
Chemical Terrorism Fact Sheet

Blood Gas Agents - Hydrogen Sulfide

Protective Equipment/Detection

HS is easily detected by its distinctive odor, and a number of chemical processes are available to detect it as well. Positive-pressure, self-contained breathing apparatus (SCBA) and chemical-protective clothing are recommended in response situations with potentially unsafe levels.

Decontamination

The foremost consideration during decontamination is removal of the victim from continued exposure and the immediate administration of 100% O₂. For skin contamination, washing with soap and water is advised. While **HS**, as a gas, will not directly contaminate clothing or equipment, it can combine with water to form sulfuric acid which can contaminate such materials and water is an effective decontaminant.

Signs and Symptoms

The spectrum of illness depends on the concentration and duration of exposure. Low-level exposures (< 40 ppm for less than 15 minutes) are most commonly seen in industrial settings and can produce local eye and mucous membrane irritation, headache, fatigue, somnolence, loss of appetite, irritability, poor memory, dizziness, asthenia, and a chemical bronchitis with repeated exposure. On exam, conjunctivitis and wheezing may be evident, while long-term exposures can produce a gray-green line on the gingiva. Ocular irritation can occur at exposures as low as 4 ppm, while pulmonary membrane irritation will typically be seen at exposures > 20 ppm. Higher-level exposures (50-400 ppm) will produce cough, dyspnea, hemoptysis, cyanosis, agitation, vertigo, confusion, nausea and vomiting, tremulousness, cardiac arrhythmias, hypertension, and, possibly, loss of consciousness. Continued exposure will result in pulmonary edema and the victim may present in fulminate acute respiratory distress syndrome (ARDS). Severe high-level exposures (>500 ppm) rapidly produce fatal systemic toxicity leading to myocardial infarction, seizure, coma, and cardiopulmonary arrest, while just 2-3 breaths of **HS** at >700 ppm can cause immediate death.

Diagnostic studies reflect the systemic effects of toxicity, and are consistent with the findings of other hemoglobinopathies. Arterial blood gases typically reveal a marked uncompensated metabolic acidosis, but oxygen tension (pO₂) and calculated oxygen saturation are within reference range unless the victim has pulmonary edema. Measured oxygen saturation, however, is often low, indicating a saturation gap. Carboxyhemoglobin or methemoglobin levels may be elevated, depending on the source of **HS**. The chest x-ray will initially be normal, unless the patient presents with pulmonary edema. The electrocardiogram may reveal ischemia or infarction patterns, and various arrhythmias may be present. With long-term, low-level exposures, a CT or MRI scan of the head will often reveal basal ganglia lesions. Urinary thiosulfate levels can be used to confirm exposure, as can blood sulfide and thiosulfate levels, although these tests are not readily available.

Chemical Overview

Hydrogen sulfide (HS) -- H₂S -- is a colorless, flammable gas that is heavier than air and has the characteristic odor of rotten eggs at concentrations as low as 0.5 ppm. **HS** poisoning is rare because its odor alerts potential victims to the danger. However, concentrations greater than 100 ppm can cause olfactory fatigue and mislead individuals that the exposure risk has resolved. Toxic exposures most frequently occur in small closed spaces into which the victim enters unaware of the toxic build-up of the gas. **HS** poisoning is mainly encountered within the petroleum, viscose rayon, rubber, food processing, tanning and mining industries as well as in coke ovens and kraft paper mills. In nature, **HS** is produced by the organic decomposition of sulfur compounds in sewers, barns, ships' holds, and sulfur springs.

The major route of toxicity for **HS** is by inhalation. At lower doses, local irritant effects predominate. At higher exposures, cellular respiration may cease as **HS** forms a complex bond to the iron ion in mitochondrial cytochrome oxidase, arresting aerobic metabolism in an effect similar to cyanide toxicity and affecting all organs, particularly the nervous system. Sudden death can occur at concentrations >700 ppm. **HS** may also be absorbed from the gastrointestinal (GI) tract and skin, although these are rarely seen. GI absorption is typically seen in victims who ingest it after collapsing from the "knockdown" effect of an inhalation exposure.

The Occupational Safety and Health Administration (OSHA) has established an acceptable ceiling concentration of 20 ppm in the workplace, with a maximum level of 50 ppm allowed for 10 minutes if no other measurable exposure occurs. The National Institute of Occupational Safety and Health (NIOSH) recommends a maximum exposure level of 10 ppm.

Signs and Symptoms (Continued)

NOTE: Be aware of the environment in which the exposure has occurred since secondary findings may be present that are not directly related to the **HS** exposure. As an example, a sewage worker who collapses from a **HS** inhalation may also present with sewage aspiration that could result in infectious disease and immunological problems that outweigh the **HS** toxicity itself.

Treatment

Treatment of **HS** poisoning is based on the creation of methemoglobinemia, and rapid identification of **HS** as the toxin and treatment are essential for recovery. Initial treatment should consist of the administration of 100% oxygen, along with amyl nitrite therapy until more definitive therapy with sodium nitrite is available. Sodium nitrite is the drug of choice and is administered at a dose of 0.33 cc/kg of 3% solution, via slow IV push, to a maximum of 10 cc. Pediatric dosing is the same and the only contraindication to its use is known hypersensitivity to the drug. Caution should be taken in patients with poor underlying cardiopulmonary reserves since high methemoglobin levels may exacerbate ischemia in these people. In severe anemia, the dose should be adjusted as detailed in the package insert.

Hypoxia may be controlled by oxygen 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.

Aerosolized bronchodilators should be administered for acute bronchospasm, with consideration of the health of the myocardium in choosing which type of bronchodilator should be used.

Consider racemic epinephrine aerosol for children who develop stridor. A dose of 0.25–0.75 mL of 2.25% racemic epinephrine solution in water, repeated every 20 minutes as needed, should be used.

Anecdotally, hyperbaric oxygen (HBO) therapy is beneficial and should be considered for patients who are unresponsive to intravenous nitrites or who are showing delayed neuropsychiatric signs. Should you encounter a mass casualty scenario involving **HS**, it would be prudent to alert the nearest HBO facility to the potential need of their services.

Children may be more vulnerable to **HS** gas than adults because of their smaller airway diameters, their increased minute ventilation per kg, their greater lung surface area to body weight ratio, their inability to evacuate an area promptly when exposed, and their short stature, when higher concentrations of the chemical are found at low-lying areas.

Long-term Medical Sequelae

Delayed neuropsychiatric sequelae (vision and memory impairment; rigid movements; reduced motor function; slight tremor; ataxia; psychosis; abnormal learning, retention, and motor function; and slight cerebral atrophy) have been reported with chronic sub-lethal exposures, as well as with higher-concentration exposures in which the victim lost consciousness. **HS** has not been shown to cause cancer in humans, and has not been classified for carcinogenicity, mutagenicity or teratogenicity.

Environmental Sequelae

Hydrogen sulfide is released primarily as a gas and will spread in the air, where it will form sulfur dioxide and sulfuric acid. Sulfur dioxide can be broken down further and is a major component in acid rain. **HS** remains in the atmosphere for about 18 hours.

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.

Additional information and references available at <http://www.bioterrorism.slu.edu>