HYDROGEN SULFIDE

Synonyms
H_2S, hydrosulfuric acid, sulfur hydride, sulfane, stink damp. Sour gas (natural gas containing > 10 ppm hydrogen sulfide). Sewer gas (may also contain methane, ammonia, carbon dioxide and other gases).

Description
Hydrogen sulfide (H_2S) is a colourless gas, heavier than air, with a characteristic odour of rotten eggs. H_2S occurs naturally in fossil fuel deposits and sulfurous rock; hazard in mines, caves, wells, oil fields and petroleum refineries. Hot tar/asphalt gives off H_2S (**see** TAR AND ASPHALT). H_2S is released by decomposition of sulfur-containing organic materials; may be present in sewers, liquid manure storage tanks, and cargo holds of fishing boats. Many industrial processes release H_2S, including pulp paper and sugar production. Compounds including calcium polysulfide, sodium sulfide and ammonium sulfide release H_2S upon contact with water and acids.

Toxicity
Toxicity is dependent on concentration and duration of exposure. Most deaths occur at the scene and are secondary to respiratory paralysis (“knockdown”). Victims may also sustain trauma from falls.

Mechanism of Toxicity
Causes cellular hypoxia by inhibiting cytochrome oxidase. Resulting anaerobic metabolism produces lactic acidosis. High concentrations may cause direct paralysis of respiratory centres. Irritant effects produce local injury to eyes and respiratory tract.

Toxic Dose
Highly toxic. Odour is an unreliable predictor of danger; prolonged exposure to low concentrations or brief exposure to high concentrations results in olfactory fatigue. ACGIH TLV-TWA (threshold limit value-time weighted average) is 10 ppm.
Concentrations of 50-150 ppm are irritating; prolonged exposure (several hours) to 250 ppm can cause pulmonary edema; > 500 ppm may produce severe toxicity within minutes; and a single breath of concentrations > 800-1000 ppm may be rapidly fatal.

Case Reports
Eight sewer workers were found collapsed, unconscious and cyanosed. Four died at the scene, the remaining 4 (ages 23, 26, 36 and 42 years) received oxygen by face mask and arrived at hospital 2 hours after exposure, where they received diazepam for agitation and 300 mg sodium nitrite IV. All 4 patients had keratoconjunctivitis, respiratory distress, pulmonary edema, pulmonary wedge pressure > 18 mmHg, blood pressure > 110/80 mmHg, and elevated CK-MB and lactic dehydrogenase levels. The 26-year-old presented with aspiration pneumonia and required mechanical ventilation; his initial ECG showed evidence of myocardial infarction, and fatal cardiac arrest occurred at 36 hours. The 36-year-old had a cardiac arrest at 6 hours but was resuscitated and discharged with a temporary pacemaker. The 42-year-old suffered a fatal myocardial infarction 2 months after exposure (no history of cardiovascular disease). The 23-year-old was discharged after 7 days with no sequelae.
A 16-year-old and an adult collapsed and lost consciousness while cleaning inside a large fish tank. Emergency crews administered oxygen and the adolescent regained consciousness; the adult died at the scene. On admission to emergency, the adolescent developed respiratory distress, hypoxia (O_2 sats 88%), and was intubated and ventilated. His ECG was suggestive of inferior myocardial ischemia. On day 4 his chest X-ray showed bilateral hilar infiltrates, and by day 10 he was begun on corticosteroids for pulmonary edema. His blood gases improved and he was extubated on day 13. Over the following week, his oxygen requirement was reduced to room air, and he was discharged to a rehabilitation facility on day 22. He recovered without sequelae.
A 33-year-old inhaled vapours while bending over a tank for several minutes. On standing, he experienced breathing difficulty and collapsed for several seconds. Chest X-ray was normal, ECG showed T-wave inversion (which returned to normal within 5 months). Patient experienced increased fatigue, somnolence, shortness of breath, anxiety and "feeling vacant" for approximately 5 weeks after the incident.

Pharmacokinetics
Rapidly absorbed via lungs and mucous membranes. Oxidized to thiosulfate or sulfate by hemoglobin and liver enzymes. Also undergoes methylation reactions and interacts with proteins. Excreted in urine; small amounts of unchanged gas may be excreted by lungs.

Clinical Effects
- **General:** Inhalation of *high concentrations* can cause immediate respiratory paralysis, resulting in rapid loss of consciousness ("knockdown") and death due to asphyxia. Patients ventilated immediately after rescue often recover fully. Continuous exposure to *moderate-high concentrations* for > one hour may result in severe ocular and respiratory tract irritation and pulmonary edema. *Long term exposure* to concentrations < 50 ppm may result in mucous membrane irritation and cognitive impairment in some patients (limited data).

- **Topical:** Possible burning, itching, pain, redness of skin.

- **Ocular:** Vapour concentrations < 50 ppm may cause irritation. Exposure to > 50 ppm for 1 hour or more causes conjunctivitis ("gas eye") with pain, lacrimation, blurred vision, photophobia; severe cases may progress to corneal ulceration, keratoconjunctivitis.

