers for Disease Control and Prevention 2010). Explosions from high-order materials cause a blast with a pressure wave in addition to causing the release of heat and light. The blast pressure wave causes compression of the surrounding medium which is physically transformed in all directions from the exact point of explosion. When an explosion occurs on land, air is the surrounding medium compressed. In bodies of water, the surrounding medium is water. The degree of medium compression and the distance that the energy wave travels is determined by the magnitude of the explosion. The power of the blast is measured in pounds per square inch (psi). The pressure blast wave has distinctive characteristics. The amplitude of the wave reaches its highest point immediately after the blast. The blast wave then rapidly decays as it travels through space. As the blast wave propagates, and compresses the surrounding medium, it leaves a vacuum because of displaced molecules in the surrounding medium and a negative phase of the wave ensues. In a land explosion, air molecules are displaced by the initial positive pressure, after which a negative pressure occurs in the vacated space. A wave that propagates through a confined space rebounds off of the wall and reverberates. It may interact with victims in the confined space many times, causing more severe injuries (Stuhmiller et al. 1991) (Fig. 5.10).



Originally described by Friedlander, a blast wave consists of a short, high-amplitude overpressure peak followed by a longer depression phase. Injury potential depends on the wave's amplitude as well as the slopes of its increase and decrease in pressure. X-axis refers to time and Y-axis refers to pressure.

Fig. 5.10 Blast Wave Physics: Friedlander Blast Wave*. *Used with permission Society of Critical Care Medicine (Jacobson and Severin 2012)

5.9.2 Mechanisms of Injury

Four kinds of injury occur in high energy explosions. Primary blast injuries occur directly from the pressure wave of the blast. Secondary injuries occur from being struck by flying objects from the blast. These injuries can be blunt or penetrating. Tertiary injuries occur when victims are displaced from a location and strike other objects or surfaces. All other injuries related to the blast are called quarternary. They include burns, inhalational injuries, toxic exposures, and traumatic injuries from structural collapse.

5.9.3 Clinical Manifestations of Primary Blast Injuries

Primary injuries from blast waves affect bodily tissues with a tissue gas interface. When a pressure wave enters the body, tissue of gas filled organs compress slower than the air inside the tissue, causing stress in the tissue, possibly damaging it. This

also known as the "spalling effect." As the negative pressure phase of the blast wave propagates through, it causes more stress on the tissue and further damage.

In addition to damaging tissues with an air tissue interface, pressure blasts can cause injury to the brain and can lead to limb detachments. Despite the fact that primary blast injuries can be ravaging, they are less common than other types of injury from blasts. The tympanic membranes, lungs, and gastrointestinal tract are the most common organs sustaining injury from pressure waves. The tympanic membrane is the most vulnerable of these three organ systems (DePalma et al. 2005; Garth 1997). Five psi, which is considered a weak blast, will rupture 50% of tympanic membranes. To put this in perspective, C4, a commonly used explosive generates a pressure of four million psi. Otoscopy can reveal ruptured tympanic membranes. Neuropraxia, deafness, tinnitus, and vertigo are symptoms that can be experienced. Severe blast injuries of the ear can result in damage to the organ of Corti, resulting in permanent hearing loss.

The second most common organ injured from a blast wave is the lung. Fifteen psi are required to cause injury to this organ. Lung injuries are more likely to occur from a blast within a closed space, or when victims sustain burns (burns commonly cause acute lung injury from release of inflammatory mediators). Direct alveolar damage, blood vessel with bleeding, and inflammation are the three different manifestations of lung injury from blasts. Alveolar damage can cause pneumothorax and pulmonary interstitial emphysema. When air dissects along the bronchovascular sheath, pneumomediastinum, pneumopericardium, and subcutaneous emphysema can occur. Air that enters the pulmonary venous system can result in a systemic arterial air embolism, and possibly, a stroke. Inflammation of the lungs from direct pressure damage to the tissue, cause acute lung injury and possibly, disseminated intravascular coagulation. Clinical signs of lung injury include tachypnea, chest pain, hypoxia, rales, and dyspnea. If there is vascular disruption, hemoptysis can occur. Air leaks from alveolar injury can result in diminished breath sounds, subcutaneous crepitance, increased resonance, and tracheal deviation. Hemodynamic compromise will occur with tracheal deviation.

Alveolar damage, leading to air in the pulmonary venous system, can lead to a systemic arterial air embolism. Air in the coronary arteries can lead to coronary ischemia with ST and/or T waves changes on ECG. Air embolism to cerebral arteries leads to cerebral vascular accidents (strokes) with focal neurological deficits. Other manifestations of systemic air embolism include mottling of the skin, demarcated tongue blanching, and/or air in the retinal vessels (the most common sign of arterial air embolus). Rapid death after initial survival is most often caused by arterial air embolus. Initiation of positive pressure ventilation may trigger this event (Ho and Ling 1999). A lung injury from a blast can also precipitate a vagal reflex resulting in bradycardia and hypotension. It is postulated that this occurs from the stimulation of C fibers in the lungs (Guy et al. 1998).

The gastrointestinal system is the third most common organ system affected by primary blast injury. Physical stress and/or mesenteric infarct leads to weakening of the bowel wall with possible rupture. Hemorrhage can also occur (Paran et al. 1996; Sharpnack et al. 1991). The most common site of injury is the colon. Injury to the bowel can be delayed and occur up to several days after the inciting incident. Solid organs are spared because of their homogeneity and lack of air tissue interface.

Brain injury is becoming increasingly recognized as a result of primary blast. Shearing injuries of the brain occur as a result of wave reverberation in the skull. Hippocampal injury causing cognitive impairment has been shown in animal studies (Cernak 2017; Cernak et al. 2001; Singer et al. 2005). Observations in humans have revealed electroencephalographic abnormalities and attention deficit disorder (Born 2005). Human autopsies have revealed punctate hemorrhages and disintegration of Nissl substance in victims who sustained blast injury without direct head trauma (Guy et al. 1998).

Research involving Yucatan minipigs revealed that the brain sustains neuronal loss in the hippocampus after being subjected to primary blast injury. Brain injury also occurred from the inflammation that ensued post blast (Goodrich et al. 2016).

Novel therapeutic approaches may be on the horizon for treatment of traumatic brain injury, including that caused by primary blast. Intranasal insulin administered to rats subjected to traumatic brain injury resulted in enhanced neuronal glucose uptake and utilization, and subsequently improved motor function and memory. Decreased neuroinflammation and preservation of the hippocampus were also noted (Brabazon et al. 2017). In a different investigation, a neuroprotective nucleotide, guanosine, was administered to rats subjected to traumatic brain injury. The treatment group of rats had better locomotor and cognitive outcomes than did the placebo group. Programmed cell death and inflammation were also attenuated in the treatment group (Gerbatin et al. 2017).

The leading cause of death from blast is from flying objects striking victims (secondary blast injury). Eyes are particularly vulnerable. Injuries resulting from displacement of the victims who strike objects are known as tertiary injuries. Lighter weight children are particularly susceptible to this type of injury.

