Effects of Terrorist Attacks on the Elderly—Part I: Medical and Psychiatric Complications of Bombings and Biological, Chemical, and Nuclear Attacks

Ryan C.W. Hall, MD, Richard C.W. Hall, MD, and Marcia Chapman

This is the first article of a two-part series dealing with the effects of terrorism on the elderly. Part I addresses specific data as it relates to the medical impact of biological, chemical, nuclear, and bombing attacks on senior citizens. Part II will review the literature as it relates to the occurrence of psychiatric disorders in the elderly following terrorist attacks. The authors will attempt to identify and review data specific to senior citizens, and to provide the clinician with useful information regarding differential diagnosis, the nature of symptoms likely to be encountered, time course, and unusual presentations seen in the elderly population.

The specific health issues that arise from terrorism and its effect on the elderly have rarely been directly studied. The available literature suggests that the elderly experience a higher physical morbidity and a more complicated hospital course post-injury when affected by terrorism. They are at increased risk due to many of the physiological changes associated with aging, the presence of comorbidities, decreased mobility, and a lack of physiological reserve. Psychiatrically, the elderly face different challenges than the general population due to their past experiences, a potential for reduced independence, and restricted access to services.[1]

Dr. Ryan Hall is in the Department of Psychiatry and Behavioral Sciences, Johns Hopkins Hospital, Baltimore, MD; Dr. Richard Hall is Courtesy Clinical Professor of Psychiatry, University of Florida; and Ms. Chapman is Research Assistant to Dr. Richard Hall.

BIOTERRORISM

Bioterrorism is the use of viruses, bacteria, fungi, or biotoxins to intentionally harm or terrorize a population in an attempt to effect political change. A 1999 report from the Monterey Institute of International Studies documented 121 biocrimes between 1960 and 1995, with a notable increase in 1995.[2] These events ranged from the 1984 use of *Salmonella* (sprayed at salad bars) in Oregon by the Rajneeshee cult in an attempt to win a local election, to the 1991 attempts by the Minnesota Patriots, a tax-resistance group, to use ricin, to the unsuccessful use of botulinum toxin, anthrax, and Q fever by the Japanese cult Aum Shinrikyo in the 1990s.[2-6] In 2001, there were 629 bioterrorism incidents reported, 603 of which were later identified as hoaxes.[7] Many of the hoaxes occurred at abortion clinics, newspaper/television stations, and governmental offices in the wake of the 2001 anthrax attacks directed toward congressional offices and news agencies.[7]

TABLE I

Categories of Bioweapons and Agents

Category A
- Easily disseminated
- Highly contagious
- High mortality
- High potential for infrastructure disruption
- Requires special public health preparedness

Examples
Bacillus anthracis (anthrax)
Variola major (smallpox)
Yersinia pestis (plague)
Francisella tularensis (tularemia)
Hemorrhagic fever viruses (filoviruses, arenaviruses)
- Ebola
- Marburg
- Lassa
- Junin
- Machupo

Clostridium botulinum (botulinum toxin)

Category B
- Moderately easy to disseminate
- Potential for contagion
- Moderate morbidity
- Low mortality
- Enhanced disease surveillance

Examples
Coxiella burnetii (Q fever)
Brucella (brucellosis)
Burkholderia mallei (glanders)
Burkholderia pseudomallei (melioidosis)
Alphaviridae (encephalitis)
- Venezuelan equine
- Eastern equine
- Western equine

Rickettsia prowazekii (typhus fever)
Toxins
- Ricin
- Staphylococcal enterotoxin B
- Conotoxin

Food safety threats
- *Salmonella*
- *Escherichia coli* O157:H7
- *Shigella*

Water safety threats
- *Vibrio cholerae*
- *Cryptosporidium parvum*

Category C
- Emerging pathogens
- Potential high mortality
- Potential high morbidity
- Could be engineered for mass dissemination

Examples
Hantavirus
- Hemorrhagic fever with renal syndrome (HFRS)
- Hantavirus pulmonary syndrome (HPS)

