Chemical Terrorism Fact Sheet

Blood Agents - Cyanides:
Hydrogen cyanide and Cyanogen chloride

Protective Equipment/Detection

Several analytical methods are available for detecting **HCN** and **CK**, with the main method of laboratory detection (including mobile field vehicles) being gas chromatography / mass-spectrometry. Military-style gas masks with filters containing silver oxide offer effective protection. [Note: Filters should be changed immediately after exposure.]

Decontamination

The foremost consideration during decontamination is removal of the victim from continued exposure. For skin contamination, washing with soap and water is advised. Decontamination of clothing or equipment is unnecessary due to **HCN's** high volatility.

Signs and Symptoms

HCN's toxicity is due to its inhibition of cytochrome oxidase, resulting in interference with aerobic respiration. Lactic acid accumulates and cells die from histotoxic anoxia. Cyanide is also reported to alter calcium metabolism and increased intracellular calcium has also been suggested as a cause of cell death.

At low concentrations the victim may sense apprehension; experience dyspnea, headache and vertigo; and notice a metallic taste. Convulsions and coma can follow and may last for hours or days depending on the duration of exposure. If coma is prolonged, there may be residual damage to the CNS, manifested by irrationality, altered reflexes and unsteady gait, which can last for several weeks or longer. Temporary or permanent nerve deafness has also been described. In mild cases there may be headache, vertigo and nausea for several hours before complete recovery.

At high concentrations, victims notice a sensation of throat constriction, giddiness, confusion and decreased vision. A vice-like gripping of the temples and pain in the back of the neck and chest may occur. Unconsciousness follows and the individual falls. By this time, continued exposure can be fatal within 2-3 minutes, preceded by brief convulsions and respiratory failure. At lower but still lethal concentrations, the severity of symptoms may increase over an hour or more. Victims notice an immediate and progressive sense of warmth (due to vasodilatation) with visible flushing. Prostration follows, with nausea, vomiting, headache, dyspnea, and chest tightness. Unconsciousness and asphyxia will follow if exposure continues. At very high concentrations, hyperventilation is the main initial symptom, followed by loss of consciousness, convulsions, and loss of corneal reflex, with death caused by cardiac and/or respiratory arrest.

After exposure to lethal amounts via ingestion or skin exposure, the effects are slower to develop. For example, after ingestion of a lethal dose of a cyanide salt, the casualty might have 15 to 30 minutes of survival time during which an antidote could be administered.

Cyanide has a bitter almond odor.
Photo courtesy of The Almond Board of California.

Chemical Overview

Cyanides have long been known as poisons that inhibit aerobic respiration at the cellular level, preventing cells from utilizing oxygen. Although most cyanides are present as salts (sodium, potassium and calcium), two cyanides – **hydrogen cyanide (HCN)** and **cyanogen chloride (CK)** – are of military, and thus terrorist, interest.

HCN is also known as **hydrocyanic acid (AC)**. At atmospheric pressure, over the temperature range of -14°C to +26°C, **HCN** is a colorless to yellowish-brown liquid. It is completely soluble and stable in water. It has an aroma of bitter almonds or marzipan, but 25-50% of people cannot smell it at all. The major route of toxicity is inhalation and its action is rapid. The gas cannot pass through the skin but the liquid can be absorbed, even when aerosolized. Exposure to 60 mg•min/m^3 may cause no serious symptoms. At 200 mg•min/m^3 death occurs after 10 minutes and above 2,500 - 5,000 mg•min/m^3, death is likely within a minute.

Cyanogen chloride (CK) is heavier and less volatile than **HCN** and has a cumulative effect on its victims. **CK** is produced by chlorinating a saturated solution of potassium cyanide at 0°C. Compared to **HCN**, it is more effective at low concentrations, it irritates the eyes and lungs, and it has a delayed toxic effect similar to lung irritants like phosgene. At high concentrations, **CK** rapidly paralyzes the respiratory system's nerve center. **CK** is a colorless liquid at atmospheric pressure over the temperature range -6°C to +14°C. The main route of exposure is by inhalation, but it is also highly toxic through eye and skin contact or when absorbed from the gut. **CK** causes irritation in man after exposure to 2.5 mg•min/m^3 for 10 minutes, while 10 minutes of exposure at 5 mg•min/m^3, or 1 minute at 50 mg•min/m^3, is intolerable. Exposure to 120 mg•min/m^3 for 30 minutes or to 400 mg•min/m^3 for 10 minutes is fatal.