Burns, toxic exposures, and crush injuries constitute quaternary injuries. Crush injuries commonly occur in explosions with structural collapse. The "crush syndrome" can occur when a trapped limb sustains prolonged compromise to the circulation, leading to rhabdomyolysis. Tissue destruction and inflammatory response then occur. Life-threatening electrolyte abnormalities including hyperkalemia, renal failure, hyperuricemia, metabolic acidosis, acute respiratory distress syndrome, disseminated intravascular coagulation, and shock can result from crush syndrome (Gonzalez 2005).

The crush syndrome is commonly seen in natural disasters that result in a lot of structural collapse. Structural collapse and fires can cause the release of toxic materials such as carbon monoxide and cyanide.

5.9.4 Clinical Management of Blast Injuries

Knowledge of the details of a blast can greatly enhance the ability of nurses and HCPs to care for victims of a blast in a hospital setting. Knowledge of whether a blast occurred in a closed or open space, whether structural collapse occurred, or if a victim was rescued from a collapsed area are details that can alert nurses and HCPs as to what kind of injuries that they may anticipate. If toxic substances are released with a blast, nurses and HCPs can prepare for decontamination techniques

and antidote therapies. It would be advantageous for a hospital to be aware of the number of victims that are arriving for care. A mass casualty incident will stress the resources of the institution. Hospital personnel should take stock of the resources that are available. The number of available ventilators and O-blood are examples of finite resources that should be considered.

Advanced Trauma Life Support (ATLS) principles should be applied to all blast injury victims. ABCD of initial resuscitation is applied. The "D" stands for disability as well as decontamination. Decontamination techniques should be deployed if there is uncertainty about toxic exposure as described elsewhere in this chapter. On completion of ABCD of initial resuscitation a secondary survey is performed, as described by ATLS protocol. Attention should be paid to potential injuries that occur with blast injuries. Ruptured tympanic membranes should alert the nurse or HCP of problems from primary blast injury. Impaled objects should remain in place and removed in the operating room by surgical staff so that bleeding may be controlled. A thoracoscopy tube should be placed with an open three point seal over a wound on the side of the chest with an open pneumothorax. A hemothorax is also treated with a thoracoscopy tube. An autotransfusion setup can be applied to recirculate the blood from the pleural cavity of a hemothorax (Wightman and Gladish 2001) that would help preserve donor blood for other victims.

For severe respiratory distress and/or impending respiratory failure, endotracheal intubation should be performed and positive pressure ventilation should be instituted. Because lung tissue could be weakened from primary blast injury, caution should be exercised because of a high risk of pneumothorax, hemorrhage, or arterial air embolus. Gentle application of positive pressure ventilation should be applied to avoid these complications. If only one lung is injured unilateral lung ventilation can be considered for larger children and adults. This technique is not suitable for babies and small children.

Supplemental oxygen with an FiO_2 of 100% should be administered to patients suspected of having an arterial air embolus. Hyperbaric oxygen therapy could even be considered to help accelerate the removal of air from the arteries. Placement of the patient in the left lateral recumbent position may reduce the likelihood of the air lodging in the coronary arteries.

5.9.5 Postresuscitation Management

Victims of blast injuries should be treated identically to those of other types of trauma after initial resuscitation is completed. If primary blast injury occurred, frequent chest and abdominal X-rays should be performed in consideration of the possibility of lung or gastrointestinal injuries. Limbs with open fractures should be immobilized and covered with sterile dressings. Systemic, broad spectrum antibiotics should be administered to patients with open limb injuries. Eyes that sustained chemical injury should be irrigated with water for an hour. All injured eyes should be covered. Most ruptured tympanic membranes will heal spontaneously. Victims with tympanic membrane injury should be advised to avoid swimming for some time. Topical antibiotics are prescribed if dirt or debris is seen in the ear canal. Oral

prednisone is prescribed for hearing loss. Victims with crush injuries should be treated with large volumes of IV fluids to treat inflammatory shock and possibly rhabdomyolysis. Electrolytes should be monitored carefully as these patients are at risk for hyperkalemia, hyperphosphatemia, hyperuricemia, hypocalcemia, and acidosis. Smoke inhalation, burns, and toxic exposures should be treated according to guidelines of burn, trauma, and toxicology protocols.

5.10 Active Shooter

Mass casualty incidents (i.e. mass shootings, active shooter events, bombings, and other multifatality crimes) often attract extensive media coverage as well as the attention of policy makers. Many agencies and organizations record and publish data on these incidents. The measurement and reporting does vary based on the absence of a common definition. However, it is clearly evident that mass casualty incidents (MCIs) continue to increase in both number and scope (Federal Bureau of Investigation 2017; Office for Victims of Crime, Office of Justice Programs, U.S. Department of Justice 2019).

In the U.S., mass shootings are the most common and most closely tracked. The Congressional Research Service (CRS) defines *mass shootings* as events where more than four people are killed with a firearm "within one event, and in one or more locations in close proximity." Congress uses the term *mass killings* and describes these events as "three or more killings in a single incident." The Federal Bureau of Investigation (FBI) uses the term *active shooter*, which it defines as "an individual actively engaged in killing or attempting to kill people in a populated area." It is important to realize that nongovernmental (NGO) organizations (Mother Jones, USA Today, and the Stanford Mass Shootings in America [MSA] data project) use various combinations of these definitions. The exclusion of *gang-* or *drug-related incidents*, the *accidental discharge* of a firearm, or *family-* and *intimate partner-related shootings* further complicates the definition of mass shooting. Mother Jones, the FBI, CRS, the MSA, and Congress do not include these incidents in their definitions, but USA Today does (Office for Victims of Crime, Office of Justice Programs, U.S. Department of Justice 2019).

The FBI has released investigative reports of active shooter events occurring from 2000 to 2013 (Blair and Schweit 2014) as well as 2014 and 2015 (Schweit 2016). In April 2018, the FBI, in collaboration with the Advanced Law Enforcement Rapid Response Training (ALERRT) Center, published a supplemental report reviewing the active shooter incidents in the U.S. for 2016 and 2017. The report revealed striking differences about active shooter incidents from 2016 and 2017 as compared to 2014 and 2015 (Advanced Law Enforcement Rapid Response Training (ALERRT) Center, Texas State University and Federal Bureau of Investigation, U.S. Department of Justice 2018). The report also compared the characteristics of the active shooters from both time periods (Tables 5.28 and 5.29) (Advanced Law Enforcement Rapid Response Training (ALERRT) Center, Texas State University and Federal Bureau of Investigation, U.S. Department of Justice 2018).