Tickborne hemorrhagic viruses
- Crimean-Congo hemorrhagic fever
- South American hemorrhagic fever
- Rift Valley fever

Nipah virus encephalitis
Yellow fever
Multidrug-resistant tuberculosis
Emerging threat agents

These bioterrorist attacks remain a great public health threat due to their potential massive scope, the difficulty of detecting an attack in a timely fashion, differentiating these agents from "normal" disease presentations, and the specific identification of the agents. Unlike bombings, where the source of the trauma is known, it may take days before the subject population realizes that a biological attack has occurred. The fact that multiple agents may be used simultaneously further confounds the clinical picture and may delay appropriate treatment. For some agents, such as hemorrhagic smallpox, there is no effective treatment aside from comfort/supportive care. Bioterrorist attacks threaten not only the general population and healthcare providers, but also the nation's physical infrastructure, as was evidenced by the disruption of the United States Postal Service when anthrax was sent through the mail, shutting down distribution centers in several states and closing the Senate office buildings for decontamination.

There are currently 35 organisms identified by the Centers for Disease Control and Prevention (CDC) as potential bioterrorism agents[2,6,8,9](Table I). They are categorized into three groups based on method of distribution, degree of lethality, and potential for disruption of public life and national security.[2,6,8,9] Unfortunately, it is beyond the scope of this article to review each of these agents as they specifically concern the elderly. Some general facts, however, are helpful. When exposed to infectious

bioweapons, the elderly are the population at greatest risk for becoming infected after exposure, have the highest mortality if infected, have the greatest severity of disease occurrence, and have the longest hospitalizations if they survive. Their reduced resistance and survival is due to less efficient immune responses, reduced physiologic reserve, and higher percentage of comorbid disease states.[10-12] Data from the 2001 anthrax attacks and from naturally occurring plague cases illustrate these findings.

Anthrax

In 2001, the United States sustained a biological attack when letters containing anthrax spores were mailed to congressional offices in Washington, DC, and news organizations in Florida and New York. These mailings resulted in pulmonary anthrax being reported in Florida, New York, Washington, DC, Connecticut, and New Jersey.[10,11,13] The mean age of the 11 patients who contracted anthrax was age 60.2 years (range, 43-94 years).[11,13] The incubation period was approximately 4 days (range, 4-6 days) with presenting symptoms of fever, chills, sweats, fatigue, malaise, minimal or nonproductive cough, dyspnea, nausea, vomiting, and x-ray changes (eg, infiltrates, pleural effusion, mediastinal widening).[13] Several of the cases were initially misdiagnosed as viral respiratory disease. These patients were not started on antibiotics in a timely fashion.[11,13] The five people who died had eventually received appropriate antibiotics, but only after they had entered the fulminant phase (respiratory distress, cyanosis, and diaphoresis) of the disease.[11,13] The infection continued to progress even after they were treated with massive doses of antibiotics and had demonstrated negative repeat blood cultures.[10,11,13] The autopsy findings included hemorrhagic mediastinal lymphadenitis and disseminated *Bacillus anthracis* infection. The mortality rate with treatment was 45%, which was lower than previously reported mortality rates for pulmonary anthrax of higher than 85%. The decrease in mortality rate was attributed to the rapid use of fluoroquinolones, rifampin, and other antibiotics in combination, as well as to a high suspicion for anthrax after the initial deaths.[13]

The 11th case, which was not initially identified, was that of a 94-year-old Connecticut woman who was believed to have contracted inhalation anthrax from the cross-contamination of her mail.[4,10,11] She presented with symptoms of fever, fatigue, malaise, dry cough, and shortness of breath.[10,11] She was initially misdiagnosed as having a viral upper respiratory syndrome with secondary dehydration, and was not started on antibiotics until blood cultures returned positive for anthrax approximately 12 hours after her hospital admission.[11] Although the exact time of her exposure is unclear, based on CDC follow-up it was felt that she had a longer incubation period and a lower exposure dose than was previously felt sufficient for infection.[10,11] The mechanisms postulated for why she developed pulmonary anthrax at a lower inoculum dose than previously reported included her advanced age leading to an incomplete immune response, a history of obstructive lung disease, and her use of bronchodilators.[10,11] Another interesting finding in this case and seven others was an initial white blood cell (WBC) count of less than 10,000 (normal for most lab results) with a shift to the left.[11,13] In the three cases in which the WBC count was greater than 10,000, the patients were either younger than age 50 or later died, possibly indicating that they presented with symptoms later in the course of the disease.[13]

