The VX Nerve Agent

Understanding the risks of a deadly threat

By Geary Randall Sugg

TERRORISM AFFECTS EVERYONE, everywhere, every day. It is fast becoming part of the SH&E professional's job to recognize the vulnerability risk factors associated with it. Many HazMat training programs and seminars now cover weapons of mass destruction (WMD). Although there are four basic types of WMD—chemical, ordnance, biological and radiological (COBRA) [DOJ(a)]—this article primarily focuses on one deadly chemical threat commonly known as the VX nerve agent. Although all nerve agents are deadly, the VX nerve agent is the "baddest of the bad." In the wrong hands and with the right devices, it could possibly be disseminated to murder millions.

The case study presented is hypothetical and similar to many training scenarios studied by first responders across the country. Because terrorists' potential targets include commercial and industrial facilities, SH&E students should also benefit from studying it, as it is designed to stimulate group discussion, and increase awareness and understanding of what terrorism is and the risks associated with such an incident. The discussion following the case study provides answers to the challenge questions posed about it.

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The Case Study

One warm summer Saturday afternoon around 2:00 pm, a security camera captured an act of terrorism occuring inside the food court of a crowded shopping mall. Other cameras were mounted inside and outside the mall area. The FBI reviewed the footage after the incident to develop a more thorough sequence of events. Following is a summary of those events.

A man of medium height and build, with short black hair and a mustache, walked up to a tall trash receptacle and dropped a large brown paper sack into it. A busboy noticed that part of the sack was hanging out of the dispenser. He removed the sack and set it on top of the trash receptacle so he could properly install a fresh plastic liner. Moments later, a sharp popping sound was heard followed by a hissing noise (like that of an aerosol can). An elderly man eating at a table less than 10 feet away from the trash can immediately started choking and fell out of his chair to the floor.

As the cashier dialed 9-1-1, the restaurant manager knelt down beside the man to attempt a first-aid maneuver learned in a recent CPR class. Suddenly, the restaurant manager grabbed his chest, vomited and fell forward to the floor. Both men lay on the floor, twitching and jerking as they began foaming at the mouth. Other people began gathering around the two men, hoping they could somehow help, but they also began coughing and staggering. In addition, many became confused and one individual suddenly lost his vision. Paramedics soon arrived, expecting to find one possible cardiac patient. What they found instead was an array of lifeless bodies. They began triage and patient assessments, but within seconds they, too, began to experience similar symptoms and lost consciousness.

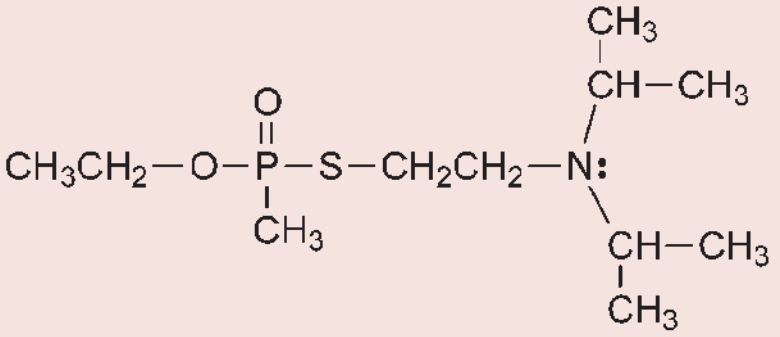
When a local fire engine company arrived, panicked customers were pouring out of the mall, tripping, falling and running over each other. Many were coughing and some appeared to be choking and gasping for breath. When the fire company officer stepped off the truck, a mall security guard

informed him that he had observed the situation on monitors located in the security office and had immediately called for full evacuation of the facility. He said he counted at least eight people down and showing no signs of movement. The fire officer tried to contact the EMTs, but received no response.

After quickly assessing the situation, he concluded that a fast-acting airborne hazard was inside, requiring Level A protection. Although difficult to resist the temptation to rush in, he knew regular structural firefighting suits with SCBAs would not offer the protection needed for such an atmosphere. Plus, it was not yet known what deadly agent had been released. The fire officer then contacted central dispatch and requested that a second alarm be given for police who could provide traffic control and that more EMS workers be summoned. He also requested that the county HazMat team be activated.

Figure 1

Chemical Structure of VX Nerve Agent



Challenge Question 1

What signs and symptoms did the victims in the food court area exhibit? Autopsies revealed that the elderly man and restaurant manager died within a few minutes. What type of hazard (e.g., inhalation, skin contact) did they likely encounter to cause death so quickly?