Table 5.28 Active shooter incidents in the United States for 2014 and 2015 versus 2016 and 2017[a]

	2014 and 2015	2016 and 2017
Total number of incidents	40 in 26 states	50 in 21 states
Casualties	231 (excluding shooters): 92 killed and 139 wounded	943 (excluding shooters): 221 killed and 722 wounded
Law enforcement officers killed	4 killed, 10 wounded	13 killed, 20 wounded
Met "mass killing" definition	20	20

[a](Advanced Law Enforcement Rapid Response Training (ALERRT) Center, Texas State University and Federal Bureau of Investigation, U.S. Department of Justice 2018)

Table 5.29 Characteristics of the active shooters 2014 and 2015 versus 2016 and 2017[a]

	2014 and 2015	2016 and 2017
Shooters	42: 39 male; 3 female	50 (all male with 17 being teens)
Body armor	2	3
Shooter committed suicide	16	13
Shooter killed by police	14	11
Shooter stopped by citizen	6	8
Shooter apprehended by police	12	18

[a]Reference: (Advanced Law Enforcement Rapid Response Training (ALERRT) Center, Texas State University and Federal Bureau of Investigation, U.S. Department of Justice 2018)

Geographically, the 50 active shooter incidents in 2016 and 2017 occurred in 21 states: six in Texas, five in California and Florida, four in Ohio, three in Maryland and Washington, two in Colorado, Kansas, Nevada, New Mexico, New York, Pennsylvania, Tennessee, Virginia, and Wisconsin, and one in Arizona, Illinois, Louisiana, Michigan, Missouri, and South Carolina. The 50 incidents resulted in 943 casualties (221 people killed and 722 people wounding, excluding shooters). The highest number of casualties (58 killed and 489 wounded) occurred during the Route 91 Harvest Festival in Las Vegas, Nevada, in 2017. The second highest number of casualties (49 killed and 53 wounded) occurred at Pulse, a nightclub in Orlando, Florida, in 2016. The third highest number of casualties (26 killed and 20 wounded) occurred at the First Baptist Church in Sutherland Springs, Texas, in 2017 (Advanced Law Enforcement Rapid Response Training (ALERRT) Center, Texas State University and Federal Bureau of Investigation, U.S. Department of Justice 2018).

Of note, in the 2000–2013 report,(Blair and Schweit 2014) the FBI identified 11 locations where the public was most at risk during an active shooter incident. These location categories include commercial areas (divided into businesses open to pedestrian traffic, businesses closed to pedestrian traffic, and malls), education environments (divided into schools [prekindergarten through 12th grade] and institutions of higher learning), open spaces, government properties (divided into military and other government properties), residences, houses of worship, and health care facilities. For the April 2018 report of 2016 and 2017 active shooter incidents, the

Table 5.30 Summary of active shooter incident locations identified for 2016 and 2017[a]

Location category	Number of incidents	Killed	Wounded
Commercial area	17	85	98
Open space	14	79 (58 in one incident)	540 (489 in one incident)
Educational	7	5	19
Government property	3	8	12
Residencies	2	4	8
Houses of worship	2	27	27
Health care facility	4	7	8
Other location	1	1	1

[a]Reference: (Advanced Law Enforcement Rapid Response Training (ALERRT) Center, Texas State University and Federal Bureau of Investigation, U.S. Department of Justice 2018)

FBI added a new location category, Other Location, to capture events that occurred in venues not included in the 11 previously identified locations (Advanced Law Enforcement Rapid Response Training (ALERRT) Center, Texas State University and Federal Bureau of Investigation, U.S. Department of Justice 2018). The following is a summary of active shooter incident locations identified for 2016 and 2017 (Table 5.30) (Advanced Law Enforcement Rapid Response Training (ALERRT) Center, Texas State University and Federal Bureau of Investigation, U.S. Department of Justice 2018).

Ranking third of all locations for 2016 and 2017, seven of the 50 incidents occurred in educational environments resulting in five killed and 19 wounded. Two incidents occurred in elementary schools, resulting in two killed (including a first-grade student) and eight wounded (one teacher shot, three students shot, and four wounded from shrapnel). One incident occurred in a junior/senior high school, resulting in none killed and four wounded (two from shrapnel, all students). Four incidents occurred at high schools (one outside a school during prom), resulting in three killed (all students) and seven wounded (all students). Fortunately, no incident occurred at institutions of higher learning during 2016 or 2017 (Advanced Law Enforcement Rapid Response Training (ALERRT) Center, Texas State University and Federal Bureau of Investigation, U.S. Department of Justice 2018). Notably, two of the 50 incidents occurred in houses of worship, resulting in 27 killed and 27 wounded. One of these incidents occurred at the First Baptist Church in Sutherland Springs, Texas, and had the third highest number of casualties (26 killed and 20 wounded) in 2017. The dead included 10 women, 7 men, 8 children (7 girls and 1 boy), and an unborn child (Goldman et al. 2017).

A summary report has also been developed for all 250 active shooter incidents from 2000 to 2017, including incidents per year (Fig. 5.11), casualties per year (Fig. 5.12), and location (Fig. 5.13) categories (Federal Bureau of Investigation 2017; Federal Bureau of Investigation 2018). Overall, there was an increase in number of active shooter incidents and casualties per year. Location categories with number of incidents and statistics of their contribution were provided: areas of

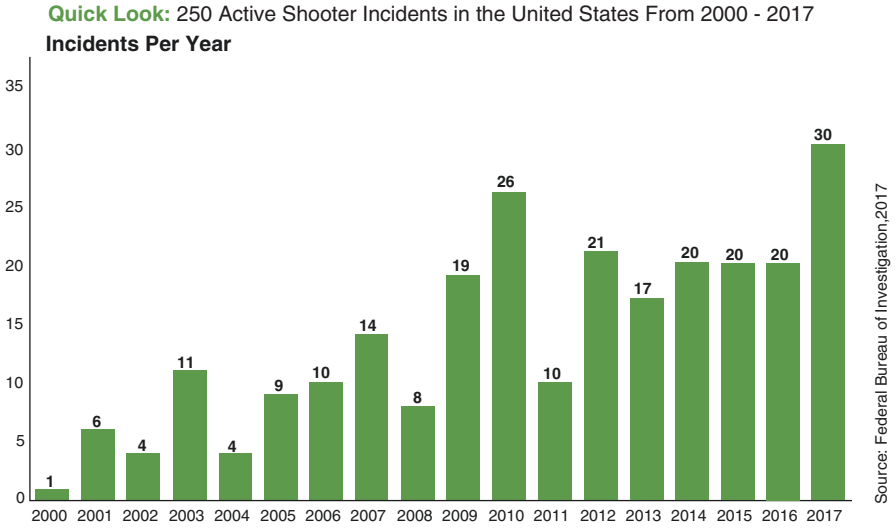


Fig. 5.11 Active shooter incident in United States: Incidents per year

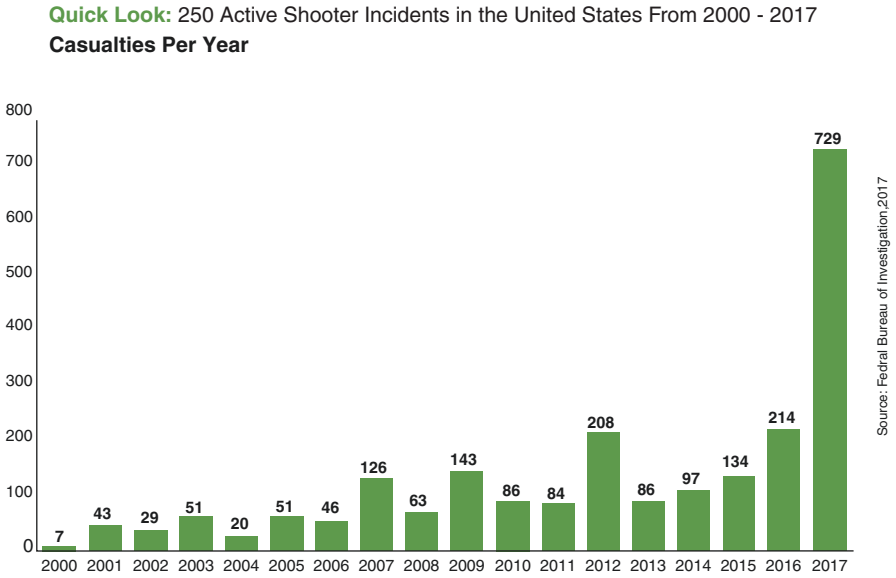


Fig. 5.12 Active shooter incident in United States: Casualties per year

commerce, 105 incidents (42%); educational environments, 52 incidents (21%); government property, 25 incidents (10%); open spaces, 35 incidents (14%); residences, 12 incidents (5%); houses of worship, ten incidents (4%); and health care facilities, ten incidents (4%) (Federal Bureau of Investigation 2017). As noted,

