262e Chemical Terrorism

Charles G. Hurst, Jonathan Newmark, James A. Romano, Jr.

The use of chemical warfare agents (CWAs) in modern warfare dates back to World War I (WWI). Sulfur mustard and nerve agents were used by Iraq against the Iranian military and Kurdish civilians. Most recently the nerve agent Sarin, GB, was used by the Syrian military against their civilian population. Since the Japanese sarin attacks in 1994–1995 and the terrorist strikes of September 11, 2001, the all-too-real possibility of chemical or biological terrorism against civilian populations anywhere in the world has attracted increased attention.

Military planners consider the WWI blistering agent sulfur mustard and the organophosphorus nerve agents as the most likely agents to be used on the battlefield. In a civilian or terrorist scenario, the choice widens considerably. For example, many of the CWAs of WWI, including chlorine, phosgene, and cyanide, are used today in large amounts in industry. They are produced in chemical plants, are stockpiled in large tanks, and travel up and down highways and railways in large tanker cars. The rupture of any of these stores by accident or on purpose could cause many injuries and deaths. In three attacks in February 2007, for example, insurgents in Iraq used chlorine gas released from tankers after explosions as a crude form of chemical weaponry; these attacks killed 12 people and intoxicated more than 140 others. Countless hazardous materials (HAZMATs) that are not used on the battlefield can be used as terrorist weapons. Some of them, including insecticides and ammonia, could wreak as much damage and injury as the weaponized chemical agents.

Many mistakenly believe that chemical attacks will always be so severe that little can be done except to bury the dead. History proves the opposite. Even in WWI, when IV fluids, endotracheal tubes, and antibiotics were unavailable, the mortality rate among U.S. forces on the battlefield from CWAs—chiefly sulfur mustard and the pulmonary intoxicants—was only 1.9%. That figure was far lower than the 7% mortality rate from conventional wounds. In the 1995 Tokyo subway sarin incident, among the 5500 patients who sought medical attention at hospitals, 80% were not actually symptomatic and only 12 died. Recent events should prompt not a fatalistic attitude but a realistic wish to understand the pathophysiology of the syndromes these agents cause, with a view to treating expeditiously all patients who present for care and an expectation of saving the vast majority. As we prepare to defend our civilian population from the effects of chemical terrorism, we also must consider the fact that terrorism itself can produce sequelae such as physiologic or neurologic effects that may resemble the effects of nonlethal exposures to CWAs. These effects are due to a general fear of chemicals, fear of decontamination, fear of protective ensembles, or other phobic reactions. The increased difficulty in differentiating between stress reactions and nerve agent–induced organic brain syndromes has been pointed out. Knowledge of the behavioral effects of CWAs and their medical countermeasures is imperative to ensure that military and civilian medical and mental health organizations can deal with possible incidents involving weapons of mass destruction.

For the reader's benefit, the CWAs, their two-letter North Atlantic Treaty Organization (NATO) codes (which were established by a NATO international convention and convey no clinical implications), their unique physical features, and their initial effects are listed in Table 262e-1. Table 262e-2 provides guidelines for immediate treatment. The focus of this chapter is on the blister and nerve CWAs, which have been employed in battle and against civilians and have had a significant public health impact.

TABLE 262e-1 RECOGNIZING AND DIAGNOSING HEALTH EFFECTS OF CHEMICAL TERRORISM

Agent Category	Agent Name	Unique Characteristics	Initial Effects
Nerve	Cyclohexyl sarin (GF)[a] Sarin (GB) Soman (GD) Tabun (GA) VX VR	Miosis (pinpoint pupils) Copious secretions Muscle twitching/ fasciculations	Miosis (pinpoint pupils) Blurred/dim vision Headache Nausea, vomiting, diarrhea Copious secretions/sweating Muscle twitching/ fasciculations Breathing difficulty Seizures
Asphyxiant/ blood	Arsine (SA) Cyanogen chloride (CK) Hydrogen cyanide (AC)	Possible cherry-red skin Possible cyanosis Possible frostbite[b]	Confusion Nausea Gasping for air in some cases; similar to asphyxiation but more abrupt onset Seizures before death
Choking/ pulmonary-damaging	Chlorine (CL) Hydrogen chloride Nitrogen oxides Phosgene (CG)	Chlorine is a greenish-yellow gas with a pungent odor. Phosgene gas smells like newly mown hay or grass. Possible frostbite[b]	Eye and skin irritation Airway irritation Dyspnea, cough Sore throat Chest tightness
Blistering/ vesicant	Mustard (HD)/ sulfur mustard (H) Mustard gas (H) Nitrogen mustard (HN-1, HN-2, HN-3) Lewisite (L) Phosgene oxime (CX)	Mustard has an odor like burning garlic or horseradish. Lewisite has an odor like penetrating geranium. Phosgene oxime has a pepperish or pungent odor.	Severe irritation Redness and blisters of the skin Tearing, conjunctivitis, corneal damage Mild respiratory distress to marked airway damage May cause death
Incapacitating/ behavior-altering	3-Quinuclidinyl benzilate, or Agent 15 (BZ)	May appear as mass drug intoxication with erratic behaviors, shared realistic and distinct hallucinations, disrobing, and confusion Hyperthermia Mydriasis (dilated pupils)	Dry mouth and skin Initial tachycardia Altered consciousness, delusions, denial of illness, belligerence Hyperthermia Ataxia (lack of coordination) Hallucinations Mydriasis (dilated pupils)

[a]Letters in parentheses indicate NATO codes for designated agents. [b]Frostbite may occur from skin contact with liquid arsine, cyanogen chloride, or phosgene.

Source: State of New York, Department of Health, as modified by the Chemical Casualty Care Division, U.S. Army Medical Research Institute of Chemical Defense.

VESICANTS: SULFUR MUSTARD

Sulfur mustard has been a military threat since it first appeared on the battlefield in Belgium during WWI. In modern times, it remains a threat on the battlefield as well as a potential chemical terrorism weapon because of the simplicity of its manufacture and its extreme effectiveness. Sulfur mustard accounted for 70% of the 1.3 million chemical casualties in WWI. Occasional cases of sulfur mustard intoxication continue to occur in the United States among people exposed to WWI- and WWII-era munitions.

Mechanism Sulfur mustard constitutes both a vapor and a liquid threat to all exposed epithelial surfaces. The effects are delayed,