Plague

Yersinia pestis (plague), which is naturally occurring in the United States and has the potential to be

weaponized (used successfully by Japan during World War II) presents differently in elderly patients.[2] People over the age of 40 years present with septicemic plague at a higher frequency than those under age 40, who are more likely to present with bubonic plague.[6,14] The septicemic form of plague occurs in about 13% of naturally occurring cases, with a corresponding mortality of 22%.[2] Septicemic plague is often misdiagnosed, resulting in delayed treatment, because the classic bubo is not present. Patients with septicemic plague usually present sooner after exposure than those with bubonic plague and usually complain of severe abdominal pain. They appear toxic and have a higher mortality rate.[14] Patients with abdominal symptoms who experience delayed diagnosis and treatment have a mortality of 30-50%, while those who do not receive any antibiotics have a mortality rate approaching 100%.[2]

BOMBING INJURIES

Bombing is the world's most frequently encountered method of terrorist attack.[15] From an Israeli study of 666 civilian fatalities related to terrorism, which occurred from September 2000 to March 2003, 402 deaths were reported as the result of suicide bombings, with the next closest cause for fatality being shootings at 92 deaths.[16] The elderly, as a group, are more isolated from this form of terrorism than the rest of the civilian population due to the locations typically chosen for these bombings, which are high-traffic areas during busy times. Bombing targets are chosen to disrupt the normal flow of life (eg, public transportation, restaurants, nightclubs, wedding halls).[17] Due to most geriatric patients being retired, they do not necessarily frequent prime bombing targets at prime times. Eshkol and Katz[18] studied 94 victims of suicide bombings in Tel Aviv and found the median age was age 41 years (range, 14-81 years). In a study by Kluger et al[19] of the Israeli National Trauma Registry, only 10% of the victims of bombings were over age 60 years, as compared to 23.8% of individuals who experienced conventional trauma. In reviewing data from nine trauma centers in Israel, Peleg et al[17] reported that 70% of the victims of terrorist bombings were younger than age 29 years, with only 3.3% being older than age 60. It should be noted, however, that these numbers may not be an accurate representation of how the elderly are affected by bombings, since they only take into account survivors of attacks; there is some evidence that the elderly are more likely to be killed at the scene.

One study of the victims at Netanya's Park Hotel bombing in March 2002 may better reflect the potential impact of bombings on the elderly.[20] The mean age of the survivors was 55 ± 27, while the mean age for the individuals killed by the blast was 82 ± 16.8. Twenty-two percent of the victims died at the scene, and an additional eight victims died after being hospitalized.[20] Head injuries and age were the strongest predictors of mortality, both at the scene and among those hospitalized.[20] The elderly are at greater risk for secondary complications from head trauma due to cortical atrophy and shearing of the bridging veins. Of the eight patients who later died in the hospital, all were over age 60 years, all had head injuries, and all had injury severity scores (ISS) greater than 24 at the time of admission.[20]

In general, injuries from bombings are divided into four categories: (1) primary blast injuries; (2) secondary blast injuries; (3) tertiary blast injuries; and (4) quaternary blast injuries. Primary blast injuries are caused by the explosion itself and oftentimes result in rupture of internal organs (lungs, bowel, spleen), tympanic membranes, and the aqueous humors of the eyes. Secondary blast injuries result from projectiles, which can often lead to internal bleeding, fractures,

and puncture wounds. Tertiary blast injuries are caused when victims are forced into stationary objects by the explosion, resulting in lacerations, brachial plexus injuries, crush injuries, and fractures. The elderly are particularly susceptible to this type of injury due to decreased bone density and reduced body mass. Quaternary blast injuries are caused by the heat and fire generated by the explosion, which results in burns of the flesh and lungs. A suggested fifth category is secondary toxic exposure to smoke and other inhaled toxins, such as the neurotoxic Teflon® fumes emitted from burning vehicles.[15,21]

