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Being Prepared
Bioterrorism and Mass Prophylaxis: Part II

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ABSTRACT
Although several biological agents have been recognized as presenting a significant threat to public health if used in a bioterrorist attack, those that are of greatest importance are known as the Category A agents: *Bacillus anthracis* (anthrax); variola major (smallpox); *Yersinia pestis* (plague); *Francisella tularensis* (tularemia); ribonucleic acid viruses (hemorrhagic fevers); and *Clostridium botulinum* (botulism toxin). In the previous issue, Part I of this review focused on the clinical presentation and treatment of anthrax, plague, and tularemia. In this second part of this 2-part review of these agents, the focus is on the clinical presentation and treatment of smallpox, viral hemorrhagic fevers, and botulism toxin. The utilization of mass prophylaxis to limit the morbidity and mortality associated with all these agents is also discussed along with the role emergency care personnel play in its implementation. **Key words:** anti-infective agents, biological warfare, botulism, bioterrorism, disaster planning, hemorrhagic fevers, smallpox, variola

BIOTERRORISM is the use of biological agents (e.g., bacteria, viruses) to intentionally harm humans, animals, or crops (Centers for Disease Control and Prevention [CDC], 2007). Many of the agents that have historically been used for this purpose have been restricted or banned (Bush, Abrams, Beall, & Johnson, 2001; Bush & Perez, 2012). This however does not negate their potential for terrorists to acquire and deploy them, as was demonstrated by the 2001 anthrax attacks on the United States (Wright, Quinn, Shadomy, & Messonnier, 2010).

In 1999, a group of experts gathered to identify those biological agents that are of the greatest concern for being utilized in a bioterrorist event (Rotz, Khan, Lillibridge, Ostroff, & Hughes, 2002). These agents were placed into three different priority categories: A, B, or C (see Table 1). Category A agents were

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deemed to present the greatest threat to the public health. The agents included in this category are *Bacillus anthracis* (anthrax), variola major (smallpox), *Yersinia pestis* (plague), *Francisella tularensis* (tularemia), ribonucleic acid viruses (hemorrhagic fevers), and *Clostridium botulinum* (botulism toxin).

Serving in their daily role, emergency departments continue to represent the front line of public health detection and response. Thus, it is critical for emergency care personnel to be aware of the most significant biological threats, their clinical presentation, and their treatment. Furthermore, it is imperative that they are cognizant of the provision of mass prophylaxis to the general population and develop their own hospital-specific plans. Part II of this two-part review focuses on the clinical presentation and treatment of smallpox, viral hemorrhagic fevers, and botulism toxin. It also explores the provision of mass prophylaxis and the role of the emergency care worker.

VARIOLA VIRUS (SMALLPOX)

Variola virus is a large, double-stranded DNA virus that causes smallpox (Darling, Catlett, Huebner, & Jarrett, 2002). It technically exists in two forms: variola major, causing severe smallpox with a mortality rate of 20%–40%, and variola minor, causing mild smallpox with a mortality rate of less than 1% (Henderson et al., 1999; Klietmann & Ruoff, 2001). It was first introduced to North America by the Spanish and resulted in the deaths of approximately 33% of the existing Native American population in the decades after their arrival (Kman & Nelson, 2008). The disease was declared eradicated in 1979, at which

Table 1. Categories of bioterrorism agents/diseases

Category A	Anthrax (*Bacillus anthracis*)
	Botulism (*Clostridium botulinum* toxin)
	Plague (*Yersinia pestis*)
	Smallpox (variola major)
	Tularemia (*Francisella tularensis*)
	Viral hemorrhagic fevers (filoviruses [e.g., Ebola, Marburg] and arenaviruses [e.g., Lassa, Machupo])
Category B	Brucellosis (*Brucella* species)
	Epsilon toxin of *Clostridium perfringens*
	Food safety threats (e.g., *Salmonella* species, *Escherichia coli* O157:H7, *Shigella*)
	Glanders (*Burkholderia mallei*)
	Melioidosis (*Burkholderia pseudomallei*)
	Psittacosis (*Chlamydia psittaci*)
	Q fever (*Coxiella burnetii*)
	Ricin toxin from *Ricinus communis* (castor beans)
	Staphylococcal enterotoxin B
	Typhus fever (*Rickettsia prowazekii*)
	Viral encephalitis (alphaviruses [e.g., Venezuelan equine encephalitis, eastern equine encephalitis, western equine encephalitis])
	Water safety threats (e.g., *Vibrio cholerae*, *Cryptosporidium parvum*)
Category C	Emerging infectious diseases such as Nipah virus and hantavirus