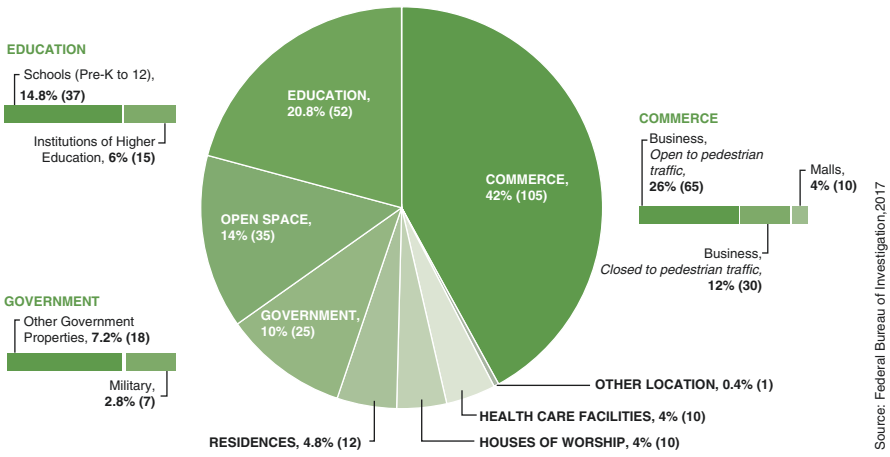


Fig. 5.13 Active shooter incident in United States: Location categories

educational environments account for a large portion of locations for active shooter incidents, ranking only second to commercial areas. Of the 37 incidents (14.8%) occurring at schools, one took place at a nursery (pre-K) school and one incident occurred during a school board meeting that was being hosted on school property but no students were involved (neither perpetrator or victim). The remainder (35 incidents) were perpetrated by or against students, faculty, and/or staff at K-12 schools (Federal Bureau of Investigation 2018). Finally, 15 active shooter incidents (6%) did occur at institutions of higher learning. As a reminder, no incident occurred at institutions of higher learning during 2016 or 2017. Table 5.31 provides a detailed summary of educational environment incidents from 2000 to 2017.

Since the beginning of 2018, other tragic active shooter attacks have occurred in the U.S. and greatly impacted children and adolescents. Two of these such events have occurred in educational environments (United States Secret Service National Threat Assessment Center 2018). On February 14, 2018, a gunman opened fire at Marjory Stoneman Douglas High School. Fourteen students and three staff members were killed while fourteen others were injured (Follman et al. 2019). Twelve victims died inside the building, three died just outside the building on school premises, and two died in the hospital. The shooter was a former student of the school. Another active shooter event occurred on May 18, 2018 at Santa Fe High School in Santa Fe, Texas. The shooter killed ten individuals including eight students and two teachers while injuring 13 others. The shooter was an enrolled student at the school (Follman et al. 2019).

Based on the statistics of active shooter incidents, casualties, and locations, it is vital to prepare schools and plan for such events. National preparedness efforts, including planning, are now informed by the Presidential Policy Directive (PPD) 8 that was signed by the president in March 2011 and describes the nation's approach

to preparedness. This directive represents an evolution in our collective understanding of national preparedness based on the lessons learned from terrorist attacks, hurricanes, school incidents, and other experiences. PPD-8 defines preparedness around five mission areas and can be applied to school active shooter incidents.

Table 5.31 Active shooter incidents involving education environments in the United States from 2000 to 2017[a]

Education site	Details
Santana High School (Education)	On March 5, 2001, at 9:20 a.m., Charles Andrew Williams Jr., 15, armed with a handgun, began shooting in Santana High School in Santee, California. Two people were killed; 13 were wounded. The shooter was apprehended by an off-duty officer who heard gunshots
Granite Hills High School (Education)	On March 22, 2001, at 12:55 p.m., Jason Anthony Hoffman, 18, armed with a shotgun and a handgun, began shooting in Granite Hills High School in El Cajon, California. No one was killed; five were wounded. The shooter was shot by police. He committed suicide in jail 1 week before sentencing
Appalachian School of Law (Education)	On January 16, 2002, at 1:15 p.m., Peter Odighizuma, 43, armed with a handgun, began shooting in the Appalachian School of Law located in Grundy, Virginia. Three people were killed; three were wounded. Three students—two of whom were off-duty police officers—tackled and restrained the shooter until police arrived and took him into custody
Red Lion Junior High School (Education)	On April 24, 2003, at 7:34 a.m., James Sheets, 14, armed with three handguns, shot and killed the school principal in the cafeteria at Red Lion Junior High School in Red Lion, Pennsylvania. Though others were present at the scene, the shooter committed suicide after killing the principal, before police arrived
Case Western Reserve University, Weatherhead School of Management (Education)	On May 9, 2003, at 3:55 p.m., Biswanath A. Halder, 62, armed with a rifle and a handgun, began shooting in the Weatherhead School of Management building at Case Western Reserve University in Cleveland, Ohio. One person was killed; two were wounded. The shooter was wounded during an exchange of gunfire with police
Kanawha County Board of Education (Education)	On July 17, 2003, at 7:00 p.m., Richard Dean Bright, 58, armed with two rifles and two handguns, began shooting during a Kanawha County Board of Education meeting in Charleston, West Virginia. He attempted to light a board member on fire and fired one round at board members before three administrators wrestled the gun away from him. No one was killed; one was wounded
Rocori High School (Education)	On September 24, 2003, at 11:35 a.m., John Jason McLaughlin, 15, armed with a handgun, began shooting in Rocori High School in Cold Spring, Minnesota. A teacher at the school confronted the shooter and ordered him to place his gun on the ground. The shooter complied. Two people were killed; no one was wounded. Police took the shooter into custody
Columbia High School (Education)	On February 9, 2004, at 10:30 a.m., Jon William Romano, 16, armed with a shotgun, began shooting while entering Columbia High School in East Greenbush, New York. No one was killed; one person was wounded. The shooter was restrained by administrators before police arrived and took him into custody

(continued)

Table 5.31 (continued)