In a study by Eshkol and Katz[18] of bombing victims, 67% of fractures were of the long bones, while 33% were facial/skull fractures. Peleg et al[17] found that 47% of bombing victims suffered injury to the head and neck. The most common blast injuries occurred to the extremities (55%).[17] The high incidence of extremity injury is likely explained as defensive wounds, which occur when people try to shield themselves from the explosion and its resulting debris. In general, the most common injuries seen in bombings among those who survive the initial explosion are multiple lacerations caused by flying metal fragments (nails, ball bearings) and other debris (glass, stone, building materials).[18] The most common internal injuries occur in thoracic organs (lungs, heart, vasculature).[17] Nearly half of the bombing victims, who were admitted to hospital, required surgical intervention for exploration, debridement, internal fixation, or other procedures.[17,19] Long-term complications included persistent osteomyelitis, chronic pain from embedded foreign objects and traumatic nerve injury, secondary infection, bed sores, loss of hearing, ataxia and other disturbances of balance, loss of sight, and chronic respiratory disorders.[15,17-21]

In their study of the Israeli National Trauma Registry, Kluger et al[19] tried to identify differences in trauma due to terrorism versus conventional trauma. They found that bombing trauma presented with greater severity of injury as indicated by higher ISS and lower Glasgow Coma Scale (GCS) scores. Bombing victims had more body regions affected, were twice as frequently hemodynamically unstable upon arrival at the hospital, required more surgical intervention (eg, debridement, open fixation), and required more intensive care time and longer hospitalizations. They also had an increased hospital mortality (both acute and delayed) and greater utilization of rehabilitation services after discharge.[19]

There are several documented case reports describing how suicide bombers are either already infected or are intentionally injecting themselves with infectious viruses such as hepatitis B, HIV, or hepatitis C prior to their attacks.[18,21-23] These infectious components increase the psychological trauma/uncertainty and drain on health resources after the attack. When they detonate their explosives, the terrorists' biological material (blood, bone, and body tissue) becomes infectious projectiles that can cause life-threatening illness in victims beyond the effective killing radius of the explosion itself. To date, there has been one documented case of hepatitis B transmitted from a suicide bomber to a victim.[22] The Israel Ministry of Health guidelines for treatment of suicide bombing victims include broad-spectrum antibiotics, anti-tetanus toxin, and hepatitis B vaccination.[23] In addition, bone fragments and any available tissue from bombers' bodies are sent to national laboratories to be tested for infectious diseases.[18]

CHEMICAL ATTACKS

Chemical agents used to be thought of as the domain of the military. They were seen as unsavory and difficult-to-control weapons, and were banned by the Geneva Convention.[4,24] They are now viewed as

potential terrorist weapons. Chemical weapons can be categorized based on their mechanism of action, persistence in the environment, and lethality.[24] The main categories are nerve agents (sarin and VX), blister agents (mustard gas), caustic agents (acids), pulmonary agents (cyanide, chloride, and bromine), and incapacitating agents (anxiogenics, nauseants, and tear gas).[5,9,24-27] (A full review of all of these categories is beyond the scope of this article; however, additional information is listed in Table II.)

Nerve agents

In 1994 and 1995, there were two incidents when Aum Shinrikyo used the nerve agent sarin against Japanese cities.[24,25,28] The cult was able to injure 3796 people and cause 12 deaths in Tokyo using plastic bags

TABLE II

Potential Chemical Agents and Characteristics

1. *Nerve agents—acetylcholinesterase inhibitor*
   - Tabun (GA)—Ethyl N,N-dimethylphosphoramidocyanidate, $C_5H_{11}N_20_2P$
   - Sarin (GB)—O-Isopropyl methylphosphonofluoridate, $C_4H_{10}FO_2P$
   - Soman (GD)—O-Pinacolyl methylphosphonofluoridate, $C_7H_{16}FO_2P$
   - Cyclosarin (GF)—Cyclohexyl methylphosphonofluoridate, $CH_3PO(F)OC_6H_{11}$
   - VX—O-ethyl-S-(2[diisopropylamino]ethyl) methylphosphonothioate, $C_{11}H_{26}NO_2PS$