Note. Retrieved from http://emergency.cdc.gov/bioterrorism/.

time vaccination programs for the general population began to cease, and the military terminated their vaccination programs in 1989 (Breman & Arita, 1980; Turnbull, 1991). As a result, it is estimated that the vast majority of the current U.S. population is likely susceptible to smallpox. Currently, smallpox virus stocks exist at two World Health Organization collaborating centers in the world: the CDC in Atlanta and the Russian State Research Center of Virology and Biotechnology in the Russian Federation (Darling et al., 2002). However, this does not exclude the possibility that other stockpiles may exist elsewhere in the world, obtained through security breaches or from unreported stockpiles (Breman & Henderson, 1998; Franz et al., 1997). Unlike anthrax, smallpox is highly contagious and given how susceptible today's population is, each primary case can lead to 10–20 secondary cases, making its potential reintroduction into the environment catastrophic (Franz et al., 1997; Henderson et al., 1999).

Clinical Presentation

Smallpox is transmitted from person to person through inhalation of virus-containing respiratory particles and direct contact, although scabs are much less infectious. It replicates in the respiratory tract and migrates to lymph nodes, the spleen, and bone marrow. Ninety percent of smallpox infections occur in three distinct clinical phases in unvaccinated individuals: incubation, the prodromal phase, and the eruptive phase (Nafziger, 2005). Infection by smallpox is followed by an incubation period of 12–14 days (range = 7–19 days), during which time the patient is asymptomatic and transmission risk is low. As the virus migrates throughout the body, the patients will progress to the prodromal phase, lasting 2–4 days, when patients will display symptoms of fever, malaise, headache, backache, rigors, and vomiting. As the fever begins to decline, a maculopapular rash develops in a centrifugal distribution within 2–4 days after the onset of fever. Transmission occurs at the height of the fever and tends to coincide with the onset of the skin rash, during which time the patients are most infectious. The rash begins in the mouth and rapidly spreads to the hands and the forearms, eventually extending to the legs and the trunk. Smallpox is contrasted with varicella (chickenpox) in that with chickenpox there is a higher concentration of lesions on the trunk than on the extremities or the face (see Figure 1). Smallpox lesions start as maculopapular, discolored skin patches, and then become vesicular, opaque, and pustular. These lesions erupt during a short period (1–2 days), and they all tend to evolve at the same rate, contrasting again with varicella in which all the lesions are found in different stages of maturation. On the eighth or ninth day of the rash, the pustules begin to dimple as they become filled with granular tissue and around day 14 they dry up and crust over. By about day 19, most pustules scab and separate (Henderson et al., 1999). Infectivity wanes as scabs develop, but patients can remain infectious even after death. Death usually occurs at the end of the first or second week of the illness and occurs from bronchitis, pneumonitis, pulmonary edema, associated bacterial pneumonia, and sepsis.

Infection Control Precautions

Within the first few days, the virus is shed into oropharyngeal secretions from lesions on the mucous membrane, and the patient

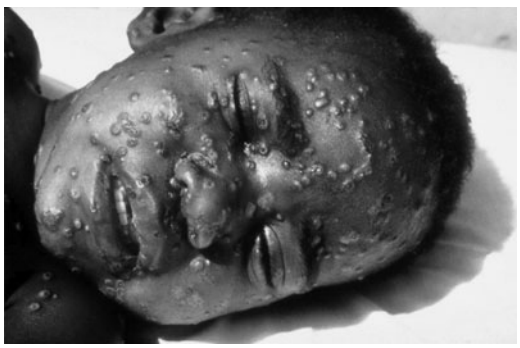

Figure 1. Face lesions on a boy with smallpox. Public Health Images Library (PHIL) ID #3. Used with permission from Cheryl Tyron, Centers for Disease Control and Prevention.