Education site	Details
Red Lake High School and Residence (Education)	On March 21, 2005, at 2:49 p.m., Jeffery James Weise, 16, armed with a shotgun and two handguns, began shooting at Red Lake High School in Red Lake, Minnesota. Before the incident at the school, the shooter fatally shot his grandfather, who was a police officer, and another individual at their home. He then took his grandfather's police equipment, including guns and body armor, to the school. A total of nine people were killed, including an unarmed security guard, a teacher, and five students; six students were wounded. The shooter committed suicide during an exchange of gunfire with police
Campbell County Comprehensive High School (Education)	On November 8, 2005, at 2:14 p.m., Kenneth S. Bartley, 14, armed with a handgun, began shooting in Campbell County Comprehensive High School in Jacksboro, Tennessee. Before the shooting, he had been called to the office when administrators received a report that he had a gun. When confronted, he shot and killed an assistant principal and wounded the principal and another assistant principal. The shooter was restrained by students and administrators until police arrived and took him into custody
Pine Middle School (Education)	On March 14, 2006, at 9:00 a.m., James Scott Newman, 14, armed with a handgun, began shooting outside the cafeteria at Pine Middle School in Reno, Nevada. No one was killed; two were wounded. The shooter was restrained by a teacher until police arrived and took him into custody
Essex Elementary School and Two Residences (Education)	On August 24, 2006, at 1:55 p.m., Christopher Williams, 26, armed with a handgun, shot at various locations in Essex, Vermont. He began by fatally shooting his ex-girlfriend's mother at her home and then drove to Essex Elementary School, where his ex-girlfriend was a teacher. He did not find her, but as he searched, he killed one teacher and wounded another. He then fled to a friend's home, where he wounded one person. A total of two people were killed; two were wounded. The shooter also shot himself twice but survived and was apprehended when police arrived at the scene
Orange High School and Residence (Education)	On August 30, 2006, at 1:00 p.m., Alvaro Castillo, 19, armed with two pipe bombs, two rifles, a shotgun, and a smoke grenade, began shooting a rifle from his vehicle at his former high school, Orange High School in Hillsborough, North Carolina. He had fatally shot his father in his home that morning. One person was killed; two were wounded. The shooter was apprehended by police
Weston High School (Education)	On September 29, 2006, at 8:00 a.m., Eric Jordan Hainstock, 15, armed with a handgun and a rifle, began shooting in Weston High School in Cazenovia, Wisconsin. One person was killed; no one was wounded. The shooter was restrained by school employees until police arrived and took him into custody
West Nickel Mines School (Education)	On October 2, 2006, at 10:30 a.m., Charles Carl Roberts, IV, 32, armed with a rifle, a shotgun, and a handgun, began shooting at the West Nickel Mines School in Bart Township, Pennsylvania. After the shooter entered the building, he ordered all males and adults out of the room. After a 20-min standoff, he began firing. The shooter committed suicide as the police began to breach the school through a window. Five people were killed; five were wounded

Table 5.31 (continued)

Education site	Details
Memorial Middle School (Education)	On October 9, 2006, at 7:40 a.m., Thomas White, 13, armed with a rifle and a handgun, began shooting in Memorial Middle School in Joplin, Missouri. His rifle jammed after firing one shot. Hearing the shot, the school principal located the shooter, escorted him from the building, and turned him over to police. No one was killed or wounded
Virginia Polytechnic Institute and State University (Education)	On April 16, 2007, at 7:15 a.m., Seung Hui Cho, 23, armed with two handguns, began shooting in a dormitory at Virginia Polytechnic Institute and State University in Blacksburg, Virginia. Two-and-a-half hours later, he chained the doors shut in a classroom building and began shooting at the students and faculty inside. Thirty-two people were killed; 17 were wounded. In addition, six students were injured jumping from a second-floor classroom and were not included in other reported injury totals. The shooter committed suicide as police entered the building
SuccessTech Academy (Education)	On October 10, 2007, at 1:02 p.m., Asa Halley Coon, 14, armed with two handguns, began shooting in SuccessTech Academy in Cleveland, Ohio. No one was killed; four were wounded. The shooter committed suicide before police arrived
Louisiana Technical College (Education)	On February 8, 2008, at 8:35 a.m., Latina Williams (female), 23, armed with a handgun, began shooting in a second-floor classroom at Louisiana Technical College in Baton Rouge, Louisiana. She fired six rounds, then reloaded and committed suicide before police arrived. Two people were killed; no one was wounded
Cole Hall Auditorium, Northern Illinois University (Education)	On February 14, 2008, at 3:00 p.m., Steven Phillip Kazmierczak, 27, armed with a shotgun and three handguns, began shooting in the Cole Hall Auditorium at Northern Illinois University in DeKalb, Illinois. He had attended graduate school at the university. Five people were killed; 16 were wounded, including three who were injured as they fled. The shooter committed suicide before police arrived
Harkness Hall at Hampton University (Education)	On April 26, 2009, at 12:57 a.m., Odane Greg Maye, 18, armed with three handguns, began shooting in Harkness Hall, a residence hall at Hampton University in Hampton, Virginia, and then shot himself before police arrived. The shooter had briefly attended the university. A dormitory manager pulled the fire alarm when the shooting began, emptying the building. No one was killed; two were wounded. He was apprehended by police
Larose-Cut Off Middle School (Education)	On May 18, 2009, at 9:00 a.m., Justin Doucet, 15, armed with a handgun, fired once at a teacher at Larose-Cut Off Middle School in Cut Off, Louisiana, then went to the bathroom and shot himself. He died a week later. No one was killed or wounded
Inskip Elementary School (Education)	On February 10, 2010, at 12:49 p.m., Mark Stephen Foster, 48, armed with a handgun, began shooting inside Inskip Elementary School in Knoxville, Tennessee. He had just been informed that his teaching contract would not be renewed. The shooting occurred after he left the office and returned with a gun. No one was killed; two members of the administration were wounded. The shooter was apprehended by responding police

(continued)

Table 5.31 (continued)

Education site	Details
Shelby Center, University of Alabama (Education)	On February 12, 2010, at 4:00 p.m., Amy Bishop Anderson (female), 44, armed with a handgun, began shooting during a biology department meeting in the Shelby Center at the University of Alabama in Huntsville, Alabama. She sat in the meeting for 30 min, then stood up, and began firing. Three people were killed; three were wounded. The shooter surrendered to responding police
Deer Creek Middle School (Education)	On February 23, 2010, at 3:10 p.m., Bruco Strongeagle Eastwood, 32, armed with a rifle, began shooting in Deer Creek Middle School in Littleton, Colorado. No one was killed; two people were wounded. The shooter was restrained by teachers until police arrived and took him into custody
The Ohio State University, Maintenance Building (Education)	On March 9, 2010, at 3:30 a.m., Nathaniel Alvin Brown, 50, armed with two handguns, began shooting in the maintenance building at The Ohio State University in Columbus, Ohio. He had just been fired for allegedly lying on his job application. One person was killed; one was wounded. The shooter committed suicide before police arrived
Kelly Elementary School (Education)	On October 8, 2010, at 12:10 p.m., Brendan O'Rourke, aka Brandon O'Rourke, 41, armed with a handgun, began shooting at Kelly Elementary School in Carlsbad, California, after having jumped the school fence. No one was killed; two students were wounded. The shooter was tackled and restrained by nearby construction workers until police arrived and took him into custody
Panama City School Board Meeting (Education)	On December 14, 2010, at 2:14 p.m., Clay Allen Duke, 56, armed with a handgun, began shooting during a school board meeting in the Nelson Administrative Building in Panama City, Florida. The shooter's wife had previously been employed by the school district. After allowing several people to leave the room, the shooter fired in the direction of board members. No one was killed or wounded. The shooter committed suicide during an exchange of gunfire with the school district's armed security
Millard South High School (Education)	On January 5, 2011, at 12:44 p.m., Richard L. Butler Jr., 17, armed with a handgun, began shooting in Millard South High School in Omaha, Nebraska. Earlier that day, the assistant principal had suspended the shooter for allegedly driving his car onto the football field. The assistant principal was killed; the principal was wounded. The shooter committed suicide after fleeing the site of the shooting.
Chardon High School (Education)	On February 27, 2012, at 7:30 a.m., Thomas Michael Lane, III, 17, armed with a handgun, began shooting in the cafeteria at Chardon High School in Chardon, Ohio. The shooter was chased out of the building by a school coach. Three people were killed; three were wounded. The shooter was apprehended by police near the school
University of Pittsburgh Medical Center, Western Psychiatric Institute and Clinic (Education)	On March 8, 2012, at 1:40 p.m., John Schick, 30, armed with two handguns, began shooting inside the lobby of the Western Psychiatric Institute and Clinic at the University of Pittsburgh Medical Center in Pittsburgh, Pennsylvania. One person was killed; seven were wounded, including one police officer. The shooter was killed by University of Pittsburgh police
Oikos University (Education)	On April 2, 2012, at 10:30 a.m., Su Nam Ko, aka One L. Goh, 43, armed with a handgun, began shooting inside Oikos University in Oakland, California. He then killed a woman to steal her car. Seven people were killed; three were wounded. The shooter was arrested by police later that day

