**Hydrogen Sulfide**
**September 2003**

## Introduction

Hydrogen sulfide, $H_2S$, is a naturally occurring compound that is produced by the organic decomposition of sulfur compounds in sewers, barns, ships' holds, and sulfur springs. It holds a spot on federal authorities' danger list because it is easily produced and highly toxic, and it has the potential to produce panic and social disruption.

## Hydrogen Sulfide – Overview

It is a colorless, highly irritating and flammable gas that is heavier than air and has a military designation of HS. Most people have encountered HS at least once in their lifetimes and would easily recognize its characteristic rotten egg odor which is detected at very low levels, 0.5 parts per million. HS is readily soluble in water where it combines with the water molecule to form sulfuric acid. The gas has numerous commercial and industrial applications and is commonly encountered in the petroleum, viscose rayon, rubber, food processing, tanning and mining industries as well as in coke ovens and kraft paper mills.

## Hydrogen Sulfide – Toxicity (1)

The major route of toxicity for HS is by inhalation. At lower doses, local irritant effects predominate, mainly of the eye and upper respiratory tract. 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. This process affects all organs, but particularly the nervous system. HS may also be absorbed from the gastrointestinal tract and skin, although this is rarely seen. GI absorption is typically seen in victims who ingest HS after collapsing from the "knockdown" effect of an inhalation exposure.

## Hydrogen Sulfide – Toxicity (2)

Because HS is heavier than air, children are likely to be more vulnerable to its affects. This is because children have smaller airway diameters, increased minute ventilation per kg, and a greater lung surface area to body weight ratio as compared to adults. In addition, inexperience and immaturity limit their ability to evacuate an area promptly when exposed, and their short stature brings them into contact with the higher gas concentrations found at low-lying areas.

Most acute poisonings occur in industry from the accidental formation of the gas in a small enclosed area. Both the Occupational Health and Safety Administration and National Institute of Occupational Safety and Health provide safety guidelines for this gas. OSHA has established an acceptable ceiling concentration of 20 parts per million in the workplace, based upon an 8-hour workday and 40-hour workweek. Their short-term exposure limit is a maximum level of 50 parts per million allowed for 10 minutes if no other measurable exposure occurs. NIOSH, with the stricter limits of the two, recommends a maximum exposure level of 10 parts per million. To put these limits into perspective, sudden death can occur with just a few breaths of the gas at levels greater than 700 parts per million.

**Protective Equipment**

While hydrogen sulfide's distinctive odor makes it easy to detect and avoid, olfactory fatigue is known to develop rapidly at levels over 100 parts per million. As a result, protective gear is advised. Because skin absorption is possible, chemical protective clothing is recommended. In response situations with potentially unsafe levels, breathing protection is required. Either a positive-pressure, self-contained breathing apparatus (SCBA) or a supplied-air respirator with a full facepiece will be protective.

**Detection**

Even though HS is easily detected by its distinctive odor, a number of chemical processes are available to detect it. Commercially available gas monitors, both portable units for personal use and fixed units for wide area use, can detect this gas in a range of 0 to 500 parts per million. The sensitivity of such units is most reliable in 5 parts per million increments, but can be adjusted to 1 part per million increments if necessary.

**Decontamination**

Since HS is a gas, the primary objective in decontamination is to move the victim from the area of exposure into fresh air. HS residue on the skin can be removed easily with soap and water. As a gas, hydrogen sulfide does not typically contaminate clothing or equipment. However, if it comes in contact with water, sulfuric acid will be formed and can become a separate contamination issue. Copious amounts of water will dilute and remove any sulfuric acid from such materials.

**Signs and Symptoms**

The spectrum of illness depends on the concentration and duration of exposure. Low-level exposures, defined as less than 40 parts per million for under 15 minutes, are the most common exposures seen in industrial settings. These low doses will produce local eye and mucous membrane irritation and can cause mild systemic effects such as headache, fatigue and somnolence, loss of appetite, irritability, poor memory, dizziness and asthenia. With repeated exposures, a chemical bronchitis can develop. 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 parts per million, while pulmonary membrane irritation will typically be seen at exposures greater than 20 parts per million.

**Signs and Symptoms**

Higher-level exposures, in a range from 50 to 400 parts per million, will produce more severe cardiopulmonary and systemic effects. Most commonly, cough, dyspnea, hemoptysis, cyanosis, agitation, vertigo, confusion, nausea and vomiting, tremulousness, cardiac arrhythmias, hypertension, and, sometimes, loss of consciousness are seen. Continued exposure will result in pulmonary edema and the victim may present in fulminate acute respiratory distress syndrome (ARDS). Severe high-level exposures, over 500 parts per million, rapidly produce fatal systemic toxicity leading to myocardial infarction, seizure, coma, and cardiopulmonary arrest. At levels over 700 parts per million, just 2 to 3 breaths of hydrogen sulfide can cause immediate death.

One other note of warning should be mentioned here. 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. For example, a sewage worker who collapses from HS inhalation may also present with sewage aspiration that could result in infectious disease and immunological problems that outweigh the HS toxicity itself.

**Laboratory**

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 ($pO_2$) and <u>calculated</u> oxygen saturation are within reference range, unless the victim has pulmonary edema. <u>Measured</u> oxygen saturation, however, is often low, indicating a saturation gap. Depending on the source of HS, carboxyhemoglobin or methemoglobin levels may be elevated. Initially, the chest x-ray will be normal, but will rapidly change to reflect the development of 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. Blood sulfide and thiosulfate levels can also confirm toxicity, but these tests are not readily available.

## Treatment

All victims should receive on-the-scene basic first aid. Beyond that, the treatment of hydrogen sulfide poisoning is based on the creation of methemoglobinemia, and rapid identification of HS as the toxin, along with prompt 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. Also, in severe anemia, the dose should be adjusted as detailed in the package insert. Monitor oxygen levels closely, and use oxygen supplementation to control and limit hypoxia.

Because of the increased risk of pulmonary edema and ARDS, consider 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) to delay and/or minimize any pulmonary edema and reduce the degree of hypoxia. Aerosolized bronchodilators should be administered for acute bronchospasm, with consideration of the health of the myocardium in choosing the type of bronchodilator to be used. For children with stridor, consider an epinephrine aerosol at a dose of 0.25–0.75 mL of 2.25% racemic epinephrine solution in water, repeated every 20 minutes as needed. Anecdotally, hyperbaric oxygen 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 hyperbaric facility to the potential need of their services. Finally, 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.

## Long-term Medical Sequelae

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

## Environmental Sequelae

Hydrogen sulfide, as a gas, will dissipate 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.

**Summary**

In summary, hydrogen sulfide has potential for terrorist use because it is easily produced, highly toxic, and an agent capable of producing panic and disruption. As a gas, its major route of exposure is by inhalation, and its toxic effects are those of a hemoglobinopathy, with disruption of cellular respiration. Low-level exposures, under 40 parts per million, cause mainly eye and mucous membrane irritation, while higher exposures, in the range of 50 to 400 parts per million, produce escalating cardiopulmonary and CNS effects including pulmonary edema, myocardial infarct and death. Treatment is based on forming methemoglobinemia, with sodium nitrite as the antidote of choice.