is contagious until all scabs are healed. Strict quarantine is therefore required for symptomatic patients until all scabs are completely healed (Franz et al., 1997). When possible, patients should be isolated at home (Henderson et al., 1999). Patients admitted to the hospital should be placed in a negative-pressure room with high-efficiency air filtration (HEPA). In addition to standard precautions, all hospital workers should use airborne and contact precautions.

Treatment and Prophylaxis

Management of acute smallpox infection is largely supportive as there are no proven treatments. Although several of potential antiviral therapies have been investigated (including thiosemicarbazone, cytarabine, and vidarabine), currently no antivirals have proven effective in humans (Branda & Ruoff, 2002). However, cidofovir, a nucleoside analogue DNA polymerase inhibitor that has been studied in animal models, has been shown to be effective in vitro (see Table 2) (Henderson et al., 1999). It might prove useful if administered within 1–2 days postinfection but may also cause renal toxicity. The Working Group on Civilian Biodefense does not currently recommend it or any other drug over postexposure vaccination (Henderson et al., 1999).

The smallpox vaccine is the oldest of all vaccines, with smallpox-infected materials being used to confer immunity as far back as 1000 BC (Cleri, Porwancher, Ricketti, Ramos-Bonner, & Vernaleo, 2006). Following the identification of a smallpox outbreak, large-scale vaccinations should begin immediately in coordination with the CDC and use of federally stockpiled supplies. Smallpox is different from many other diseases in that postexposure vaccination may be effective in preventing or modulating the disease if given within 2–3 days after the infection and it offers substantial protection against a fatal outcome (see Table 3; Franz et al., 1997; Kman & Nelson, 2008). Subjects who have been exposed (i.e., face-to-face contact with a symptomatic patient), but are unvaccinated and asymptomatic, should be immediately

Table 2. Category A agent treatment

Agent	Therapeutic options	Dosage
Variola virus (smallpox)	Cidofovir[a]	Still under investigation
Viral hemorrhagic fevers	Ribavirin	*Intravenous:* Loading dose of 30 mg/kg once 16 mg/kg intravenously every 6 hr for 4 days 8 mg/kg intravenously every 8 hr for 6 days *Oral:* Loading dose of 2,000 mg once 600 mg twice daily
Clostridium botulinum toxin (botulism)	Trivalent equine botulinum antitoxin (A, B, and E)	One 10-ml vial per patient, diluted 1:10 in 0.9% normal saline intravenously
	Monovalent human antiserum (Types A and B)	50 mg/kg intravenously once
	Heptavalent antitoxin[a] (Types A–G)	Still under investigation

Note. From Hendricks et al. (2014); Franz et al. (1997); Darling et al. (2002); Dennis et al. (2001); Eliasson, Broman, Forsman, and Back (2006); Kman and Nelson (2008); Shapiro et al. (1997).
[a]Not Food and Drug Administration approved for this indication.

Table 3. Category A agent vaccines

Agent	Therapeutic options
Variola virus (smallpox)	A multiple-puncture technique uses a bifurcated needle that holds the recommended dosage of vaccine. Vaccinators should refer to the materials that accompany the vaccine shipment for instructions on the number of required punctures.

Note. From Branda and Ruoff (2002); Franz et al. (1997); Kman and Nelson (2008).

vaccinated and be placed under surveillance (Henderson et al., 1999). Asymptomatic, exposed patients who have been vaccinated previously should be considered for revaccination regardless of the duration since their last shot (Franz et al., 1997; Henderson et al., 1999). It is possible that individuals in this category have only partial immunity and revaccination will provide additional protection against smallpox. Health care workers who have been recently vaccinated should avoid patients until the scab at the vaccination site has separated from the skin. Adverse reactions from the vaccine include progressive vaccinia (i.e., the live virus contained in the smallpox vaccine), eczema vaccinatum, generalized vaccinia, accidental infection, and contact infection (Darling et al., 2002). However, the most serious complications include postvaccinia encephalopathy and encephalitis. For those who do develop complications because of the vaccine, a vaccinia immunoglobulin has been developed and is available from the CDC. Civilian health care providers who need consultation and potential release of vaccinia immunoglobulin should contact their state and local health departments or the CDC at 770-488-7100.