Table 5.31 (continued)

Education site	Details
Perry Hall High School (Education)	On August 27, 2012, at 10:45 a.m., Robert Wayne Gladden Jr., 15, armed with a shotgun, shot a classmate in the cafeteria of Perry Hall High School in Baltimore, Maryland. The shooter had an altercation with another student before the shooting began. He left the cafeteria and returned with a gun. No one was killed; one person was wounded. The shooter was restrained by a guidance counselor before being taken into custody by the school's resource officer
Sandy Hook Elementary School and Residence (Education)	On December 14, 2012, at 9:30 a.m., Adam Lanza, 20, armed with two handguns and a rifle, shot through the secured front door to enter Sandy Hook Elementary School in Newtown, Connecticut. He killed 20 students and six adults, and wounded two adults inside the school. Prior to the shooting, the shooter killed his mother at their home. In total, 27 people were killed; two were wounded. The shooter committed suicide after police arrived
Taft Union High School (Education)	On January 10, 2013, at 8:59 a.m., Bryan Oliver, 16, armed with a shotgun, allegedly began shooting in a science class at Taft Union High School in Taft, California. No one was killed; two people were wounded. An administrator persuaded the shooter to put the gun down before police arrived and took him into custody
New River Community College, Satellite Campus (Education)	On April 12, 2013, at 1:55 p.m., Neil Allen MacInnis, 22, armed with a shotgun, began shooting in the New River Community College satellite campus in the New River Valley Mall in Christiansburg, Virginia. No one was killed; two were wounded. The shooter was apprehended by police after being detained by an off-duty mall security officer as he attempted to flee
Santa Monica College and Residence (Education)	On June 7, 2013, at 11:52 a.m., John Zawahri, 23, armed with a handgun, fatally shot his father and brother in their home in Santa Monica, California. He then carjacked a vehicle and forced the driver to take him to the Santa Monica College campus. He allowed the driver to leave her vehicle unharmed but continued shooting until he was killed in an exchange of gunfire with police. Five people were killed; four were wounded
Sparks Middle School (Education)	On October 21, 2013, at 7:16 a.m., Jose Reyes, 12, armed with a handgun, began shooting outside Sparks Middle School in Sparks, Nevada. A teacher was killed when he confronted the shooter; two people were wounded. The shooter committed suicide before police arrived
Arapahoe High School (Education)	On December 13, 2013, at 12:30 p.m., Karl Halverson Pierson, 18, armed with a shotgun, machete, and three Molotov cocktails, began shooting in the hallways of Arapahoe High School in Centennial, Colorado. As he moved through the school and into the library, he fired one additional round and lit a Molotov cocktail, throwing it into a bookcase and causing minor damage. One person was killed; no one was wounded. The shooter committed suicide as a school resource officer approached him
Berrendo Middle School (Education)	On January 14, 2014, at 7:30 a.m., Mason Andrew Campbell, 12, armed with a shotgun, began shooting in Berrendo Middle School in Roswell, New Mexico. A teacher at the school confronted and ordered him to place his gun on the ground. The shooter complied. No one was killed; 3 were wounded: 2 students and an unarmed security guard. The shooter was taken into custody

(continued)

Table 5.31 (continued)

Education site	Details
Seattle Pacific University (Education)	On June 5, 2014, at 3:25 p.m., Aaron Rey Ybarra, 26, armed with a shotgun, allegedly began shooting in Otto Miller Hall at Seattle Pacific University in Seattle, Washington. He was confronted and pepper sprayed by a student as he was reloading. One person was killed; 3 were wounded. Students restrained the shooter until law enforcement arrived
Reynolds High School (Education)	On June 10, 2014, at 8:05 a.m., Jared Michael Padgett, 15, armed with a handgun and a rifle, began shooting inside the boy's locker room at Reynolds High School in Portland, Oregon. One student was killed; 1 teacher was wounded. The shooter committed suicide in a bathroom stall after law enforcement arrived
Marysville-Pilchuck High School (Education)	On October 24, 2014, at 10:39 a.m., Jaylen Ray Fryberg, 15, armed with a handgun, began shooting in the cafeteria of Marysville-Pilchuck High School in Marysville, Washington. Four students were killed, including the shooter's cousin; 3 students were wounded, including one who injured himself while fleeing the scene. The shooter, when confronted by a teacher, committed suicide before law enforcement arrived
Florida State University (Education)	On November 20, 2014, at 12:00 a.m., Myron May, 31, armed with a handgun, began shooting in Strozier Library at Florida State University in Tallahassee, Florida. He was an alumnus of the university. No one was killed; 3 were wounded. The shooter was killed during an exchange of gunfire with campus law enforcement.
Umpqua Community College (Education)	On October 1, 2015, at 10:38 a.m., Christopher Sean Harper-Mercer, 26, armed with several handguns and a rifle, began shooting classmates in a classroom on the campus of Umpqua Community College in Roseburg, Oregon. Nine people were killed; 7 were wounded. The shooter committed suicide after being wounded during an exchange of gunfire with law enforcement.
Madison Junior/ Senior High School (Education)	On February 29, 2016, at 11:30 a.m., James Austin Hancock, 14, armed with a handgun, allegedly began shooting in the cafeteria of Madison Junior/Senior High School in Middletown, Ohio. He shot two students before fleeing the building. No one was killed; four students were wounded (two from shrapnel). The shooter was apprehended near the school by law enforcement officers
Antigo High School (Education)	On April 23, 2016, at 11:02 p.m., Jakob Edward Wagner, 18, armed with a rifle, began shooting outside a prom being held at his former school, Antigo High School in Antigo, Wisconsin. Two law enforcement officers, who were on the premises, heard the shots and responded immediately. No one was killed; two students were wounded. The shooter was wounded in an exchange of gunfire with law enforcement officers and later died at the hospital
Townville Elementary School (Education)	On September 28, 2016, at 1:45 p.m., Jesse Dewitt Osborne, 14, armed with a handgun, allegedly began shooting at the Townville Elementary School playground in Townville, South Carolina. Prior to the shooting, the shooter, a former student, killed his father at their home. Two people were killed, including one student; three were wounded, one teacher and two students. A volunteer firefighter, who possessed a valid firearms permit, restrained the shooter until law enforcement officers arrived and apprehended him