Challenge Question 2

Why did the company officer in this scenario elect not to have his firefighters rush in for a search-and-rescue immediately on arrival?

Challenge Question 3

Assuming drugs were available, how would you treat a patient who was exposed to VX nerve agent?

What Is VX?

VX nerve agent is one of the most dangerous manufactured chemicals. It was first developed in Port Down, Wiltshire, England in 1952, where its devastating effects were tested. The U.S. received it by trading information on thermonuclear weapons to Great Britain. (The agent was featured in the film *The Rock,* as a somewhat green liquid used by a terrorist to threaten San Francisco.)

Classes of nerve agents have not changed in more than 50 years. What has changed, however, is the understanding of their potency. New considerations include the fact that more women (who may be affected differently than men) are now serving in the military and the growing likelihood that civilians will be targeted. Concern regarding the nonlethal but delayed effects that may be produced by nerve agents is growing as well (Reutter).

Biological & Chemical Weapons: A Brief Overview

Biological and chemical weapons have been used throughout history in warfare and terrorist activities. As far back as 600 BC, the Assyrians contaminated the water supply of their enemies with Rye Ergot. When the plague broke out among the ranks of the Tartar army during its siege of Kaffa in 1346, they used catapults to hurl the corpses over the city's walls in order to start an epidemic. Even Napoleon attempted to infect the people of Mantua with swamp fever during his Italian campaign in 1797. The Japanese army reportedly used various biological agents during World War II to contaminate food and water supplies of Chinese cities (Stockholm International Peace Research Institute).

Eventually, chemical weapons were intentionally developed and used. Mustard gas and nerve agents are the two major classes of chemical weapons that pose a significant threat to the world's population. Mustard was first synthesized in the early 1800s and used by the Germans against the British at Ypres, Belgium, on July 12, 1917, during World War I. The use of mustard and chlorine resulted in approximately 400,000 casualties during that war. Even though the Geneva Convention of 1925 prohibited the use of chemical weapons, production continued and many countries began to stockpile them.

Since World War I, it is believed that mustard gas has been used by Great Britain in the Middle East; the French in Morocco; Italy against Ethiopia in 1936; Japan against China in 1937; Poland against Germany in 1939; the Russians in central Asia; Egypt against Yemen from 1963 to 1967; Iraq against Iran during the Iran-Iraq War; and Iraq against the Kurds [Reutter(b)]. Terrorists have also used these agents on civilian populations in Japan. On March 20, 1995, members of the Shinrikyo religious sect released sarin at several points in the Tokyo subway system, killing 11 and injuring more than 5,500 people (Ohbu and Yamashina 587).

Nerve agents consisting of basically two classes,

Table 1

Chemical Agent Comparison: Symptoms & Hazards

Symbol/ Common Name	Agent Type	Symptoms	Hazard
GB (sarin) GD (soman) GA (tabun) VX	Nerve agents	Dim or blurred vision; pinpointing of the pupils; running nose; salivation; nausea and vomiting; sweating; tightness of chest; difficulty breathing; convulsions; loss of consciousness; tachycardia; incontinence; death.	Skin dose effective in minutes to hours. Respiratory dose effective within seconds to minutes. Skin exposure has a better survival rate but VX is fatal if inhaled or absorbed through the skin. These agents are extremely toxic and lethal.
HD	Blister agent, commonly called "mustard"	Blisters and reddening of skin; eye pain, reddening and damage; sneezing and coughing; airway irritation and damage; diarrhea, fever and apathy.	Although it absorbs rapidly into the skin, eye effects may appear in a few hours; respiratory effects and blisters in two to 24 hours. It can be lethal in large doses.
L (lewisite)	Blister agent	Immediate stinging pain or irritation of skin; instant eye pain with severe permanent damage or blindness; running nose; coughing, sneezing and lung edema; other symptoms similar to the HD agent.	Immediate pain. Most symptoms in about 12 hours. It can be lethal in large doses.
CX (phosgene oxime)	Blister agent	Immediate burning; corrosive to skin; eyes, nose and airway irritation and damage; victim can drown in own mucous.	Immediate pain with other symptoms shortly thereafter. It can be lethal in large doses.
CK (cyanogen chloride) AC (hydrogen cyanide)	Blood agents	Flushed (reddish skin); cherry red lips (blue in African-American or other dark-skinned races); confusion; drowsiness; headache; gasping for air; frothing or vomiting, then weakness, unconsciousness, collapse and death.	It can cause death in six to eight minutes.
CG (phosgene) CL (chlorine)	Choking agents	Corrosive to skin and eyes; irritates throat and lungs; dizziness; tightness of chest; delayed pulmonary edema.	In very high doses, it can cause death after several days.