VIRAL HEMORRHAGIC FEVERS

Viral hemorrhagic fevers are caused by a variety of ribonucleic acid viruses: Arenaviridae, Bunyaviridae, Filoviridae, and Flaviviridae (Darling et al., 2002). The diseases caused by these viruses include yellow fever, dengue, hantavirus fever, Lassa fever, Ebola virus hemorrhagic fever, and Marburg virus hemorrhagic fever, and Argentine, Bolivian, Brazilian, and Venezuelan hemorrhagic fevers. These agents all cause fever, malaise, vomiting, bleeding diatheses, edema, and hypotension that can progress to death. In general, all of these are highly infectious via aerosolization and have animal reservoirs with arthropod vectors. Although each disease has its own unique characteristics, all have the final common pathway of diffuse hemorrhage and bleeding diathesis.

Most viral hemorrhagic fevers are spread by fine-droplet aerosol. However, in Africa, the transmission of these viruses has been associated with unsterile needle reuse and the provision of patient care without appropriate barrier precautions. In their endemic forms, they reside in animal hosts or arthropod vectors (e.g., mosquitoes); therefore, the viruses are confined to the geographic distribution of their hosts (Borio et al., 2002). Humans can be infected via aerosol generated from infected rodent excreta, through the bite of an infected arthropod, or by direct contact with infected animal carcasses, blood, or bodily secretions. Although humans are not natural hosts for viral hemorrhagic fevers, infected people can spread the disease and cause community outbreaks. Travel to these endemic areas can result in the spread of these viruses to nonendemic areas. Infections acquired percutaneously are typically lethal and are associated with the shortest incubation times (Saks & Karras, 2006).

Clinical Presentation

Each type of fever can present quite differently and can be influenced by the route of infection, the virulence of the virus, the specific viral strain, the health of the individual patient, vaccination status, and the quantity of the inoculums (Darling et al., 2002). The major differences among the viruses are their primary organ targets. Hemorrhagic fever viruses are attracted to the vascular system and exert their deleterious effects by causing increased vascular permeability, leading to flushing and petechial hemorrhages (Kman & Nelson, 2008). Clinically, the early stages of disease are associated with fever, headache, malaise, myalgia, arthralgia, abdominal pain, vomiting, and nonbloody diarrhea. The incubation period of the fever ranges from 2 days to 3 weeks. The nonspecific prodrome of symptoms described lasts less than 1 week. Later in the disease progression, patients may show signs of bleeding diatheses. When the bleeding occurs, it may manifest as mucous membrane or conjunctival hemorrhage, hematemesis, hematuria, or melena. Disease progression can lead to disseminated intravascular coagulation, hypotension, and circulatory shock. Signs of central nervous dysfunction, such as delirium, seizures, or coma, portend a poor prognosis. Death with these agents can come quickly, typically between days 6 and 9 of infection. Death from viral hemorrhagic fever is preceded by hemorrhagic diathesis, shock, sepsis, or multisystem organ failure. Patients who survive this disease may be left with permanent hearing or vision loss, impaired motor coordination, transverse myelitis, hepatitis, pancreatitis, as well as damage to various other organ systems.

Infection Control Precautions

As aerosol transmission is possible with the majority of these viruses, it is important that aerosol precautions are strictly maintained (Darling et al., 2002). All of the bodily fluids are also contagious. Each patient should be cared for in a negative airflow room, with an anteroom to be used for decontamination and handwashing. Normal barrier protective garments should be adequate; however, HEPA-filtered positive pressure masks combined with impermeable Level A protective suits would provide an added level of protection. Strict barrier protective measures against nosocomial infection include hand hygiene, double gloves, impermeable gowns, N-95 masks or air-purifying respirators, negative pressure isolation rooms, leg and shoe coverings, face shields and eye protection, restricted access to all nonessential staff, dedicated disposable (or single-use) medical equipment, and Environmental Protection Agency–approved disinfectant.

