TERRORIST ATTACK WITH CHEMICAL WEAPONS DURING THE 21 CENTURY CHALLENGE FOR ANESTHESIOLOGISTS

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ABSTRACT
This review article addresses the leadership of anesthesiologists in terrorist acts using chemical weapons. Since treatment of diseased with chemical warfare agents requires not only supportive and ventilating control but also aggressive treatment titrated optimal medical therapy applied in a hazardous environment, in which the rescue should be prepared for a large volume of emergency activities subject to anesthesia providers. From initial sorting, rescue of victims and evacuation to final hospitalization. With little effort anesthesiologist can be prepared for the worst.

Key words: hazardous substances, nerve agent, antidotes, Acetylcholinesterase

In recent years the intentions and behavior of terrorist organizations appear to have changed. In their zeal to hasten the end of this world, the Japanese Aum Shinrikyo ("Supreme Truth") doomsday cult repeatedly released anthrax and botulinum spores in the winter of 1993/1994. Failing these attempts at biological mayhem because of insolvable technical glitches, the Aum turned to nerve agent (nerve gas), this time achieving their sinister end. An elite strike team first released sarin vapor in the central Japanese city of Matsumoto in June 1994, causing 300 or more casualties and seven deaths. Five two-man teams next (March 1995) deployed nerve gas in Tokyo's subway cars by puncturing a dozen liquid sarin-filled plastic bags. The underground bedlam was unspeakable: of the 5510 injured seeking medical care, more than 1000 were held for observation or treatment, including 17 severe exposures that survived and 12 that did not. To bring home the potential enormity of the chemical threat to civilians, recall the 1984 industrial accident that released a plume of methylisocyanate and chlorine gas over the sleeping Indian city of Bhopal. Of the estimated 38,000 immediate inhalation casualties, some 8000 eventually perished (1). With the chemical nerve gas attack in the Tokyo subway system in 1995 and two simultaneous bomb blasts at United States embassies in East Africa in 1998, it became evident that these organizations were ready to sacrifice thousands of civilians for their causes. At that time there were already those saying that we had to prepare ourselves for living in a world increasingly at risk from terrorist attacks. And if anyone was still in doubt, this was confirmed by the terrible events on September 11, 2001. After the nerve gas attack in Tokyo (2), both civil and military authorities in several countries started pursuing a number of activities to counter paramilitary and terrorist threats from nuclear, biological, and chemical agents. Several review articles on the management of victims exposed to chemical weapons appeared in the medical literature. Since the September 11, 2001 attacks and the subsequent anthrax scare, there has been growing concern about the risk of terrorist use of chemical and biological agents. As a part of continuing preparation for this new
reality, many governments have been preparing guidelines on how to respond to this new threat (3), and some local communities have conducted simulated terrorist incidents (drills) to provide training for the primary response teams and hospital staff. The dispersal of a chemical warfare agent in an urban area has the potential to cause thousands of casualties, rapidly overwhelming local health and medical resources (2). Because treatment of nerve gas mass casualties requires not only ventilatory assistance or control, but also acute pharmacologic therapy titrated to a clinical end-point, all in a hostile environment, incident responders must be trained in largely anesthesia-related emergency measures from initial rescue through triage and evacuation to eventual in-hospital stabilization (1). Under these circumstances, anesthesiologists may have a major role in the treatment of casualties brought to the health services. Their role may be mainly in the initial resuscitation and airway management of critically-ill patients before decontamination, treating patients with combined conventional and toxic injuries in the perioperative period, and in treating ventilated patients with severe nerve gas intoxication. Training anesthesiologists and other members of the medical teams to treat casualties in this situation is complicated because the scenario is one of mass casualties and also because of the unfamiliar medical situation and physical conditions. This includes the need to train in realistic conditions where medical teams are expected to be physically protected from secondary contamination, and to decontaminate patients before providing definitive medical treatment. The traditional training included not only