Source: DOJ. COBRA WMD Responder Guide.

G and V, were conceived in the 1930s. They belong to a group of chemicals called "organophosphates" that were originally developed in 1854 to control insects and save crops. German scientist Gerhard Schrader synthesized the GA nerve agent, also called "tabun," in 1936. The GB agent "sarin" was synthesized about a year later, and "soman" or "GD" was made in 1944. Although it appears that Germany did not use these agents during World War II, it is believed that the country had tons of both. After the war, the U.S. and Russia also became large producers and stockpilers of tabun and sarin. As noted, England developed the worst nerve agent yet, named VX, in the 1950s. It is the primary V-type agent that is the least volatile but most potent of all nerve agents. The "V" stands for venom because of its extreme potency (Reutter, et al).

Accidental Release of VX

An accidental release of VX occurred on March 13, 1968, at the Dugway Proving Grounds in Utah. A malfunction in a spray tank used to test aerial spraying, coupled with unfortunate winds, resulted in the contamination of grazing land outside the test site's boundaries. Although no human injuries were reported, more than 6,000 sheep in the area died.

In July 1969, a VX leak from weapons at a storage facility on Okinawa led to the hospitalization of 23 U.S. military personnel and one civilian. There is no proof that the VX agent has been used in any large-scale military operation as of yet (CBWInfo).

Physical/Chemical Properties

Nerve agents are similar to insecticides in that they contain either a fluorine, sulfur or cyanide substituent group. VX contains a sulfur substituent group. It is 1,000 to 10,000 times more potent than the most potent organophosphate insecticides commercially available and it is theoretically possible to disseminate it in high enough vapor concentrations

so that one breath can kill (Opresko 440). The agent's chemical formula is:

$CH_3CH_2O\text{-}P\ (O)(CH_3)\text{-}SCH_2CH_2N\ (C_3H_7)_2$

It is also known as O-ethyl S-[2-(diisopropylamino) ethyl]-methylphosphonothioate. Figure 1 (pg. 33) depicts its structure.

Although VX is sometimes referred to as a gas, it is normally a colorless to straw-colored liquid. It has a molecular weight of 267.4; a vapor density of 9.2 (air = 1 STP); and a liquid density of 1.0083 g/ml at 25ºC. The agent has a water solubility of 30 g/L per 100g at 25ºC and a vapor pressure of 0.0007 mm Hg at 25ºC. It has a boiling point of 298ºC at 760 mm Hg; specific gravity of 1.0113; melting point of -50ºC; viscosity (centistokes) of 9.958 at 25ºC; and a volatility of 8.9 mg/m^3 at 25ºC.

Effects on the Nervous System

It is important to first discuss how acetylcholine (a common neurotransmitter) works in order to understand how the VX nerve agent affects the nervous system. Acetylcholine (ACh) is found in the central and peripheral nervous system. When it is released from an axon terminal, ACh moves across the synaptic cleft to bind to a receptor on the other side of the synapse (on the post-synaptic membrane). In the peripheral nervous system, ACh is located at the "neuromuscular junction," where it acts to control muscular contraction. Basically, nerve agents are inhibitors of acetylcholinesterase (AChE), an enzyme responsible for stopping the action of acetylcholine at some neuronal synapses and myoneural junctions. By a mechanism of phosphorylation, VX acts as a substrate for AChE, thereby preventing deactivation of ACh. Simply put, with a depletion of AChE and a buildup of ACh, acetylcholine continues to act (Opresko 442).

Toxicity

Organophosphate nerve agents can act by dermal, oral or inhalation routes of exposure. VX nerve agent attacks all synapses that use acetylcholine as a neurotransmitter. This means both the central and peripheral nervous systems are affected. The anticholinesterase effects can be characterized as being muscarinic, nicotinic or central nervous system (CNS) related. Muscarinic effects occur in the parasympathetic system (bronchi, heart, pupils; and salivary, lacrimal and sweat glands) and result in signs of pulmonary edema, bradycardia, miosis, tearing and sweating. Nicotinic effects occur in the somatic (skeletal/motor) and sympathetic systems, and result in muscle fasciculation, muscle weakness, tachycardia and diarrhea. CNS effects include headache, depression, convulsions and coma, as well as respiratory arrest (it causes copious secretions, paralyzes the respiratory muscles and inhibits the respiratory center of the brain), confusion and slurred speech. Breathing a lethal dose (10 mg-min/m^3) can kill in seconds to minutes, while a lethal dose on the skin can kill in minutes to hours (Munro, et al 952).