Table 5.31 (continued)

Education site	Details
West Liberty-Salem High School (Education)	On January 20, 2017, at 7:36 a.m., Ely Ray Serna, 17, armed with a shotgun, allegedly began shooting inside West Liberty Salem High School, in West Liberty, Ohio, where he was a student. After assembling the weapon in a bathroom, the shooter shot a student who entered, then shot at a teacher who heard the commotion. The shooter shot classroom door windows before returning to the bathroom and surrendering to school administrators. No one was killed; two students were wounded. School staff members subdued the shooter until law enforcement arrived and took the shooter into custody
Freeman High School (Education)	On September 13, 2017, at 10:00 a.m., Caleb Sharpe, 15, armed with a rifle and a pistol, allegedly began shooting at Freeman High School in Rockford, Washington, where he was a student. One student was killed; three students were wounded. A school employee confronted the shooter, ordered him to the ground, and held him there until law enforcement arrived and took him into custody
Rancho Tehama Elementary School and Multiple Locations in Tehama County, California (Education)	On November 14, 2017, at 7:53 a.m., Kevin Janson Neal, 44, armed with a rifle and two handguns, began shooting at his neighbors, the first in a series of shootings occurring in Rancho Tehama Reserve, Tehama County, California. After killing three neighbors, he stole a car and began firing randomly at vehicles and pedestrians as he drove around the community. After deliberately bumping into another car, the shooter fired into the car and wounded the driver and three passengers. The shooter then drove into the gate of a nearby elementary school. He was prevented from entering the school due to a lockdown, so he fired at the windows and doors of the building, wounding five children. Upon fleeing the school, the shooter continued to shoot at people as he drove around Rancho Tehama Reserve. Law enforcement pursued the shooter; they rammed his vehicle, forced him off the road, and exchanged gunfire. The shooter's wife's body was later discovered at the shooter's home; the shooter apparently had shot and killed her the previous day. In total, five people were killed; 14 were wounded, eight from gunshot injuries (including one student) and six from shrapnel injuries (including four students). The shooter committed suicide after being shot and wounded by law enforcement during the pursuit
Aztec High School (Education)	On December 7, 2017, at approximately 8:00 a.m., William Edward Atchison, 21, armed with a handgun, began shooting inside Aztec High School in Aztec, New Mexico. The shooter was a former student. Two students were killed; no one was wounded. The shooter committed suicide at the scene, before police arrived

[a]In A Study of Active Shooter Incidents in the United States Between 2000 and 2013, the FBI identified 11 locations where the public was most at risk during an incident. These location categories include commercial areas (divided into business open to pedestrian traffic, businesses closed to pedestrian traffic, and malls), education environments (divided into schools [prekindergarten through 12th grade] and institutions of higher learning), open spaces, government properties (divided into military and other government properties), residences, houses of worship, and health care facilities. In 2018, the FBI added a new location category, Other Location, to capture incidents that occurred in venues not included in the 11 previously identified locations (Federal Bureau of Investigation 2017). This table only includes educational environments. An entire list of all incidents from 2000 to 2017 at all locations can be found at https://www.fbi.gov/file-repository/active-shooter-incidents-2000-2017.pdf/view (Federal Bureau of Investigation 2018)

Prevention means the capabilities necessary to avoid, deter, or stop an imminent crime or threatened/actual mass casualty incident. Prevention is the action schools take to prevent a threatened or actual incident from occurring. *Protection* means the capabilities to secure schools against acts of violence and man-made or natural disasters. Protection focuses on ongoing actions that protect students, teachers, staff, visitors, districts, networks, and property from a threat or hazard. *Mitigation* means the capabilities necessary to eliminate or reduce the loss of life and property damage by lessening the impact of an event or emergency at the school. It also means reducing the likelihood that threats and hazards will happen. *Response* means the school's or school district's capabilities necessary to stabilize an emergency once it has already happened or is certain to happen in an unpreventable way, establish a safe and secure environment, save lives and property, and facilitate the transition to recovery. *Recovery* means the capabilities necessary to assist schools affected by an event or emergency in restoring the learning environment. It also means teaming with community partners to restore educational programming, the physical environment, business operations, and social, emotional, and behavioral health. The majority of Prevention, Protection, and Mitigation activities generally occur before an incident, although these three mission areas do have ongoing activities that can occur throughout an active shooter incident. Response activities occur during an incident, and recovery activities can begin during an incident and occur after an incident (United States Department of Education, Office of Elementary and Secondary Education, Office of Safe and Healthy Students 2013; United States Department of Homeland Security 2018b; United States Department of Homeland Security 2018).

In the *K-12 School Security* guide, the U.S. Department of Homeland Security (DHS) focuses on prevention and protection since the activities and measures associated with them occur prior to an incident (2018). Effective preventative and protective actions decrease the probability that schools (or other facilities) will encounter incidents of gun violence or should an incident occur, it reduces the impact of that incident. The guide emphasizes that the level of security at a facility will be based on hazards relevant to the facility, people, or groups associated with it. It also warns that as new or different threats become apparent, the perception of the relative security changes and insecurity should drive change to reflect the level of confidence of the people of groups associated with the facility. The DHS utilizes a Hometown Security approach that emphasizes the process of Connect, Plan, Train, and Report (CPTR) with the objective to realize effective, collaborative outcomes (United States Department of Homeland Security 2018b).

The initial phase is *Connect* and occurs by a school or district reaching out and developing relationships in the community, including local law enforcement. Having these relationships before an incident or event can help speed up the response when something happens. Each school must begin with identification or development of a security team, group, or organization. This phase also emphasizes outreach, collaboration, and building of a coalition. There should be coalition members from within a school and may include district/school administrators, teachers, aides, facility operations personnel, human resources, administrative, counseling, and

