Appendix VII

Examples of Chemical Warfare Agents:

Agent Type	**Agent Identification and Common Name**	**Mechanism**	**Time for Effect**	**Symptoms**
Nerve Agents	GA, tabun GB, sarin GD, soman GF, cyclohexyl sarin VX	These agents effectively prevent the transmission of nerve signals by inhibiting the enzyme cholinesterase. This enzyme normally breaks down acetylcholine, the neurotransmitter at cholinergic receptor sites. Cholinergic receptor sites are found at smooth and skeletal muscles, the central nervous system, and most exocrine glands. Accumulation of acetylcholine leads to continued stimulation and clinical symptoms such as muscle paralysis.	**Vapor:** within seconds to several minutes after exposure **Liquid:** within minutes to an hour after exposure. Commonly, there is an asymptomatic period of one to thirty minutes, which is followed by a sudden onset of symptoms.	**Vapor:** *Small exposure:* contraction of pupils, dim vision, headache, mild difficulty breathing *Large exposure:* sudden loss of consciousness, severe breathing difficulty or cessation of respiration, convulsions, muscular twitching, weakness or paralysis, copious secretions **Liquid on skin:** *Small to moderate exposure:* localized sweating, muscle twitching at site of exposure, vomiting, feeling of weakness *Large exposure:* severe breathing difficulty or cessation of breathing, sudden loss of consciousness, convulsions, muscle twitching, weakness or paralysis, copious secretions.
Vesicants	H, HD mustard L, lewisite CX, phosgene oxime	Following absorption, the structure of mustard changes. In this form, it is extremely reactive to water and binds with intra- and extra-cellular enzymes and proteins. Lewisite causes an increase in capillary permeability. The exact mechanisms of mustard, lewisite, and phosgene oxime are not known.	**Mustard:** binds irreversibly to tissue within several minutes after contact. Clinical signs and symptoms may appear as early as two hours after a high dose exposure or extend to twenty-four hours after a low dose vapor exposure.	**Mustard:** Skin, eyes, and airways most commonly affected. Appearance of redness and blisters on skin, irritation, conjunctivitis and corneal opacity and damage in the eyes, irritation of nares, sinus and pharynx and increasingly severe productive cough if the lower airways are affected. **Lewisite:** Skin, eyes, and airways affected by direct contact. Redness and blister formation occur more rapidly than following exposure

			Exposure does not cause immediate pain. **Lewisite:** immediate pain or irritation. Lesions develop within hours. **Phosgene Oxime:** immediate burning and irritation.	to mustard. Eye exposure causes pain and twitching of the eyelid. Edema of the conjunctiva and lids follow, and eyes may be swollen shut within an hour. Contact with airways leads to similar signs and symptoms to mustard. Increased permeability of capillaries resulting in low intravascular volume and shock. May lead to hepatic or renal necrosis with vomiting and diarrhea. **Phosgene Oxime:** Does not cause blisters. Elongated, wheal- like lesions on skin. Damage to eyes similar to that caused by Lewisite. Causes pulmonary edema.
Cyanide	AC, hydrocyanic acid CK, cyanogen chloride	Cyanide ion combines with iron in a component of the mitochondrial cytochrome oxidase complex. This complex is necessary for cellular respiration, an energy-providing process using oxygen. If respiration is prevented, then normal cell functions can not occur.	Death occurs 6 to 8 minutes after inhalation	**Cyanide:** *Small exposure*: No effects. *Moderate exposure*: Dizziness, nausea, feeling of weakness *Large exposure:* Central nervous system and heart are most susceptible to cyanide. 15 seconds after inhalation of a highly concentrated vapor, there is a period of rapid breathing which is followed in 15-30 seconds by convulsions. Respiratory activity stops 2-3 minutes later, followed by cessation of cardiac activity. **Cyanogen Chloride:** *Small Exposure:* Irritation, nausea, feeling of weakness *Large Exposure:* Similar symptoms as those following large exposure to cyanide

SOURCE: Frederick R. Sidell, William C. Patrick IIIm Thomas, R. Dashiell, *Jane's Chem-Bio Handbook* (London: Jane's Information Group, 1998)

United States Army Medical Research Institute of Chemical Defense. *Medical Management of Chemical Casualties Handbook*, Second Edition, April, 1995. Located at http://chemdef.apgea.army.mil/ChemCasu/titlepg.htm

Mitretek Systems. *Background on Chemical Warfare* located at http://ww.mitretek.org/mission/envene/chemical/chem_back.html.

Organization for the Prohibition of Chemical Weapons. *Chemical Warfare Agents: An Overview of Chemicals Defined as Chemical Weapons* located at http://www.opcw.nl/chemhaz/cwagents.htm