- **Inhalation:**
  Acute: *Exposure to high concentrations producing rapid onset of severe symptoms:*
  Respiratory: Immediate respiratory paralysis, resulting in rapid loss of consciousness ("knockdown") and death due to asphyxia. Patients rescued alive have severe hypoxia and cyanosis. Respiratory tract irritation is usually absent in those who collapse rapidly (e.g. after 1 breath), but occurs if gas was inhaled for several minutes prior to respiratory paralysis (**see Subacute**). Aspiration pneumonitis; pulmonary edema may develop.
  CVS: Tachycardia (sometimes bradycardia), cardiac dysrhythmias, myocardial ischemia or infarction, hypotension (hypertension has also been reported).
  Neurologic: Patients ventilated *immediately* after rescue often recover fully; those who remain unconscious for longer periods are at risk for permanent hypoxic brain injury, neuropsychiatric sequelae or death. Convulsions have been reported.
  Fluids/Lytes/Acid-Base: Elevated lactate, metabolic acidosis (generally transient). Sulfhemoglobin levels are generally not significant.

  Subacute: *Continuous exposure to moderate-high concentrations for up to several hours:*
  Respiratory: Respiratory tract irritation (may be severe), bronchitis, cough, chest tightness, hemoptysis, increased pulmonary secretions; possible pulmonary edema (onset delayed up to 72 hours), pneumonia (bacterial infection); possible death due to respiratory failure (may be delayed hours to days).
  CVS: Possible myocardial ischemia (anginal chest pain).
  Neurologic: Dizziness, confusion, agitation, weakness, headache (may be rapid onset or delayed); loss of consciousness, convulsions may occur.
  GI: Nausea, vomiting, diarrhea, "rotten egg" breath or belching.

  Chronic: *Long term exposure to concentrations < 50 ppm:* Controversial; existence of true "chronic" toxicity not confirmed; observed effects may be secondary to single or multiple acute or subacute exposures, or to personal subject variability.
  Fatigue, headache, irritability, chronic bronchitis, nasal and ocular irritation, ataxia, cognitive impairment have been reported by some authors.

Treatment

General: Prompt rescue and treatment is required. Rescue of an unconscious victim exposed to hydrogen sulfide gas should only be performed by personnel equipped with respiratory protection, protective clothing, and devices that can quickly measure hydrogen sulfide air concentration.

1. **Topical:** Flush skin thoroughly with soap and water.

2. **Ocular:** Flush eyes with a gentle stream of tepid water for at least 15 minutes. Obtain ophthalmologic opinion if irritation persists. Keratoconjunctivitis is treated symptomatically.

3. **Inhalation:** Remove from exposure. Rescuers must wear self-contained breathing apparatus. Once victims are removed from immediate area of exposure they do not pose a risk to rescuers.

4. *Primary treatment* is oxygenation and ventilation; many patients respond to this treatment alone. Oxygen enhances detoxification of sulfide. Give 100% humidified oxygen by tight-fitting mask or mechanical ventilation (as indicated). Patients with respiratory paralysis may not start breathing spontaneously for minutes to hours after removal from exposure. Aspiration and pulmonary edema are common in severe cases.

5. *Asymptomatic* patients should be observed at least 2 hours. *Symptomatic* patients should be observed and monitored for delayed pulmonary edema for up to 2-3 days.

6. Monitor vital signs. ECG, chest X-ray, blood gases (request measured pO_2), electrolytes, serum lactate should be monitored in symptomatic patients. If nitrite therapy is used, monitor methemoglobin levels.

7. Maintain fluid and electrolyte balance. Treat severe metabolic acidosis with IV sodium bicarbonate.

8. Hypotension unresponsive to fluids may require vasopressors.

9. Agitation and seizures may be treated with IV benzodiazepines.

10. Pulmonary edema should be treated with supportive care.

11. **Sodium nitrite:** Use is controversial. Limited animal and human data suggest benefit; place in therapy not clearly established. Early use of a single dose may be considered for *markedly symptomatic* patients after ventilation and oxygen have been provided. Do **NOT** give to *asymptomatic* or *minimally symptomatic* patients (i.e., nausea, dizziness, drowsiness, coughing) or those with a short-lived symptomatic episode (including brief loss of consciousness) with spontaneous recovery (i.e., no residual CNS depression or vital sign abnormalities by the time patient reaches hospital).
    Converts hemoglobin to methemoglobin, which binds hydrogen sulfide to form sulfmethemoglobin. Other mechanisms may also be involved.
    Caution can cause rapid onset hypotension. Do *not* exceed methemoglobin level of 25%. Avoid in G6PD deficiency (may cause hemolysis), pregnancy, or patients suspected of concurrent carbon monoxide exposure (e.g. smoke inhalation).
    See SODIUM NITRITE antidote monograph. Note: sodium nitrite is available in the Cyanide Antidote

Package. Other components of this package (sodium thiosulfate, amyl nitrite) have *not* been shown to be of use in hydrogen sulfide poisoning.

12. ***Hyperbaric oxygen*** may be of value in patients not responding to ventilation and supportive therapy; case reports indicate positive results.

Key Points

- ✓ Inhalation of *high concentrations* can cause immediate respiratory paralysis, resulting in rapid loss of consciousness ("knockdown") and death due to asphyxia.
- ✓ Patients ventilated *immediately* after rescue often recover fully.
- ✓ Those who remain unconscious for longer periods are at risk for permanent hypoxic brain injury.
- ✓ Severe ocular and respiratory tract irritation may be seen in those exposed to *moderate-high concentrations* for > 1 hour.
- ✓ Treatment is primarily supportive: 100% oxygen, ventilation as required, and irrigation of eyes.
- ✓ Sodium nitrite IV may be of value in *markedly symptomatic* patients only (anecdotal evidence only).
- ✓ *Asymptomatic* patients should be observed at least 2 hours.
- ✓ *Symptomatic* patients should be observed and monitored for delayed pulmonary edema for up to 2-3 days.