Nerve agents also inhibit blood cholinesterases, but these are not the targets of toxicity and are of unknown function. There is lacking correlation between toxic signs and symptoms, dose and degree of blood cholinesterase inhibition. Repeated small exposures can suppress nearly all blood cholinesterase activity while producing only negligible clinical effects. However, its inhibition is often an excellent indicator of nerve agent exposure. As far as chronic toxicity of VX, litte data can be found in the literature.

Reproductive & Developmental Effects

Studies have been conducted on pregnant rats that were dosed with 0.25, 1.0 or 4.0 micrograms VX/kg by subcutaneous (sc) injection on day six to 15 of gestation. The animals were killed on the twentieth day of gestation and the examined fetuses showed no evidence of malformations. Fetal body weight, litter size and sex ratio were within normal limits.

VX effects on the development and reproduction of sheep were also evaluated following the accidental release in Utah. The 79 surviving animals that were pregnant at the time of exposure were evaluated for changes in red blood count and AChE activity, and for signs of toxicity over a six-month post-exposure period. The researchers concluded that VX had little or no effect on fetal growth and development (Opresko 452-455).

Carcinogenicity & Genotoxicity

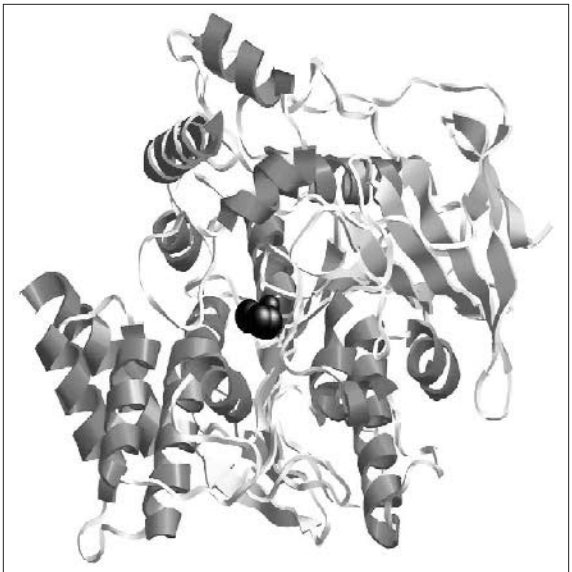
Little information is available on the potential carcinogenicity of VX in humans. No standard, long-term carcinogenicity studies have been performed on lab animals exposed to the agent.

Similarly, information regarding the genotoxicity of VX in humans is not available. Although tests on microorganisms and mammalian cell cultures have been conducted, VX was not found to be mutagenic or was only weakly mutagenic (Opresko 452-455). However, children would have some special vulnerabilities to nerve agents. The release of chemical toxins would disproportionately affect children through several mechanisms. With aerosolized VX, the higher number of respirations per minute in children would result in exposure to a relatively greater dosage. Also, the more permeable skin of newborns and children combined with a larger surface-to-mass ratio results in greater exposure to transdermally absorbed toxicants (AAP).

Treatment

First responders, heathcare providers and other rescue workers wearing protective gear should decontaminate the patient's skin with hypochlorite (household bleach diluted 1:10) or soap and water, and rinse the eyes with plain water. Treatment with drugs can be lifesaving as well. Two drugs, atropine and pralidoxime chloride, have been used as antidotes for nerve agent poisoning. Atropine blocks the action of excess ACh primarily at muscaric sites, decreasing secretions, bronchoconstriction and intestinal motility. The initial dose is 2mg for mild dyspnea to 6mg or more for severe dyspnea or multisystem signs. An

Classes of nerve agents have not changed in many years. What has changed, however, is the understanding of their potency.



Source: Millard, C.B., et al.

This image depicts the structure of acetylcholinesterase with part of the VX nerve agent molecule (black) in the acetylcholine-binding site. The VX nerve agent reacts with the enzyme and a portion of the agent remains covalently attached to the enzyme, thus inhibiting its function.

automatic injection device called the AtroPen auto-injector delivers this intramuscularly; pressing the device against the thigh activates a spring that injects the dose into the muscle. The drug can be given as often as every five minutes until secretions are minimal; some patients require 15 to 20mg during the first three hours after exposure. Miosis may persist but its resolution should not be used as a therapeutic endpoint. There is no direct effect on the nerve agent or AChE.