Signs and Symptoms (Continued)

CK exposure produces both the symptoms of cyanide poisoning noted above, and the symptoms of lung irritants. Contact with the eyes produces lacrimation and blepharospasm, while exposure of the respiratory tract produces irritation of the nose and throat, cough, dyspnea, chest tightness, and ultimately, pulmonary edema (with concentrations of **CK** between 50 and 300 mg•min/m^3). Slowed breathing will be followed by collapse, coma and death. Liquid **cyanogen chloride** can cause second and third degree burns following skin contact.

Useful lab findings include: high anion gap metabolic acidosis, and elevated lactate, methemoglobin, and urinary thiocyanate levels.

Treatment

Victims who are asymptomatic several minutes after exposure require no oxygen or antidotes. However, with victims showing acute effects (convulsions, dyspnea) or with those recovering from acute exposure (unconscious but breathing), administer oxygen and antidotes immediately. Antidotes work by dissociating the cyanide ion from cytochrome oxidase. Therapies include sodium thiosulfate (to increase rhodanase activity); sodium nitrite, amyl nitrite, or 4-dimethylaminophenol-hydrochloride (DMAP) (to form methemoglobin, which in turn combines with cyanide to form cyanmethemoglobin); or cobalt (which also combines with cyanide ions) in the forms of dicobalt edetate or hydroxocobalamin.

In adults, sodium nitrite, 10 mL of a 3% solution (300 mg), is given intravenously over a period of 3 minutes. For children, the dose is 0.33 mL of the 10% solution per kilogram of body weight. Amyl nitrite is only effective when used in a closed respiratory system, and little is actually absorbed from a capsule that is broken for inhalation through the mouth or nose. DMAP, slowly injected IV at a dose of 250 mg, has proven very effective. With DMAP, muscular necrosis may follow IM injection, so that route should be avoided. These drugs are life saving, but not curative. Following their administration, sodium thiosulfate, [Adults: 12.5 grams IV (50 mL of a 50% solution) / Children: 1.65 mL/kg of 25% solution, over 10 minutes] must be given to aid clearance of the methemoglobin. [NOTE: The Pasadena (formerly Lilly) Cyanide Antidote Kit contains amyl nitrite, sodium nitrite, and sodium thiosulfate.]

Hydroxocobalamin (vitamin B12a) binds with cyanide to form cyanocobalamin (vitamin B12). It is only given in large doses, intravenously (4 g IV over 30 minutes in adults). Dicobalt edetate, 600mg given intravenously (40 ml of a 1.5% solution in glucose/water solution), is also effective, but should be followed by IV sodium thiosulfate. Cobalt edetate is toxic to the kidney and may cause hypotension.

With **cyanogen chloride**, treat any irritation, as well as the pulmonary symptoms. Eyes may be washed with a weak boric acid solution. Injured skin may respond to soothing lotions, such as calamine, or cool compresses. For severe skin injury, assess and treat as for burns. Hypoxia may be controlled by O_2 supplementation, and the early use of positive airway pressure intermittent positive pressure breathing (IPPB), a positive end-expiratory pressure (PEEP) mask or, if necessary, intubation (with or without a ventilator) may delay and/or minimize the pulmonary edema and reduce the degree of hypoxia. Although the effectiveness of steroids in this chemically induced pulmonary edema is not proven, they are still advised if they can be given within 15 minutes of exposure.

Resources permitting, resuscitation should be attempted on subjects with no pulse, in case cardiac arrest is recent.

Additional information and references available at http.www.bioterrorism.slu.edu

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A Division of Saint Louis University School of Public Health

Long-term Medical Sequelae

Low concentration exposures produce no long-term medical problems. At near lethal concentrations, the effects on cellular respiration can be detrimental to CNS function and result in deterioration in intellect, confusion, loss of concentration and Parkinsonism. An ataxic neuropathy can be seen in victims of chronic cyanide poisoning. Specific data on developmental and reproductive effects, mutagenicity or carcinogenicity are not available.

Environmental Sequelae

HCN is highly volatile and is removed from the environment in less than an hour. **CK** is even less persistent.

Disclaimer

Information contained in this fact sheet was current as of September 2002, and was designed for educational purposes only. Medication information should always be researched and verified before initiation of patient treatment.

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