Confined-space accidents on the farm: the manure pit and the silo

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ABSTRACT: Manure pits and silos are potentially dangerous, gas-containing confined spaces on the farm that may generate hazardous levels of hydrogen sulphide and nitrogen dioxide respectively. Although these inhalation injuries are uncommon, emergency physicians who work within transport distance of rural areas should be aware of the toxic agents involved and their acute management. This article reviews the toxicology, clinical manifestations and treatment of acute, farm-related hydrogen sulphide and nitrogen dioxide exposures.

RÉSUMÉ ANALYTIQUE : Les fosses de fumier et les silos sur les fermes sont des espaces clos qui renferment des gaz potentiellement dangereux pouvant générer des niveaux toxiques d'hydrogène sulfuré et de dioxyde d'azote. Bien que ces blessures par inhalation soient peu courantes, les médecins qui travaillent près des zones rurales doivent se familiariser avec les agents toxiques et leur traitement actif. Le présent article fait un compte-rendu de la toxicologie, des manifestations cliniques et du traitement des expositions à l'hydrogène sulfuré et au dioxyde d'azote en milieu rural.

Key words: hydrogen sulphide, silo, nitrogen dioxide, farm accident

Introduction

The National Institute for Occupational Safety and Health (NIOSH) defines a confined space as a "space with limited openings for entry and exit, with unfavourable natural ventilation that could contain or produce dangerous air contaminants, and that is not intended for continuous worker occupancy."[1] Two such potentially dangerous gas-containing spaces exist on the farm: the manure pit, containing hydrogen sulphide, and the silo, containing nitrogen dioxide. Entry into these spaces may lead to multiple fatalities when coworkers, bystanders or family members die during rescue attempts.

Case report

A farmer, his son and two other workers were renovating a poorly ventilated section of a hog barn. They decided to blow liquid manure into this area to loosen up old manure adhering to the structure. During the process, hydrogen sulphide gas overcame the farmer and then his son, who was attempting rescue. A third person collapsed while attempting to help, but recovered and summoned help. Eventually, family members removed the victims from the barn and attempted to revive them. Sadly, both died later in hospital.[2]

Discussion

Although toxic hydrogen sulphide (H_2S) and nitrogen dioxide (NO_2) exposures are uncommon, emergency physicians who work within transportable distances from rural areas should be aware of these entities and be familiar with their acute management.

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The manure pit (hydrogen sulphide)

Dairy, beef, hog and poultry operations now use liquid manure systems as a fast and economical method of handling animal wastes. Animal excrement is collected for 4 to 6 months in manure holding lagoons outside the barn or in pits or tanks beneath it. In spring and fall, these tanks are agitated to suspend the solids that accumulate at the bottom, then the manure suspension is transferred into spreader tanks and applied to the fields as fertilizer.

Anaerobic decomposition of livestock and poultry manure gives rise to approximately 80 compounds, including hydrogen sulphide, methane, ammonia and carbon dioxide.[3] Hydrogen sulphide presents the major hazard, but hypoxia and the synergistic effects of other gases contribute to its toxic effects. During agitation and pumping, the period of highest risk, H_2S is released from liquid manure in the same manner that carbon dioxide is liberated after shaking a carbonated soft drink.[4,5] Hot, humid weather increases the danger by optimizing microbial metabolism and increasing gas production.[6]

Hydrogen sulphide is a clear, colourless, irritating gas that can be identified in relatively low concentrations by its characteristic "rotten-egg" odour. At higher concentrations, this warning disappears because of olfactory nerve fatigue and paralysis.[7] Hydrogen sulphide is heavier than air, therefore has a tendency to accumulate on the surface of the manure. Most sources agree that, like cyanide, H_2S binds reversibly to cytochrome oxidases and inhibits oxidative phosphorylation; however, in an recent review, Reiffenstein and colleagues[8] concluded that there must be additional nonmetabolic processes operating to fully explain hydrogen sulphide's toxic effects.

