Update in Prevention, Diagnosis and Treatment of Hydrogen Sulfide Poisoning
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Recent Manure Pit Event

- 48 yo Mennonite dairy farmer, 18 and 14 yo sons died in 20’ deep[ 2 million gallon storage tank.
- Working on 180 cow dairy
- Father had multiple injuries thought to be from auger used to circulate manure and thought that son’s went in to rescue him.
- All three found in manure asphyxiated
- Conveyor attached to tractor. Apparently working on auger. Tractor and pickup running
- Went out that AM to pump manure out to spread on fields and never returned
- Baltimore Sun May 25, 2012
Case report

- Two brothers cleaning out manure tanker in August 2006 in Dane County
- Both collapsed but one was able to climb out
- Transported to UW-Madison
- One brother unconscious for 2 days, other discharged the next day
- Unresponsive, metabolic acidosis, mild pulmonary edema
- Seizure activity, asymmetric spasticity
- No hyperbaric oxygen or sodium nitrate/amyl nitrate given as he improved after 2 hours of ventilation
- Treated with 10cm PEEP, 100% O2, benzodiazepines, dilantin
- Recovered fully 2 months later
- Amnestic for event
Conditions at time of rescue

- Warm, foggy, with 1.5” rain in the AM
- Temp 76 deg. F
- Wind speed 3-5 mph
- Relative humidity 88-90%
- Malodorous, estimated heat index interior of 100 deg. F
- Solid manure, 6” deep 2” wide front to back
Tanker design

- Confined space by definition
- Only light and ventilation from top opening
- Rear discharge open but blocked with no light
Rescue Issues

- Victims had been working ~ 20 “
- Victim #2 unable to rescue Victim #1 but able to crawl out and flag down wife driving by and 911 called
- Victim #1 head down 0.5-1” above manure head in downward slop
- Unresponsive and foaming at the mouth when rescuer arrived
Rescue Issues

- EMT first responder (neighbor drove over directly) had no personal protective equipment but held breath and made 2 attempts
- Spent 4-5” in tanker later had headache and lightheadedness
- Other help (neighbor and victims father) arrived and then could pull him out.
- Deputy sheriff arrived, applied oxygen and then EMTs arrived.
- Estimated 20 minutes in tanker
Rescue Issues to consider

- All turned out well but:
- Rescue attempted with no personal protective equipment.
- No lines or safety harnesses available
- Others health and safety jeopardized in the heat of the moment
- How many lives should be put at risk to safe others?
Manure tanker
An example of a confined space

Courtesy William Field, Purdue
Summer Conditions
Winter Conditions
Under-barn manure storage
In some conditions, this has caused fatalities
Gases In Hog Confinement

Figure 23.1. Swine confinement building.

ORGANIC DUST EXPOSURES

Organic Dust Toxic Syndrome
Manure storage environment

- $\text{H}_2\text{S}$, ammonia
- Methane
- Carbon dioxide
- Oxygen deficient
- Lack of air monitoring of $\text{H}_2\text{S}$ in most agricultural settings
- Lack of knowledge of sublethal exposures or long term chronic exposures
Common Issues in Gases

- Worse in winter
- Inadequate ventilation increases concentration
- Agitation increases levels
- Dust masks not adequate
- High concentrations require SCBA
  - H2S, CO,
Occupational health concerns

• Concentrations are higher during hot humid summer days
  ✷ May have previously entered during other weather conditions with no effects
• Increased generation of H₂S during summer conditions
• Less dispersal by winds
Occupational health concerns

- Lethal concentrations raised with agitation
  - Pumping out of manure storage
- Levels of 1500 ppm can be reached
- Manure levels should be at least 12” below surface
- All animals and humans should be out of building or adjacent area during pumping of manure slurry and for a day afterwards
Manure pit fatality review

- 77 fatalities and 21 severe injuries identified between 1975 and 2004
- 34% occurred in those conducting repair or maintenance on manure handling equipment
- 21% in those performing rescue of another person
- Over 50% involved dairy operations
- Peak period during hottest part of summer
  - Beaver and Field. 2007 J Agromed 12(2)
Manure-related incidents