frontal lectures, but also hands-on training with simulated patients undergoing decontamination and simulated treatment while medical personnel were in full protection gear. However, traditional drills have always focused more on the logistic aspects of the scenarios and were deficient in providing opportunities for medical teams to actually exercise and practice clinical procedures and the actual management of critically-ill patients. The developing field of interactive patient simulators has opened new horizons in simulation-based medical training. The availability of these simulators, coupled with the growing need to improve health professional competencies in disaster medicine, have led to the creative use of advanced simulators in chemical warfare training. Medical performance in this situation may be influenced by physiological and/or psychological variables, namely, increased respiratory effort and restricted visual field induced by the gas mask, interference with manual dexterity by the chemical protective gloves, and excessive heat load induced by the multilayered overgarment (3). Chemical terrorism is causing such concern for the local community-based authorities responsible for civil emergency issues because chemical agents, especially nerve gases, are fast-acting and can cause mass casualties in urban areas within a very short time. Counter-measures can save many lives but require appropriate emergency response at the scene with detoxification facilities, administration of antidotes, and emergency life-support. As nerve agents have such rapid action, the counter-measures must be available within minutes rather than hours. This means that basic decontamination and protection equipment, supplies of antidote, and trained rescue and medical teams, must be available without delay in urban areas. To offer reasonably good safety coverage for urban communities, major hospitals would have to be equipped to take care of victims who have been exposed to chemical nerve agents. Although paramedics and emergency department staff would, in most cases, be in the front line in a major chemical warfare incident, they would soon be overwhelmed and anesthesiologists would certainly be the next in line because of their ability to respond to the need for life-support. Anesthesiologists’ extensive knowledge of physiology and pharmacology helps them to understand the effects of chemical nerve agents. They should also know the basic principles of treatment of mass casualties after exposure to chemical nerve agents, as their expertise may be needed when a mass terrorism event occurs and such an event may occur virtually anywhere, anytime. The so-called nerve agents, or acetylcholinesterase blockers, are the most dangerous chemical weapons today. They have acquired their name because of their effects on the
nervous system: they prevent the breakdown of acetylcholine, causing widespread peripheral and central neuronal paralysis, which can be rapidly fatal without timely administration of antidotes (4). The nerve agents, including tabun, sarin, soman, and VX, are all organophosphorous compounds closely related to the organophosphate pesticides. They are chemically rather stable, easily dispersed, highly toxic, and are effective within minutes when absorbed either through the skin or through the airways. Nerve agents can be manufactured using fairly simple chemical techniques in improvised laboratories. The raw materials are inexpensive and generally readily available. In addition, these agents are easy to transport and difficult for controlling authorities to detect. Sarin, which was used in the Tokyo subway blast, is one of the most likely agents to be used by terrorists. It is a colorless and odorless gas/vapor and is more than 20 times more deadly than cyanide gas and potassium cyanide. The vapor is slightly heavier than air; thus it remains close to the ground during calm weather conditions. Until now most protocols have suggested the use of IM injection of antidote in the case of mass casualties from chemical nerve agents. However, IM resorption is delayed and insufficient in many cases. It is also usually very difficult to obtain IV access in these victims, and therefore other options for administration of antitoxin, such as intraosseous injection, are needed. The intraosseous needles used in clinical practice in emergency departments are not easily used in the field by personnel dressed in heavy protective clothing (2,5). Anesthetic education, however, is different for weapons of mass destruction, especially as regards to protective gear, specialized equipment, and the interactions between antidotes and anesthetics. Regardless of how many anesthesiologists are trained, they are only capable of being effective if supplied with the necessary medications, equipment, and facilities.