2. *Blister agents*
   - Nitrogen mustards (HN1, HN2, HN3)—chemotherapy agents with properties similar to sulfur mustards
   - Sulfur mustards (HD, H, HT, HL, HQ)—odor of onion, garlic, or mustard, colorless to yellow-brown appearance, family includes mustard gas which was used during WWI
   - Lewisite (L)—oily brown liquid, smells like geraniums

3. *Caustic agents*
   - Hydrochloric acid—corrosive to skin, vapor irritates mucus membranes, HCL
   - Sulfuric acid—corrosive to skin, vapor irritates mucus membranes, H_2SO_4

4. *Pulmonary agents*
   - Arsine—colorless gas, heavier than air, with odor of garlic, AsH_3
   - Bromine (Br)—red vapor, toxic on inhalation, vapor irritates eyes and mucus membranes
   - Chlorine gas (Cl)—lighter than air, smells like bleach
   - Hydrogen sulfide—colorless gas, heavier than air, odor of rotten eggs, H_2S
   - Phosgene (CG)—results in pulmonary edema, smells like moldy hay, $COCl_2$
   - Cyanide (-CN)—inhibits cytochrome oxidase, preventing Krebs cycle from properly functioning
   - Hydrogen cyanide (AC)—lighter than air, bitter almond smell, which some people are genetically unable to detect, also known as prussic acid when in water
   - Cyanogen chloride (CK)—heavier than air

5. *Incapacitating agents*
   - 2-chlorobenzalmalononitrile (CS)—form of tear gas, C and S are the initials of the discoverers, $C_{10}H_5ClN_2$
   - Chloroacetophenone (CN)—form of tear gas used by American military in Vietnam, $C_8H_{13}ClO$
   - Dibenzoxazepine (CR)—British-developed lachrymatory agent, $C_{13}H_9NO$
   - Oleoresin capsicum (OC) spray—lachrymatory agent derived from peppers, often used for riot control by police
   - Pelargonic acid morpholide (MPK)—synthetic pepper spray developed in Russia
   - 3-quinuclidinyl benzilate (QNB, BZ, Agent 15)—anticholinergic agent designed to cause confusion and mental status changes, $C_{21}H_{23}NO_3$
   - Kolokol-1—Russian-developed opiate-derived agent, which results in loss of consciousness. Used in the Moscow theater siege in 2002

filled with sarin that were punctured with sharpened umbrella tips.[24] There has also been a failed sarin attack attempted against a Paris train station.[29] Nerve agents are a perfect weapon of choice against any city with underground transportation, since many of these agents are heavier than air and will linger in a confined low-lying area. Chemical agents can also be added to conventional bombs, which increase the mortality and delay rescue efforts.[21]

Nerve agents interfere with neurosynaptic transmission by preventing the breakdown of acetylcholine by binding to acetylcholinesterases.[5,24,25] This produces increased levels of acetylcholine at the synaptic cleft, which overstimulates the nicotinic (neuromuscular junction and sympathetic ganglia) and muscarinic (smooth muscle and exocrine glands) receptors.[5,24,25] Symptoms from nerve agent exposure depend on route (inhalation vs skin absorption) and the degree of exposure. Symptoms can include rhinorrhea, increased salivation, vision changes, miosis, headache, bronchospasm, dyspnea, vomiting, abdominal pain, urinary and fecal incontinence, muscle fasciculation, mental status changes, convulsions, coma, and respiratory failure[5,24,25] (Table III).