Pralidoxime chloride attaches to and releases the inhibiting agent from AChE. The initial dose is 1g, given intravenously over 20 to 30 minutes, which can be repeated at hourly intervals for one or two additional doses. An automatic injector dispenses a 600mg dose intramuscularly when the device is pressed against the thigh.

Newer oximes may be more effective than pralidoxime against some nerve agents, but they are not currently commercially available due to ongoing testing. These oximes are termed the H-series oximes and dioximes (HI-6, HL07). They have more ability to reactivate phosphorylated AChE, and have proven to be more combative against the effects of all nerve agents in animal studies because they take direct antimuscarinic and antinicotinic actions to counter these effects. Other promising treatments include the use of exogenous cholinesterase and human monoclonal antibodies. They prevent nerve agents from binding to AChE (Med CEU).

Nerve agents can still cause prolonged convulsive activity leading to severe neuropathology, even if atropine and pralidoxime are used. Because animal experiments suggest that anticonvulsants such as diazepam can prevent brain damage after exposure to nerve agents, a 5 or 10mg dose of diazepam (valium) has been added to the usual regimen in cases of severe nerve agent poisoning ("Treatment of Nerve"). These drugs were issued to U.S. troops during the Persian Gulf War in the form of an antidote kit called the Mark I. (More information on treatment, uses and dosages of lifesaving drugs can be found at www.er365.com/germ%20warfare-nerve%20gas-anthrax-biological%20warfare%20treatment.htm.)

Personal Protective Equipment

It is foolish for first responders to rush into a hazardous situation without first evaluating the incident area and determining the severity of danger. After the initial assessment of a HazMat incident, special considerations must be given to the selection of proper PPE and how it may affect the wearer's ability to execute the rescue.

Chemical protective clothing is designed to shield or isolate first responders from the chemical, physical and biological hazards that may be encountered during HazMat operations. Guidelines, descriptions and recommendations for PPE are based on EPA's widely used levels of protection: Levels A, B, C and D (details at www.epa.gov/superfund/programs/er/hazsubs/equip.htm).

Chemical warfare agents are easily and inexpensively produced and pose a significant threat to military forces, first responders and the public. VX presents both a respiratory and a skin hazard. Although it does not readily produce vapors at room temperature, and is mainly a skin contact hazard, it can be disseminated into the air through devices such as an aerosol can, a bomb or a crop-dusting plane. In liquid form, droplets of nerve agent may be absorbed by the skin; in a vapor state, they may enter the body through the lungs, eyes or GI tract.

Upon initial entry into an area suspected of nerve agent contamination, Level A protection must be worn to ensure full protection of both the respiratory tract and the skin. After a more-accurate assessment of agent concentration in the atmosphere is made, protection may be downgraded to Level B if it is determined that a vapor hazard no longer exists.

Level A includes a totally encapsulated chemical-resistant suit, with a self-contained breathing apparatus (SCBA) or supplied air respirator (SAR) with escape tank. Because this level provides maximum respiratory and skin protection, it is used when there is a high potential for liquid splash, a toxic respiration and skin vapor hazard, or where the chemical agent is unidentified. The responder Level A suit produced by Kappler has been tested and found effective against chemical agents, as has the Trelleborg Trellchem suit. Both support the mission profile used by the U.S. Army Technical Escort Unit (TEU), which provides a secure escort for WMD and is tasked with providing support to civilian agencies in the event of a WMD incident.

Some models of chemical-resistant gloves (e.g., North's SilverShield and Safety4's 4H) have been tested and found to be effective against some types of chemical agents. Various types of chemical-resistant boots are also available to allow full encapsulation. In summary, Level A protection should be worn when the highest level of respiratory, eye, skin and mucous membrane protection is needed. In all cases, the gear must be absolutely vapor tight.

Level B protection includes a nonencapsulating chemical splash-resistant suit with hood and SCBA. The air tank is outside the suit so it provides maximum respiratory protection with less skin protection.

This level is mainly used when an agent presents no skin vapor hazard and a low splash potential exists. Level B can also be worn in low-oxygen environments. This nonencapsulated system requires either an SCBA or SAR and a Tyvek-F or equivalent chem/biosuit with inner gloves. Outer butyl rubber gloves and chemical-resistant boots must be worn.