Acetylcholinesterase (AChE), one of nature’s most efficient enzymes, hydrolyzes approximately 10,000 acetylcholine (ACh) molecules per second to inactive products. Predictably, its inhibition causes nearinstant endogenous ACh flooding that presents as a fulminating cholinergic crisis composed of: (a) muscarinic effects at holinergic end-organs such as secretory glands (rhinorrhea and bronchorrhea), smooth muscle contraction (miosis, asthmatic wheezing, and bladder and bowel hyperactivity), and cardiac pacemaker (vagal bradycardia); (b) nicotinic effects at the neuromuscular junction (skeletal muscle fasciculation, weakness, and eventual depolarization paralysis); and (c) central manifestations of neurotransmitter accumulation (convulsions and respiratory center depression). The proximate cause of death is respiratory, due to a combination of massive airway flooding, intense bronchospasm, progressive ventilatory muscle paralysis, and impaired respiratory center drive (1,6). It is imperative that rescue personnel be protected at the least with chemical filter respirator (gas mask) and butyl rubber gloves before entering a chemical vapor contaminated incident scene. If VX droplet spray or liquid spill are suspected, the complete protective suit, butyl rubber boots, gloves, and hooded mask ensemble must be donned. The immediate rescue scenario merely sorts walking unexposed and mildly exposed subjects from the more severely exposed downed casualties who require medical attention before evacuation. Triage granularity can be refined progressively as medical personnel and materiel converge on the scene. Decontamination of vapor-exposed casualties requires little more than removal to an airy outside location and, where feasible, perhaps an exchange of clothing. Conversely, decontamination of liquid droplet exposure to eyes, skin, or clothing becomes a most urgent priority to minimize transcutaneous nerve agent absorption. (Caution: hot water, scrubbing, and strong detergents or bleaches increase perfusion of abraded skin, thereby hastening chemical agent absorption. Although casualties respiratory depression or apnea require immediate assistance with ventilation, pulmonary gas exchange must be isolated from chemical vapor-containing ambient air. Mouth-to-mouth insufflation is contraindicated because the rescuer not only would inhale toxic air, but also, during exhalation, will pass it on to the casualty. A self-inflating (Ambu®) bag and mask open to ambient air, likewise, forces toxin-containing air down the casualty’s
lungs. However, adequate lung isolation can be secured by connecting the self-inflating bag’s unidirectional valve tail to an oxygen tank or filtered compressed air source. An oral airway greatly facilitates gas exchange and aids suction access by lifting the tongue from the lax posterior pharynx. Optimally, endotracheal intubation of apneic and near-apneic casualties isolates the lungs from toxic vapor, secretions, and vomitus, forces gas transport through constricted bronchi, and permits suctioning of the deeper airways. The cuffed cricothyroidotomy rescue device offers features comparable to tracheal intubation, but the safety of rescue cricothyroidotomy is suspect, and its use in nerve agent casualties is undocumented. The laryngeal airway mask seals insufficiently when lung compliance is poor and offers inadequate airway protection from voluminous secretions and vomitus, whereas the esophageal-tracheal CombiTube® precludes suction access to the trachea, according to recent Israeli Defense Force analysis. Apneic oxygenation has the potential for satisfying the short-term oxygen demand of apneic casualties, without further ventilatory assistance, by flowing oxygen at a low rate (approximately twice the metabolic requirement) through a wide-bore transtracheal needle. Apneic oxygenation has been proposed as an expedient alternative to intubation for apneic mass casualties because it can sustain oxygenation for 30-45 min, albeit at the price of progressive hypercarbia, provided that the lower airways have been cleared by lateral positioning and suction, and are kept dry by atropine administration. Nerve agent antidote administration during the initial rescue phase relies on IM injection because IV lines are far too awkward to insert while wearing a gas mask. Auspiciously, therapeutic blood levels of atropine and pralidoxime appear within minutes in fit volunteers injected IM with the Mark-I selfadministration kit; however, antidote absorption becomes erratic when muscle perfusion declines as circulation falters. Large and repeated amounts of atropine, up to 3 separate 2-mg IM doses initially and then more after, must be given to block the muscarinic actions of endogenous Achetilcholine (secretions, sweating, and wheezing). Pralidoxime (Protopam® [2-PAM]), up to 3 separate 600-mg IM doses initially (1,7), if available to rescuers, is co-administered with atropine to regenerate AChE at the muscle end plate and restore skeletal (respiratory) muscle function(1,8).

As a profession, anesthesiology should take a lead role in the training all of our personnel. Protecting ourselves as providers is key to maintaining our preparedness and well-being. We believe that at the minimum, anesthesiologists should receive lectures on the management of patients exposed to toxic chemicals, infectious agents, and warfare mass destruction coupled with hands-on training with the proper use of protective gear (such as suits and respirators). Familiarity with basic decontamination techniques and locations of facilities will greatly enhance warfare mass destruction agents treatment and reduce the commotion associated with these events. By being well prepared and increasing the awareness of the anesthesiology community to threats posed by warfare mass destruction agents, we will ensure better patient safety and reduce the harm associated with these events. Because of the apparent threat of warfare mass destruction agents, it seems prudent to have this type of training available at all anesthesiology residency programs. Whether this training should be mandatory and how often it should be repeated is a subject of some debate (3 ). The world is changing, and anesthesiologists need to be prepared to respond to those changes—terrorist attacks may occur virtually anywhere, any time(2,9,10).

REFERENCES
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