Organophosphates were the first nerve agents, and were originally developed by Germany in the 1930s while searching for a commercial insecticide. The organophosphates are clear, colorless liquids whose vapor is heavier than air, allowing them to sink to lower levels and remain in the location in which they are released.[24,25,29] They present the greatest danger and speed of action when inhaled in a vaporized form.[24,25,29] These agents can be absorbed through contact with mucus membranes or the skin.[24] Treatment of nerve agent poisoning includes atropine (muscarinic antagonist), pralidoxime (class of oxime used to reactivate acetylcholinesterase), anticholinergics, and diazepam (muscle relaxant and convulsion prevention).[5,24,25,29] The effectiveness of pralidoxime decreases with time due to a process called "aging," where the acetylcholinesterase undergoes spontaneous de-alkylation, making it difficult to reactivate.[24]

Decontamination is a critical step in treating victims of chemical exposure. It must be accomplished

TABLE III

Symptoms Caused by Chemical Agents

Nerve toxins	Cyanide	Blister agents (Vesicant)	Chlorine
Rhinorrhea	Irritated eyes	Tearing	Blurred vision
Excessive salivation	Upper airway irritation	Eye pain	Tearing
Sweating	Nausea	Corneal edema	Shortness of breath
Twitching	Dizziness	Conjunctival edema	Coughing
Nausea	Weakness	Blindness	Sore throat
Vomiting	Anxiety	Nausea	Wheezing
Diarrhea	Loss of consciousness	Vomiting	Chest tightness
Loss of consciousness	Convulsions	Hypotension	Chest pain
Convulsions	Respiratory arrest	Bradycardia	Dyspnea
Respiratory arrest	Mydriasis (dilated pupils)	Burning pain	Tachypnea
Flaccid paralysis	Muscular fasciculations	Nasal irritation	
Miosis (pin-point pupils)		Sneezing	
Injected conjunctiva		Pulmonary edema	
Eye pain		Skin erythema	
Blurred vision		Thin-walled blisters	
Chest tightness		Bone marrow suppression	
Shortness of breath		Immunosuppression	
Cough		Respiratory compromise	
Fecal incontinence			

TABLE IV

Symptoms of Radiation Sickness Based on Exposure

Acute Radiation Sickness (> 0.7 Gy Initial Exposure)	Hematologic Syndrome (> 0.75 Gy)	Gastrointestinal Syndrome (> 10 Gy)	Nervous/ Cardio-vascular Syndrome (> 30-50 Gy)	Long-Term Effects
Nausea	Lymphopenia (detected first)	Damage to epithelial cell lining	Listlessness	Amenorrhea
Anorexia	Neutropenia (day 7-14)	Bloody diarrhea	Drowsiness	Sterility
Vomiting	Thrombocytopenia (day 14-21)	Bacteremia secondary to bowel necrosis	Tremors	Hematological disturbances
Headache	Pancytopenia	Alteration in bowel motility	Convulsions	Cataract formation
Malaise	Lymph system dysfunction	Electrolyte imbalances	Ataxia	Premature aging
Diarrhea (bloody?)	Spleen dysfunction	Vomiting	Fluid shifts	Leukemia
Tachycardia	Bone marrow suppression	Sepsis	Cerebral edema	Skin cancer
Fatigue (72 h)	Impaired wound healing	Death usually in weeks	Hypotension	Breast cancer
Fever			Hyperpyrexia	Lung cancer
Prostration			Death (hours to days)	Thyroid cancer
Respiratory distress				
Hyperexcitability				

Gy = grays.

in a well-ventilated area to prevent continued re-exposure and cross contamination of first responders and other healthcare providers. The importance of decontamination was evident in the sarin attack on the Tokyo subway, where many of the first responders and medical staff were secondarily poisoned from patients, who were improperly decontaminated and treated in poorly ventilated areas.[21,30-33]

The elderly have a higher risk of mortality when exposed to chemical agents due to their already diminished pulmonary and neurological reserves, bodily limitations for clearing toxins, and reduced tolerance to many of the antidotes used.[25,29] When atropine is used, physicians need to be prepared for its potential complications, such as arrhythmias, delirium, and heat stress.[24]