The advantage of using Level B is that the air cylinders can be removed or changed without opening the suit. A disadvantage is that the SCBA is exposed to the atmosphere. In summary, Level B should be should be worn when the highest level of respiratory protection is needed, but a lesser level of skin and eye protection is required. It is the minimum level recommended on initial site entries until hazards have been further identified and defined by monitoring, sampling and other reliable methods of analysis and personnel equipment corresponding with those findings [DOJ(b)].

Gas masks have received wide media coverage, but they have limitations as well. How well they actually work depends on factors such as their design, manufacture and storage. The nerve agent used, wind speed and the individual's distance from the source of exposure should also be considered. Masks that fit only over the nose and mouth will not likely be effective against nerve agents because the vapor can be absorbed through the conjunctiva. U.S. military standard full-facepiece masks have the adsorbent capacity to protect against many times the LCt50 (concentration and time of exposure that would be lethal for 50 percent of the population) of nerve agents, but some other masks may be less efficient. Any gas mask can lose protective capacity if it is crushed or stored in high humidity. The tightness of the seal around the face is usually more of a limiting factor than the filter's adsorbent capacity. Beards and eyeglasses, among other variables, may interfere as well ("Treatment of Nerve").

To help validate the use of PPE for protection against VX, Wester, et al performed a study to predict VX toxicity to a uniformed soldier using parathion in vitro human skin exposure and absorption (Wester, Quan, et al). The study determined the amount of percutaneous absorption of parathion when applied to bare human skin and uniformed skin with and without sweat. "Parathion percentage dose absorbed through naked skin (1.78 +/- 0.41) was greater than dry uniform skin (0.29 +/- 0.17; $p = 0.000$) and sweated uniform skin (0.65 +/- 0.16; $p = 0.000$). Sweated and dry uniform skin were also different ($p = 0.007$)."

These results were then applied to VX skin permeability for bare skin (head, neck, arms and hands) and the remaining uniformed skin over the various regions of the human body. Risk assessment shows VX 50 percent lethality within the first hour for a soldier wearing a sweated uniform. An eight-hour post-exposure to bare skin plus trunk area predicted lethality for both dry and sweated uniform, and, at 96 hours post exposure, all body regions individually exposed would produce lethality. Military uniform and public clothing provide some immediate protection but VX will be absorbed through cloth and skin. Time following exposure is very critical so exposed clothing must be removed and the skin decontaminated (Wester, Tanojo, et al).

Decontamination

The two levels of decontamination are emergency and technical. Together, these cover the rapid decontamination of victims and the more deliberate decontamination of the first responders at the scene. Emergency decontamination involves neutralizing the agent on the skin and physically removing the agent hazard; it must be performed quickly. Technical decontamination is performed to remove contamination from PPE of rescue workers, equipment and facilities in a deliberate manner. Technician-level responders primarily focus on emergency decontamination of casualties and technical decontamination of responders.

The best decontaminant for the VX nerve agent is 10 percent sodium hydroxide in alcohol. However, it can be neutralized by the commercial types HTH (calcium hypochlorite) and sodium hypochlorite (bleach) or the military types DS-2 and super tropical bleach. Removal of the agent can be accomplished with ethylene glycol, soap and water, adsorbents and sealants [DOJ(c)].

During a basic hydrolysis of VX, it is important to consider that up to 10 percent of the agent is converted to EA2192 (an Edgewood arsenal number and common synonym for diisopropylaminoethyl methylphosphonothioic acid). A Class B poison would result, based on the concentration expected to be formed during hydrolysis and its toxicity (according to animal studies).

The large-scale decontamination procedure, which uses both HTH and NaOH, destroys VX by oxidation and hydrolysis. Normally the large-scale product contains 0.2-0.4 wt. % EA2192 at 24 hours; at pH 12, it has a half-life of about 14 days. Therefore, the 90-day holding period at pH 12 results in about a 64-fold reduction of EA2192 (six half- lives). This holding period is sufficient to reduce the toxicity of the product under that of a Class B poison. Other less-toxic products include diisopropyaminoethyl mercaptan, ethyl methylphosphonic acid, methylphosphonic acid, diethyl methylphosphonate and ethanol. The small-scale decontamination procedure uses enough HTH to oxidize all VX so no EA2192 is formed (Munro, et al 967, 952-953).