Treatment and Prophylaxis

Therapy for all viral hemorrhagic fevers is aggressive supportive care (Kman & Nelson, 2008). Large quantities of fluids, electrolytes, and blood products are likely to be needed. Invasive procedures, although often necessary, should be limited secondary to the risk of needlestick infections, which carry high morbidity and mortality rates (Darling et al., 2002). The goal of therapy is to maintain function and allow recovery in causalities where possible. Close personal contacts of patients and personnel exposed to blood or secretions from a patient should be monitored for signs and symptoms during the incubation period.

Currently, no antiviral drugs are Food and Drug Administration approved for this indication; however, ribavirin, a nucleoside analogue, has had some activity in treatment if the hemorrhagic fever is caused by an arenavirus or bunyavirus (see Table 2; CDC, 1988; Darling et al., 2002). Because diagnostic confirmation takes days, when a viral hemorrhagic fever is identified, treatment with ribavirin is initiated as soon as possible. If infection with an arenavirus or bunyavirus is confirmed, a 10-day course of ribavirin is continued. If infection with a filovirus or flavivirus is confirmed or another diagnosis is pursued, ribavirin treatment is discontinued. Ribavirin can be given intravenously or orally depending on disease severity, drug availability,

and the number of patients requiring treatment. In a situation with a limited number of patients, the Working Group on Civilian Biodefense recommends intravenous therapy (Borio et al., 2002). As casualties grow, such as in a mass casualty situation, oral ribavirin can be given in the same dose recommended in chronic hepatitis C infection (see Table 2). Ribavirin is not recommended for prophylactic treatment of asymptomatic contacts (Borio et al., 2002). Vaccines are currently not useful in preventing the spread of viral hemorrhagic fever. Yellow fever is the only viral hemorrhagic fever with a licensed vaccine. Unfortunately, this vaccine would not be helpful in treating exposed patients because it takes longer than the 3- to 6-day incubation period to produce antibodies (Borio et al., 2002).

CLOSTRIDIUM BOTULINUM TOXIN (BOTULISM TOXIN)

This is a gram-positive bacterium found in its natural habitat, the soil, around the world (Darling et al., 2002). The syndrome that is caused by this toxin is produced by the bacteria, *Clostridium butyricum* and *Clostridium baratii*. Cases of botulism are typically categorized according to transmission as a foodborne illness acquired from the ingestion of the toxin in food, wound botulism secondary to soil-contaminated wounds, and infantile illness from the ingestions of spores (Arnon et al., 2001). *Clostridium* spores are resilient and able to survive at a wide range of temperatures. Clostridia vegetate and produce botulinum toxin in oxygen-poor, low-salt, low-sugar, and low-acidity environments (Villar, Elliott, & Davenport, 2006). Seven distinct types of toxin exist with similar mechanisms of action and have similar effects on the body, identified as Types A–G. Types A, B, and E are most often responsible for human disease, whereas Types C and D cause disease in other animals. The toxin contains an enzyme that blocks acetylcholine transmission, resulting in flaccid paralysis. By causing paralysis of the diaphragm, this toxin causes death through respiratory compromise.

Botulinum toxin is the most poisonous substance known; it is 100,000 times more lethal than sarin gas and 15,000 times more lethal than the chemical agent VX gas (Osterbauer & Dobbs, 2005; Torok et al., 1997). A single gram of inhaled crystalline toxin could kill more than 1 million people. The Soviet Union and Iraq have both tested and developed botulinum toxin as a weapon. It is also believed that Iraq, Iran, North Korea, and Syria are developing botulinum toxin as a weapon (Arnon et al., 2001).