student groups. External groups directly related to the school might include boards of education, parent organizations, mental health groups/agencies, and teacher and bus driver unions. External groups indirectly related to the school include all responder organizations such as police and fire departments, sheriff's office, emergency medical services, emergency management, and the local DHS Protective Security Advisor (PSA). Other tangential groups such as volunteer organizations, utility providers, and facilities in close geographic proximity should also be considered. Core and advisory members of the coalition are established. A coalition champion is also identified and is the person who owns the majority of the responsibility for achieving a school's security goals. The champion organizes the coalition as it grows and matures (United States Department of Homeland Security 2018b). The next phase is *Plan*. This will bring the coalition together. *The Guide for Developing High Quality School Emergency Operations Plans* (United States Department of Education, Office of Elementary and Secondary Education, Office of Safe and Healthy Students 2013) is an excellent resource for the coalition. A *School Security Survey for Gun Violence* can be completed and the coalition or user can quickly and effectively determine a facility's security proficiency (United States Department of Homeland Security. 2018). Specific portions of or topics within a school plan should be assigned to individuals, committees, or working groups most qualified to address them. The planning process must be sustainable. The amount of time spent in the planning phase should be commensurate with the amount of effort expended on the other phases (United States Department of Homeland Security 2018b). The next phase of the process is to *Train* on the plan developed by the coalition. Determining who is responsible for what and how it should be done is the basic function of planning. In fact, telling various members of the team what is expected of them and when to do that activity is the function of training. It is vital to utilize the curricula development expertise possessed by the K-12 community. School administrators should take advantage of this skill set and find creative ways to address difficult topics, such as gun violence. It should be carried out in an effective and nontraumatic way. Presenting the training in pieces or steps allows for a more comprehensive learning experience. It is important to validate training through exercises and drills, all of which should include the students. The training event should be followed by the completion and implementation of an after-action improvement plan with adjustment of the CPTR as indicated (United States Department of Homeland Security 2018b). The final phase in the process is *Report*. The reporting phase is arguably the most important of all the phases. Reporting principles underlie the other three phases and have profound prevention and protection impacts by driving forward information. DHS models the reporting phase using the "If You See Something, Say Something®" campaign (U.S. DHS, 2018) and the Nationwide Suspicious Activity Reporting (SAR) Initiative (Nationwide Suspicious Activity Reporting Initiative (NSI) 2019). "If You See Something, Say Something®" focuses on empowering anyone who sees suspicious activity to do something about it by contacting local law enforcement, or if an emergency to call 9–1–1 (United States Department of Homeland Security 2018a). This is a compelling capability when well organized and managed. A good plan for reporting, especially for a K-12

school, involves training staff and students on what is considered suspicious. There are many methods in which schools can employ to facilitate this, such as dedicated telephone numbers, websites for anonymous reporting, email or text messaging, and mobile phone applications. Conducting simple drills for reporters and receivers keeps skills sharp and reinforces the importance of the effort with the goal to save lives. If the plan includes sharing all suspicious activity calls with the local fusion center then the probability of higher fidelity reporting increases (United States Department of Homeland Security 2018b).

When making changes to a school's plans, procedures, and protective measures, it is imperative the needs of individuals with special health care needs be addressed throughout the process. Planning, training, and execution should always consider accessible alert systems for those who are deaf or hard of hearing; students, faculty, and staff who have visual impairments or are blind; individuals with limited mobility; alternative notification measures; people with temporary disabilities; visitors; people with limited English proficiency; sign cards with text- and picture-based emergency messages/symbols; and involving people with disabilities in all planning (United States Department of Homeland Security, Interagency Security Committee 2015).

It is important to understand that no "profile" exists for an active shooter (United States Department of Education, Office of Elementary and Secondary Education, Office of Safe and Healthy Students 2013). However, research indicates there may be signs or indicators. O'Toole (2000) presents an in depth, systematic procedure for school shooter threat assessment and intervention. The model was designed to be used by educators, mental health professionals, and law enforcement agencies. Its fundamental building blocks are the threat assessment standards, which provide a framework for evaluating a spoken, written, and symbolic threat, and the four-pronged assessment approach which provides a logical, methodical process to examine the threatener and assess the risk that the threat will be carried out. Schools should learn the signs of a potentially volatile situation that may develop into an active shooter situation and proactively seek ways to prevent an incident with internal resources, or additional external assistance (United States Department of Education, Office of Elementary and Secondary Education, Office of Safe and Healthy Students 2013). Potential warning signs of a school shooter may include increasingly erratic, unsafe, or aggressive behaviors; hostile feelings of injustice or perceived wrongdoing; drug and alcohol abuse; marginalization or distancing from friends and colleagues; changes in performance at work or school; sudden and dramatic changes in home life or in personality; pending civil or criminal litigation; and observable grievances with threats and plans of retribution (United States Department of Homeland Security 2018b). At a minimum, schools should establish and enforce policies that prohibit, limit, or determine unacceptable behaviors and consequences of weapons possession/use, drug possession/use, alcohol/tobacco possession/use, bullying/harassment, hazing, cyber-bullying/harassment/stalking, sexual assault/misconduct/harassment, bias crimes, social media abuse, and any criminal acts (United States Department of Homeland Security 2018b).

In addition to policies and positive school climates, school districts and administrators should establish dedicated teams to evaluate threats, such as a Threat Assessment Team (TAT). The Team should include mental health professionals

(e.g., forensic psychologist, clinical psychologist, and school psychologist) to contribute to the threat assessment process (United States Department of Homeland Security 2018b). It is the responsibility of the TAT to investigate and analyze communications and behaviors to make a determination on whether or not an individual poses a threat to him/herself or others (United States Department of Education, Office of Elementary and Secondary Education, Office of Safe and Healthy Students 2013). As well as TATs, some schools have even opted to establish social media monitoring teams which look for keywords that may indicate bullying or other concerning statements. If a school opts to create such a team, it should work very closely with the TAT to ensure that applicable privacy, civil rights and civil liberties, other federal, state and local laws, and information sharing protocols are followed. Please refer to Chap. 14 for further information.

After an active shooter incident, field triage (e.g., JumpSTART) must commence and the patient must be evaluated by an experienced emergency medicine or trauma surgeon, preferably by a pediatric specialist in those disciplines. If an active shooter incident is coupled with detonation of an explosive device, the child must be screened and decontaminated for radiation exposure ("dirty bomb"). Triage tags are extremely helpful when multiple victims present in a short period of time. Medical response to an active shooter event will focus on control of external hemorrhage along with circulatory stabilization. Operative emergencies will be common and receive the highest priority. Severe extremity injuries may be controlled with tourniquet application or other forms of hemorrhage control. Re-evaluation is paramount to prevent ischemia to distal regions. However, thoracic or abdominal (truncal) injuries will need immediate surgical exploration and intervention. Penetrating trauma will cause more vascular injuries than blunt trauma, and vascular surgical trays may be in short supply at a hospital. Major procedure or surgical trays may become short in supply based on the increased operative demand. Resuscitative blood transfusion therapy may utilize a massive blood transfusion protocol. Since whole blood may be short in supply, some will simply use the 1:1:1 rule (administer one unit of packed cells: one unit of fresh frozen plasma: one unit of platelets). A unit for children may be substituted as an aliquot based on size of the patient (e.g., administer 10 mL/kg of packed cells: 10 mL/kg of fresh frozen plasma: 10 mL/kg of platelets). Calcium must also be replaced when there is a large volume transfusion. Due to extensive blood product utilization, there may be a heavy impact on institutional or regional blood supplies. Plans should be in place to address these problems, including the implementation of allocation of scarce resources. Mental health support and staff debriefs are essential and should be included after an active shooter event (Hick et al. 2016).

5.11 Conclusion

In conclusion, all forms of disasters, whether man-made or natural, impact infants, children, and adolescents throughout the world. Effective and efficient interventions remain the cornerstone of sustaining a child's well-being while reducing untoward complications due to all forms of disasters. Having a deep understanding of

pediatric physiology and pathophysiology is crucial to all levels of disaster diagnostics and therapeutics. All nurses and HCPs have an obligation to understand these principles and deliver excellent, compassionate care to the pediatric disaster victim.

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