Environmental Fate

The volatility of the VX nerve agent is relatively low, having a vapor pressure of 0.0007 mm Hg. A vapor concentration of 10.5 mg/m^3 has been reported for a temperature of 250ºC. Photodegradation is not a significant environmental fate for VX because it does not absorb ultraviolet radiation above 290nm. By reacting with photochemically produced hydroxyl radicals in the troposphere, VX is estimated to have a half-life of approximately six hours. The water solubility of VX is 3g per 100g solvent at 250ºC and 7.5g

Key Terms

Acetylcholine: A neurotransmitter ($C_3H_{17}NO_3$) released at autonomic synapses and neuromuscular junctions, active in the transmission of nerve impulses and formed enzymatically in the tissues from choline.

Acetylcholinesterase: An enzyme that hydrolyzes choline esters and that is found especially in blood plasma.

Atropine: A racemic mixture of hyoscyamine usually obtained from belladonna and related plants of the family Solanaceae and used especially in the form of its sulfate for its anticholinergic effects (as relief of smooth muscle spasms of dilation of the pupil of the eye).

Axon terminal: A usually long and single nerve-cell process that conducts impulses away from the cell body.

Boiling point: The temperature at which the vapor pressure of a liquid equals the pressure of the atmosphere in contact with its surface.

Bradycardia: Relatively slow heart action whether physiological or pathological.

Bronchi: Either of the two primary divisions of the trachea that lead respectively into the right and the left lung.

Central nervous system: The part of the nervous system which in vertebrates consists of the brain and spinal cord, to which sensory impulses are transmitted and from which motor impulses pass out, and which supervises and coordinates the activity of the entire nervous system.

Conjunctiva: The mucous membrane that lines the inner surface of the eyelids and is continued over the forepart of the eyeball.

Convulsion: An abnormal violent and involuntary contraction or series of contractions of the muscles.

Density: Mass per unit volume.

Half-life: The time required for half of something to undergo a process as a) the time required for half of the atoms of a radioactive substance to become disintegrated; b) the time required for half the amount of a substance (as a drug or radioactive tracer) in or introduced into a living system or ecosystem to be eliminated or disintegrated by natural processes.

Intravenous: Situated within, performed within, occurring within or administered by entering a vein.

In vitro: Outside the living body and in an artificial environment.

Lacrimal: Of, relating to, associated with, located near or constituting the glands that produce tears.

Melting point: The temperature at which a solid melts.

Miosis: Excessive smallness or contraction of the pupil of the eye.

Molecular weight: The mass of a molecule that may be calculated as the sum of the atomic weights of its constituent atoms.

Muscarinic effects: Relating to, resembling, producing or mediating the effects (as a slowed heart rate, increased secretion by exocrine glands and increased activity of smooth muscle) that are produced on organs and tissues by acetylcholine liberated by postganglionic nerve fibers of the parasympathetic nervous system and that are mimicked by muscarine.

Neuromuscular junction: The junction of an efferent nerve fiber and the muscle fiber plasma membrane.

Neurotransmitter: A substance (as norepinephrine or acetylcholine) that transmit nerve impulses across a synapse.

Nicotinic effects: Relating to, resembling, producing or mediating the effects that are produced by acetylcholine liberated by nerve fibers at autonomic ganglia and at the neuromuscular junctions or voluntary muscle and that are mimicked by nicotine which increases activity in small doses and inhibits it in larger doses.

Organophosphates: Phosphorus-containing organic pesticides that act by inhibiting cholinesterase.

Oxime: Any of various compounds obtained chiefly by the action of hydroxylamine on aldehydes and ketones and characterized by the bivalent group C=NOH.

Parasympathetic system: The part of the autonomic nervous system that contains chiefly cholinergic fibers, that tends to induce secretion, to increase the tone and contractility of smooth muscle, and to slow the heart rate, and that consists of 1) a cranial part made up of preganlionic fibers leaving and passing the midbrain by the oculomotor nerves and the hindbrain by the facial, glossopharyngeal, vagus, and accessory nerves and passing to the ciliary, sphenopalatine, submandibular, and otic ganglia of the head or to ganglionated plexuses of the thorax and abdomen and postganglionic fibers passing from these ganglia to end organs of the head and upper trunk; and 2) a sacral part made up of preganglionic fibers emerging and passing in the sacral nerves and passing to ganglionated plexuses of the lower trunk and postganglionic fibers passing from these plexuses chiefly to the viscera of the lower abdomen and the external genital organs.

Parathion: An extremely toxic thiophosphate insecticide ($C_{10}H_{14}NO_5PS$).

Peripheral nervous system: The part of the nervous system that is outside the central nervous system and comprises the cranial nerves excepting the optic nerve, the spinal nerves and the autonomic nervous system.

Phosphorylation: The process of phosphorylating a chemical compound either by reaction with inorganic phosphate or by transfer of phosphate from another organic phosphate.