NUCLEAR ATTACKS

The issues with nuclear terrorism will depend primarily on the exposure encountered. If there is a nuclear attack, effects like those seen at Hiroshima and Nagasaki will occur, such as radiation skin burns, radiation sickness, hematological conditions secondary to bone marrow failure, increased risk of cancer, and temporary or permanent blindness[29,34-37] (Table IV). If an attack occurs with a "dirty bomb," then the results will be difficult to predict.[38] Chernobyl serves as one model for the long-term health effects of radiation if the concentration of the radiological agent is high. In general, lymph tissue and bone marrow are the most sensitive tissues, while the most sensitive organs are the skin, testes, and gastrointestinal (GI) tract, which are areas characterized by rapid cell division and growth.[37] Risk factors that determine the severity of illness include the degree of exposure, pre-existing health conditions, advanced age or extremely young age, and male gender.[37]

Acute radiation sickness characteristically occurs after a brief full-body exposure to a large dose of ionizing radiation.[35,36] Radiation sickness presents in

four phases: (1) a prodromal phase; (2) a latent phase with decreased or clearing symptoms; (3) a return-to-illness phase; and (4) the period of recovery or death.[36] The latent phase usually begins 1-2 days after the prodrome, with its duration depending on the degree of exposure. Patients in the illness phase can present with fever, infection, electrolyte imbalance, diarrhea, skin burns, bleeding, cardiovascular collapse, and/or changes in mental status.[35-37] The presence of bloody diarrhea is an indicator of high mortality. It may result from bone marrow aplasia, injury to vascular structures, or GI injury.[37] Death may occur after acute exposure of 1 gray (SI unit of absorption that equals 1 joule/kg or approximately 100 rads) or more.[35-37] Death can occur as soon as 1-2 days post-exposure, with a peak incidence at about 4 weeks post-exposure and cases tapering to completion at about 8 weeks.[37] Of note, inhaled plutonium usually results in acute respiratory failure within 1 week.[37]

OTHER MEDICAL EFFECTS

The indirect medical effects following terrorist events are difficult to predict. Much depends on the type of attack and general disruption of life following the attack. The elderly are at a much greater risk to societal disruption than the general population due to their decreased mobility, higher usage of prescription medications, and pre-existing health problems. Conditions most at risk if a sudden discontinuation of medicine occurs include chronic obstructive pulmonary disease (COPD), congestive heart failure, hypertensive urgency, diabetes, glaucoma, anxiety, depression, and psychosis.[1] Health problems secondary to disruptions of the health infrastructure and daily routine include increased rates of malnutrition, disruption in hemodialysis, and lack of colostomy and other medical supplies.

An increase in somatic complaints is commonly seen following terrorist attacks. Studies have reported an increase in pain complaints, headaches, sleeplessness, and heart palpitations since the September 11, 2001, attacks.[39] What is particularly interesting is that these somatic effects are seen well outside of the areas directly affected by the attack.[39-41] Four percent of the population of Ontario, Canada, visited a healthcare professional for relief from stress and anxiety following the September 11 attacks.[40]

The scope of how widespread somatic effects are on a population can be seen from the radiation accident that occurred in Goiânia, Brazil, in 1987. In that incident, a radiotherapy capsule of cesium-137 spread throughout a neighborhood, which resulted in four deaths from radiation sickness. More than 100,000 unexposed people (approximately 10% of the city's population) sought medical treatment for anxiety-related somatic complaints during this emergency.[39]

Other life-threatening medical complications that occur after a terrorist attack include an increased number of heart attacks, arrhythmias, and firings of internal defibrillators.[41,42] After September 11, these events were noted to occur as far away as Florida.[41] One study of prescheduled heart-monitored patients in New Haven, Connecticut, detected decreased parasympathetic tone during the week of September 11 as compared to controls. The study concluded that the psychological stress caused by the terrorist attack might have led to an increased incidence in sudden cardiac death from arrhythmias induced by changes in sympathetic tone.[43]

CONCLUSION

No matter what form terrorism takes, the elderly are at increased risk for mortality, severity of effects, secondary complications, and increased hospitalizations. Some of the unique challenges that are pre-

sented by the elderly are atypical presentations secondary to immune deficiencies, increased comorbidity, and limited data for specific treatment and dosing. Since the probability of future terrorist attacks remains high, further study of the impact of these attacks on the elderly is critical.

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