Hydrogen sulphide gas is an asphyxiant and an irritant, with direct inflammatory effects on ocular and respiratory mucous membranes. H_2S toxicity follows a consistent dose-response relationship,[9] causing keratoconjunctivitis at low levels[8] and lacrimation, mucopurulent discharge and corneal ulceration — the so-called "gas eye syndrome" — with prolonged exposure. At sub-lethal concentrations, cardiac, respiratory and neurologic effects appear. Cardiac manifestations include myocardial depression, conduction defects, repolarization abnormalities and dysrhythmias.[10] Respiratory effects include dyspnea, cough, chest pain, pulmonary edema, hypoxia, cyanosis and hemoptysis.[8,11,12] Neurological effects include vertigo, seizures and syncope,[7,8,11,12] but delayed neuropsychiatric sequelae have also been reported after acute exposure.[13,14] At high concentrations (> 1000 parts per million) victims rapidly develop systemic intoxication, coma, respiratory paralysis and death.[7,10] Drowning can occur when victims are overcome and fall into the manure.

The first priority in managing a hydrogen sulphide exposure is to remove the victim from the gas source while ensuring the safety of the rescuers. Rescuers must not enter the pit or exposure site without a self-contained breathing apparatus, a lifeline attachment, and adequate rescuer backup. If cardiac or respiratory arrest has occurred, advanced cardiac life support (ACLS) guidelines should be followed and, as with any unconscious or seriously poisoned patient, aggressive supportive care is the cornerstone of treatment. Transport to a tertiary care centre is required only if these resources are unavailable.

Ongoing management remains controversial and largely anecdotal. Because H_2S, like cyanide, binds reversibly to cytochrome oxidases, nitrites may be therapeutic;[15,16] however, some authors suggest they are only effective if given immediately at the exposure site.[17] Nitrites induce the formation of methemoglobin, which has a greater affinity than cytochrome oxidase for H_2S. Methemoglobin theoretically promotes the dissociation of sulphide from cytochrome, thereby facilitates the return of aerobic metabolism. Again, there may be other ill defined mechanisms for nitrite effectiveness.[9]

Table 1. Clinical features and management of hydrogen sulphide (H_2S) and nitrogen dioxide (NO_2) exposures

	H_2S (manure pit)	NO_2 (silo)
Signs and symptoms	Mucous membrane irritation	Laryngospasm, bronchospasm, asphyxia, pulmonary edema
	Dyspnea, cough, hemoptysis, chest pain, hypoxia, pulmonary edema, cyanosis	
	Myocardial depression, dysrhythmias	Bronchiolitis obliterans (late)
	Vertigo, seizures, and syncope	Methemoglobinemia
	Delayed neuropsychiatric sequelae	Neurological symptoms
	Coma, respiratory paralysis, death	Respiratory arrest, death
Management	Extrication/resuscitation/supportive	Same
	Amyl and sodium nitrite (cyanide kit)*†	Bronchodilators,† steroids†
	Hyperbaric oxygen therapy†	

*Do *not* administer thiosulfate.
†Unproven benefit

Recommended nitrite dosages are similar to those used in cyanide poisoning and are contained in the cyanide antidote kit. After adequate oxygenation has been achieved, an amyl nitrite perle is broken and inhaled for 30 seconds every minute until intravenous access is established. Then the adult dose of sodium nitrite, 300 mg (10 ml of 3% solution), is given intravenously over 4 minutes. Caution is warranted, however, since nitrites may cause hypotension, and because methemoglobin interferes with oxygen delivery, causing tissue hypoxia — especially concerning in already compromised patients.[11,18] When using the cyanide kit to treat H_2S exposures, it is important to remember that thiosulfate is not useful because the body spontaneously detoxifies sulfmethemoglobin.[10]

Hyperbaric oxygen (HBO) has also been advocated for the treatment of hydrogen sulphide exposure. Some case reports suggest benefit from HBO when given after a course of nitrite therapy[19–21] while others fail to show benefit.[22] In theory, HBO inhibits sulphide–cytochrome binding, promotes sulphide detoxification, and reduces post-exposure tissue injury;[20,23] however, there are insufficient data to conclude that HBO is beneficial in cases of hydrogen sulphide toxicity. Future research will address agents such as dithiothreitol, which is thought to actively remove sulphide from its cellular site of action.[24]