Although botulinum toxin could be used to sabotage food supplies, the more likely scenario in a bioterrorist attack would involve the attempted dissemination of this agent as an aerosol. Iraq is known to have produced 20,000 L of botulinum toxin during the Gulf War, 12,000 L of which were used in field-testing and to fill warheads (Zilinskas, 1997). Despite efforts to weaponize this agent, it is difficult to deploy this as a weapon of mass destruction because of rapid degradation of the toxin in the environment, rendering it nontoxic minutes after its dispersal.

Clinical Presentation

All seven neurotoxins act through the same mechanism and impair acetylcholine release (Darling et al., 2002). Recovery occurs when the neuron regenerates to replace the toxin-damaged one. In adults, this process can take weeks to months. Chemical effects are seen at the neuromuscular junction and cholinergic sites, resulting in cranial nerve deficits and descending muscle weakness that are the clinical hallmarks of all forms of botulism. This is in contrast to other disease states, such as Guillain-Barré syndrome, which have an ascending pattern.

Botulism cannot be spread from person to person. Naturally acquired botulism is most commonly contracted by eating food that has been contaminated with spores (e.g., honey, home-canned foods) (Arnon et al., 2001). The spores germinate to bacilli that produce toxin that is rapidly absorbed by the gastrointestinal epithelium. Inhalational botulism is the most likely bioweapon-induced clinical syndrome. Health care providers could

expect victims to manifest symptoms between 12 and 80 hr after attack. Patients with botulism are afebrile and have a clear sensorium. All patients with botulism show signs of diplopia, ptosis, blurred vision, sluggishly reactive pupils, dysphonia, dysarthria, and dysphagia. Paralysis of the pharyngeal and upper airway muscles results in obstruction, and paralysis of the diaphragm results in inadequate ventilation. Eventually, dysphagia and loss of the gag reflex make intubation inevitable, and if untreated, death results from airway compromise. The Working Group on Civilian Biodefense has described a classic triad of botulism infection: (1) symmetric, descending flaccid paralysis with prominent bulbar palsies in (2) afebrile patients with (3) a clear sensorium (Arnon et al., 2001). The bulbar palsies can be summarized as the "4 D's": diplopia, dysphagia, dysarthria, and dysphonia.

Infection Control Precautions

Botulism is not contagious and so health care workers should be instructed to use standard precautions (Darling et al., 2002). Because botulism is difficult and costly to treat once illness occurs, avoidance of contamination is paramount (Kman & Nelson, 2008). Proper food preparation, storage, and consumption can easily eliminate most cases of foodborne botulism. Contaminated clothing and skin should be washed with soap and water; other objects should be cleaned with 0.1% hypochlorite bleach solution.

Treatment and Prophylaxis

The mainstay of treatment is supportive care with fluids, nutrition, and, often, mechanical ventilation (Kman & Nelson, 2008). Patients should be monitored for their ability to protect their airway. Treatment of the patient with botulinum toxin poisoning relies on ventilator support because respiratory failure secondary to muscle paralysis is the most serious complication (Darling et al., 2002). Supportive care can be supplemented with passive immunization with equine or human antitoxin. Three different antitoxin preparations are available in the United States (see Table 2) (Shapiro, Hatheway, Becher, & Swerdlow, 1997). A trivalent equine botulinum antitoxin, containing antibodies to Types A, B, and E, is available through the CDC and some state health departments (Kman & Nelson, 2008; Shapiro et al., 1997). Local and state health departments should call the CDC 24-hr telephone number at 770-488-7100. For infant botulism, a monovalent human antiserum (Type A) is available from the California Department of Health Services. For suspected cases of infant botulism, consultation, and to obtain BabyBIG (R), call 510-231-7600, 24-hr a day. An equine heptavalent antitoxin against all seven serotypes (A–G) is available from the U.S. Army Medical Research Institute of Infectious Diseases, with its permission and support under an Investigational New Drug protocol (www.uamriid.army.mil).