• Swiss series (Knoblauch et al 1999)
  ♦ 61 accidents from 1951-1995 with 105 individuals (43 rescuers)
  ♦ 44 accidents from inhalation of manure gas
  ♦ 49 fatalities
  ♦ 12 (24%) of those were rescuers
  ♦ 1/3 of those attempting to rescue died
  ♦ 13 incidents, 7 fatalities occurred in immediate vicinity of pit, not in pit
ASPHYXIANT GASES

- CARBON MONOXIDE
  - Odorless, colorless
  - Heaters, internal combustion engines, pressure washers
  - Headache, dizziness, chest pain, coma
  - Fetuses at greater risk
  - PEL  35 ppm/ 8 hours
Other asphyxiant gases

- **CARBON DIOXIDE**
  - More of an indirect indicator

- **METHANE**
  - Less dense than air
  - Dizziness at high concentrations
  - Most concern as an explosive
  - TLV 1000 ppm/ 8 hours
Mechanisms of exposure

- Under barn manure storage pits
- Agitation of slurry releases bubbles of $\text{H}_2\text{S}$
- Entering manure pits to repair equipment
- Falling into manure pits
Draeger (Colorimetric) Tubes
Electronic direct reading gas meters-multiple gases and re-usable
Properly attired rescue personnel
Rescue issues in recently filled silos or manure pits

- Continuous monitoring of oxygen and nitrogen oxides
- SCBA
- Safety harness
- Hospitalize those with hypoxemia, dyspnea, or altered consciousness for minimum of 24 hours
AMMONIA

- Pungent, irritant
- Lighter than air
- Low odor threshold-5 ppm
- TLV 25 ppm but adverse effects from exposure over 7 ppm on chronic basis
- Tolerance develops
- Irritation eyes, nose, throat, chest
- Associated with sinusitis, bronchitis
ASPHYXIANT GASES

• HYDROGEN SULFIDE
  - Chemical asphyxiant similar to cyanide
  - Denser than air
  - Rotten egg smell at 0.13 ppm
  - Paralyzes olfactory nerve above 150 ppm
  - PEL-10 ppm/ 8 hour day
  - 500 ppm unconsciousness and death
  - Up to 1500 ppm with agitation
ASPHYXIANT GASES

- HYDROGEN SULFIDE
  - Irritant at lower levels
  - Persistent neurologic effects after exposure to high levels and unconsciousness for longer than 5 minutes
  - Pulmonary edema over 250 ppm
  - Evacuate if over 50 ppm
H₂S Toxicology

- Primary determinants of toxicity are concentration dependent, not duration
- Primary toxic effects are neurological
Toxicology of H₂S

- Colorless
- Heavier than air
- Systemic absorption with inhalation
- Low odor threshold
- Potent inhibitor of cytochrome oxidase interrupting oxidative phosphorylation
  - Leads to cellular hypoxia and anaerobic metabolism
  - Binds to ferric moiety of cytochrome a3 oxidase complex with higher affinity than cyanide
- Forms sodium sulfide when reacts with water on skin or mucous membranes
Physiologic effects of $\text{H}_2\text{S}$ concentrations

- 0.01-0.3 ppm odor threshold
- 3-10 ppm unpleasant odor
- 20-30 ppm strong offensive odor
- 20-50 ppm conjunctival irritation
- 50-100 ppm irritation of respiratory tract

- 100-150 ppm olfactory paralysis
- 250 ppm pulmonary edema
- 500 + ppm knockdown
- 500-1000 ppm respiratory paralysis
  - Guidotti, 1994
  - Reffenstein 1992
Pulmonary effects of H$_2$S

- Tissues most sensitive to H$_2$S are mucous membranes and those with high oxygen demands
- Respiratory and nervous systems are the primary target organs although proposed mechanism of death is paralysis of respiratory center
Pulmonary effects of higher concentrations

- Dyspnea, chest pain, cough
- Pulmonary edema at 250 ppm
- Paralysis of respiratory center
- Sequellae may include pneumonia
- Reactive airways dysfunction syndrome (RADS) not known to occur
Respiratory symptoms resulting from knockdown