The silo (nitrogen dioxide)

Domed cylindrical silos, a common sight in any rural area, serve two important functions: crop storage and fermentation. Mature standing corn, oats and hay are cut and chopped, then blown into the silo to ferment in an anaerobic environment. Fermentation enhances nutritional content, thus increases the number of livestock that can be supported per crop acre. However, ensiled crops produce carbon dioxide and nitrogen oxides, which usually peak 48 to 72 hours after the silo is filled.[25] Dangerous levels of nitrogen dioxide persist for about 10 days but may persist for months on the surface of silage depressions in tightly sealed silos.[26] Workers may enter a freshly filled silo to set up for unloading or to cap the silo. "Capping" refers to the process of applying a plastic sheet over the top layer of silage to retard surface spoilage.

Nitrogen dioxide (NO_2) as an air pollutant is well described. In addition, military researchers have studied the effects of high levels of NO_2 encountered in combat situations.[27] Unfortunately, little research addresses farm-related nitrogen dioxide exposures. Nitrogen dioxide is heavier than air. It collects just above the silage and may flow down the silo chute into feed rooms and connected buildings. At low concentrations it has a bleach-like odour and a faint yellow colour, not always visible. At higher concentrations, it appears orange-brown and inhalation provokes an acrid, offensive sensation like that induced by chlorine or ammonia.[28] Nitrogen dioxide is relatively insoluble in water, therefore tends to bypass the upper airway without any warning irritation of the eyes or nasopharynx. But in the moist, mucoid environment of the lower respiratory tract, NO_2 dissolves, penetrates bronchiolar and alveolar membranes, generates free radicals, nitric and nitrous acid, and causes an acute lung injury known as "silo-filler's disease."[26]

Once absorbed, nitrite ions interact with vascular smooth muscle to cause vasodilatation, and react with hemoglobin to cause methemoglobinemia.[29,30] The spectrum of illness is variable and the response severity unpredictable. Most exposures probably go unreported and most reported exposures are mild and self-limited.[26] Patients destined to experience serious sequelae generally present early and are acutely ill at the time of presentation.[25] Massive NO_2 exposure can lead to death from bronchiolar or laryngeal spasm, respiratory arrest, or simple asphyxia,[26] but death may also result from exposure-induced falls.[28] The mortality rate of acute silo-filler's disease is from 9% to 20%.[25,28]

Survivors of high-level nitrogen dioxide exposures may experience a biphasic clinical response characterized by acute laryngospasm and bronchospasm, then the development of pulmonary edema after 8 to 24 hours.[31] Ten to 31 days later, following a period of apparent improvement, a recurrent cough and dyspnea may herald the onset of bronchiolitis obliterans.[26] Most such cases will resolve without permanent impairment, and late bronchiolitis obliterans with scarring and irreversible airflow obstruction is rare.[32]

The first priority in managing a nitrogen dioxide exposure is to remove the victim from the exposure site while ensuring the safety of the rescuers. Self-contained breathing equipment, lifelines and adequate rescuer numbers are essential. Subsequently, aggressive supportive therapy, including oxygen, cardiopulmonary resuscitation, intubation and mechanical ventilation should be instituted as required. Bronchodilators and corticosteroids are generally recommended[26,28,32] but have not undergone adequate study.

Summary

Although uncommon, hydrogen sulphide and nitrogen dioxide exposures occurring on the farm can cause devastating illness and multi-organ damage. The top priority is prompt removal of the victim(s) from the exposure site with adequate rescuer numbers, self-contained breathing equipment, and lifelines. Aggressive supportive care is important; adjunctive modalities are poorly studied but may be useful. Improved

awareness by rural emergency physicians is essential to optimize the acute care of these patients. Most importantly, these accidents are readily preventable by following established safety guidelines for entry into confined spaces.

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