The administration of antitoxin may prevent progression or shorten the course of the illness (Darling et al., 2002). It acts by neutralizing toxin that is not yet bound to nerve terminals and has a half-life of 5–8 days in the circulation. It should therefore be given as soon as possible after the clinical diagnosis. Botulism toxin binds irreversibly; therefore, antitoxin cannot reverse effects that have already occurred but can help stop disease progression. Antitoxin is generally not recommended if exposure occurred greater than 72 hr before administration.

MASS PROPHYLAXIS

The Strategic National Stockpile (SNS) is a collection of antibiotics, vaccines, chemical antidotes, medical equipment, antitoxins, and supplies to serve as countermeasures to Category A threats and supplement local supplies (CDC, 2000, 2012). The SNS is deployed to local jurisdictions following a request that originates at the local level. It is intended to assist local jurisdictions prepare and respond to a large-scale natural disaster or a terrorist event. Local health departments are tasked with the

responsibility to dispense these assets within their jurisdiction. The SNS does not provide personnel, facilities, or transportation support outside of the initial delivery of the supplies to the region.

Mass prophylaxis plans for health care providers, emergency response personnel, and the general population should be based on the concept of Point of Dispensing (POD). These PODs serve as a modality for dispensing medicines and supplies to the population during an emergency (Khan & Richter, 2012). The CDC and the Department of Health and Human Services encourage local jurisdictions to be able to provide antibiotics to the entire population within their area within 48 hr. It is felt that this time frame provides the maximum benefit of postexposure prophylaxis among those people who have been exposed to a Category A agent.

Points of Dispensing sites are typically locations that the community is familiar with such as convention centers, community centers, and, in some cases, schools (Khan & Richter, 2012). They are usually located in areas with high population densities and can provide easy access and parking, and they are located close to public transportation. It is recommended that PODs be nonclinical sites to help ensure that treatment centers are free to continue treating their existing patients during an emergency.

Emergency personnel have knowledge that can assist planning efforts at various stages in this process. In addition, an important aspect of POD planning involves determining what the role will be for health care professionals during such a response. If professionals are expected to participate directly in activities such as triage, evaluation, and dispensing, it may be necessary to develop educational materials and provide in-service training sessions directed at these providers. Even if health care professionals are not expected to have direct patient care responsibilities during such an event, their expertise is most assuredly necessary for the education of patients about mass prophylaxis, providing directions to dispensing sites and providing follow-up care after the event.

Although preparation for these events occurs largely at federal and state levels, it remains necessary for health care providers to establish their own preparedness plans (Lee, Johnson, & Sohmer, 2009). Ensuring that health care professionals and first responders receive adequate prophylaxis and/or vaccination is essential for the provision of medical care to the general population during an event. In addition, it is essential that they are also well aware of the response plan and have received adequate training prior to an event. A template preparedness plan for use by health care facilities has been made available by the CDC and the Association for Professionals in Infection Control and Epidemiology (Klietmann & Ruoff, 2001). Institutions are encouraged to develop specific response plans in conjunction with local and state health departments. In all likelihood, the initial recognition of a bioterrorist action will be at the local level and hence a rapid public health response will depend on the effective recognition and response by local health care providers (Henderson, et al., 1999; Khan, Morse, & Lillibridge, 2000; Simon, 1997). Up-to-date information on bioterrorist agents, treatment, prophylaxis, and response plans can be found at http://emergency.cdc.gov/bioterrorism/ and http://www.fda.gov/drugs/emergencypreparedness/.

CONCLUSION

Category A agents are of the utmost concern for public health and health care practitioners in preparing to effectively respond to a bioterrorist event. The causal agents of smallpox, viral hemorrhagic fevers, and botulism toxin are some of the most complex, lethal, and challenging agents in this category. In the case of smallpox and viral hemorrhagic fevers, the potential for human-to-human transmission further adds to the intricacies of responding to such an event. Emergency departments and emergency care personnel will invariably serve as the first line of defense against such

an event and will be tasked with diagnosing and treating patients in its early phases. Becoming knowledgeable regarding all of the Category A agents, and the response necessary for the provision of mass prophylaxis, will be critical to protecting the public and limiting potential morbidity and mortality.

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