- Alberta oil and gas workers respiratory symptoms and “knockdown”
- Excess of wheeze, dyspnea on exertion
- No measurable pulmonary health effects in either group
Pulmonary sequellae of H₂S exposure

- Generally no long term pulmonary effects
- Recovery is generally good if victim can be adequately medically supported
- May have chronic CNS effects after several knockdowns
  - cognitive dysfunction
  - affective disorders
Neurologic effects

• Neuropsychiatric-memory failure, lack of insight, disorientation, delirium
• Neurosensory-transient hearing loss, vision loss, anosmia
• Motor-basal ganglia injury
   ataxia, position/intention tremor, muscle rigidity
• Neuropathological-subcortical white matter demyelination
Treatment of manure pit (H₂S) toxicity

• Maintain oxygenation, treat acidosis, control seizures with benzodiazepines (Micromedex)
• Ventilatory support with PEEP in face of pulmonary edema providing 100% O₂
• In those with suspected hydrogen sulfide poisoning and with altered mental status, coma, hypotension, or dysrhythmias should received sodium nitrite by slow infusion at same dose given for cyanide poisoning.
Three elements of therapy

1. Promote competitive inhibition of sulfide-cytochrome binding: theoretical effect of nitrate therapy. Sulfide bound to nitrite-induced methemoglobin.

2. Promote permanent detoxification of sulfide by oxidization to sulfates and sulfur.

3. Minimize hypoxic tissue damage by supportive care
Potential benefits of HBOT

- Tissue oxygen concentration elevated by HBOT.
- HBO might enhance detoxification of sulfide by maximizing oxyhemoglobin levels.
- HBO can improve oxygen diffusion through liquid and oxygenation in the presence of pulmonary edema.
Diagnosis of H$_2$S poisoning

- History of exposure such as found in a confined space with contaminated or decomposing organic material nearby
- Smell on clothing/body
- CXR, ABGs, serum lactate, monitor for cardiac arrhythmias, myocardial infarction, electrolyte disturbance, pulmonary edema, CNS damage
- Sulfide and thiosulfates are not available for acute exposure clinical decision making but may be helpful in confirming exposure if drawn readily.
- Whole blood sulfides $> 0.05$ mg/L
- Normal urine thiosulfate $< 8$ mg/L, normally undetectable
- serum thiosulfate $> 1.3$ associated with toxicity
- Only urine may be elevated in survivors in acute exposure
- Must be obtained within 2 hrs of exposure and analyzed immediately.
  - Goldfrank’s 8th Ed. 2006
Hyperbaric Oxygen therapy (HBOT) in H₂S toxicity

- No studies evaluating role of HBO for preventing delayed neurologic sequelae are available
- No reason to transfer for HBOT therapy alone as efficacy not established by any clinical outcome studies
- HBOT when other RX, especially 100% O₂, intubation, nitrite treatment, and supportive measures have failed and when neurologic, cardiovascular, and acid-base abnormalities persist
  - After adequate initial supportive care but early in presentation..

Efficacy of treatment

- Animal studies indicate HBOT + nitrate therapy shows better survival compared to:
  - HBOT alone;
  - nitrates alone;
  - supportive care only alone
Nitrate Therapy
Follows cyanide protocols

- Amyl nitrate (on scene or during transport)
  - one ampule on 15 s and off 15s until IV sodium nitrite (HCP or EMT should not breath it)
- IV sodium nitrite 300 mg (10 ml of 3% solution) at 2.5-5 ml/min
- Repeat at half dose if symptoms of toxicity reappear
- After IV infusion start 12.5 g (50 ml of 50% solution) sodium thiosulfate IV
- Can repeat in hrs at half dose of initial.
- Children
  - 6-8 ml/m2 (~0.2 ml/kg) of 3% sodium nitrite not to exceed 10 ml or 300 mg.
  - 0.2 ml/kg dose of 3% sodium nitrite(6 mg/kg sodium nitrite) would be safe in child with Hb of 7.
  - Repeat as above
Additional therapeutic points

- Nitrate therapy can result in severe methemoglobinemia if anemia present.
- Monitor methemoglobin when nitrate therapy instituted
- Generally those that survive will have minimal residual effects but ongoing neuropsychometric pathology, and rarely pulmonary fibrosis, can occur.