Photodegradation: Degradation by means of radiant energy (as light).

Pralidoxime: A substance ($C_7H_9ClN_2O$) that restores the reactivity of cholinesterase and is used to counteract phosphorylation (as by an organophosphate pesticide)—also called 2-PAM.

Pulmonary edema: Abnormal accumulation of fluid in the lungs.

Somatic: Of, relating to, supplying or involving skeletal muscles.

Specific gravity: The ratio of the density of a substance to the density of some substance (as pure water) taken as a standard when both densities are obtained by weighing in air.

Substituent: An atom or group that replaces another atom or group in a molecule.

Substrate: A substance acted upon (as by an enzyme).

Sympathetic system: The part of the autonomic nervous system that is concerned especially with preparing the body to react to situations of stress or emergency, that contains chiefly adrenergic fibers and tends to depress secretion, decrease the tone and contractility of smooth muscle, increase heart rate, and that consists essentially of preganglionic fibers arising in the thoracic and upper lumbar parts of the spinal cord and passing through delicate white rami communicantes to ganglia located in a pair of sympathetic chains situated one on each side of the spinal column or to more peripheral ganglia or ganglionated plexuses and postganglionic fibers passing typically through gray rami communicantes to spinal nerves with which they are distributed to various end organs.

Synapse: The place at which a nervous impulse passes from one neuron to another.

Tachycardia: Relatively rapid heart action whether physiological (as after exercise) or pathological.

Vapor pressure: The pressure of a vapor in equilibrium with its liquid.

Viscosity: The internal resistance or friction offered to an object moving through a fluid.

Volatile: Readily vaporizable at a relatively low temperature.

Source: MEDLINEplus. <http://medlineplus.nlm.nih.gov/medlineplus/mplusdictionary.html>.

per 100g solvent at 150ºC. A Henry's law constant estimated to be 3.5 x 10^{-9} atm m^3/mol indicates a low potential for evaporation from water. Its evaporation rate is about 1/1500 that of water. VX's reported half-lives in water at 250ºC and pH 7 ranges from 400 to 1,000 hours. Its half-life does increase under acidic conditions (100 days at pH 2 to 3). Low temperatures decrease its rate of hydrolysis even though its solubility is increased at lower temperatures. VX may sink in surface waters and be adsorbed by sediment (U.S. Army). It will remain on the ground in significant concentrations for varying periods of time, depending on factors such as temperature, organic carbon content of the soil and moisture (Opresko 440-442).

Conclusion

If the VX nerve agent is much more dangerous and toxic than its cousins—"G" agents tabun and sarin, which dissipate quickly and have only short-term effects—why hasn't it received greater attention in recent years? U.S. armed forces are currently searching for VX and other agents in Iraq, yet the media has not described its deadly properties in detail. How many Americans are aware that it is present in missiles at seven army depots or arsenals in the U.S., and is stored in ton containers at Tooele Army Depot, Johnston Island in the Pacific Ocean, and Newport Chemical Activity in Newport, IN? In addition, eight nonstockpile sites are located in six states ("Chemical Weapons").

Some of this indifference may be because VX has yet to be used to its fullest potential for local attacks. That raises the obvious question: Why hasn't VX been used as a massive killer to date? One reason may be the way it must be disseminated to produce the greatest harm. If VX is released high into the atmosphere with a bomb or some type of spraying device, it is possible that the wind could shift, blowing the agent right back into the attacker's face. A nuclear counterattack may also be the ultimate deterrent.

On the other hand, terrorism executed on a smaller scale (e.g., suicide missions) is an entirely different scenario that has proven more difficult to defend against. It is possible that VX could be used in a manner similar to that described in the case study. Make no mistake, a little VX could go a long way and it is a WMD, being 600 times more toxic than chlorine if inhaled and requiring 200 times less the quantity of sarin for skin toxicity (Benitez, et al). Even British Prime Minister Tony Blair mentioned VX in a March 18, 2003, speech to the House of Commons on the crisis in Iraq. Blair said that "three kilograms [6.6 pounds] of VX from a rocket launcher would contaminate a quarter of a square kilometer [1/10 of a square mile] of a city" (Blair).

First responders, SH&E professionals and the general public must prepare to face this kind of potential domestic enemy. In the possession of a terrorist, VX nerve agent is like a poisonous viper waiting coiled in the shadows, poised to strike. If wisdom begins with caution, then caution must begin with knowledge of the VX nerve agent. ■

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