

GASEOUS TOXICITIES



BASICS

OVERVIEW

- Gaseous toxicities in ruminants are often due to gases associated with animal housing and production; many of these toxic gases such as NH_3 , CO_2 , H_2S , and CH_4 are produced during decomposition of animal waste in anaerobic conditions.
- Other toxic gases include NO_2 , produced during the fermentation process of ensiled forages; sulfur oxides (such as SO_2 and SO_3), produced by industrial pollution; and CO , produced as a by-product of incomplete combustion of hydrocarbon fuels.
- Toxic gases may be lighter or heavier than air which plays an important role in the determination of exposure.

INCIDENCE/PREVALENCE

Occurs in animal housing and/or where feeds are stored. Mortality can be as high as 100%

GEOGRAPHIC DISTRIBUTION

Worldwide

SYSTEMS AFFECTED

Respiratory, nervous system

PATHOPHYSIOLOGY

The gases displace oxygen and the animal asphyxiates. Exposure to toxic gases is primarily by the respiratory, ocular and dermal route. See below Physical Examination Findings

SIGNALMENT

- Cattle, sheep, goats and camelids.
- No breed, sex or age predilections

PHYSICAL EXAMINATION FINDINGS

- Death, recumbency, arrhythmias, lacrimation, salivation, dyspnea, depression, ataxia, tremors, weakness
- NH_3 reacts with water to form ammonium hydroxide (NH_4OH) which is caustic, especially to the mucous membranes of the eye and respiratory tract (points of first contact). Tearing, shallow breathing and nasal discharge are common; at higher concentrations, laryngospasm and coughing may be seen. NH_3 also causes non-specific stress and reduces resistance to infections. Concentrations greater than 50-100 ppm can cause reduced growth rate and mucous membrane irritation. At >1000 ppm airway and alveolar damage occurs.

- CO₂ at high concentrations replaces oxygen from the environment leading to dyspnea and anoxia. Concentrations greater than 50,000 ppm (5%) may cause labored breathing and at concentrations greater than 400,000 ppm (40%), severe signs of anoxia including anxiety, staggering, coma and death from suffocation may ensue.
- CO competes with oxygen for binding sites on various proteins such as hemoglobin forming COHb, which it has a high affinity for (> 250 times that of oxygen). CO can also bind with the cytochrome oxidase system to reduce cellular utilization of oxygen leading to adverse effects and potentially hypoxia in tissues most sensitive to oxygen deprivation such as the brain and myocardium. CO causes drowsiness, lethargy, weakness and incoordination when blood concentrations of COHb reach 30% (normal < 1-3%). At > 60%, dyspnea, coma, terminal clonic spasms, and acute death may occur.
- H₂S binds to iron within the cytochrome oxidase enzymes inhibiting them and ultimately disrupting aerobic metabolism leading to anoxia and direct damage to brain cells. H₂S has a direct paralytic effect on the carotid body causing inhibiting of respiration. In addition, H₂S can combine with hemoglobin to form sulf-Hb, decreasing oxygen transport. At concentrations greater than 50-100 ppm in air, H₂S causes irritation of the eyes and lungs and at > 500-1000 ppm can be neurotoxic and directly paralyze the respiratory center, leading to hyperpnea, apnea and death.
- CH₄ displaces oxygen in an enclosed environment and can produce asphyxiation at concentrations > 85-90% (oxygen concentrations <15-16%). The primary danger is its explosive hazard.
- NO₂ combines with water to form nitric acid which is corrosive to the moist mucous membranes of the eye and respiratory tract. NO₂ at ambient concentrations of > 50 ppm can cause choking, coughing, and labored breathing. At > 50 ppm, bronchitis, pneumonia and edema of the lungs may occur.
- Sulfur oxides are readily converted to acids upon contact with water leading to a pronounced irritant effect on the mucosa of the nasopharynx, respiratory tract and eyes. Bronchoconstriction may also occur. At concentrations of > 10-50 ppm sulfur oxides can cause ocular irritation, salivation, laryngeal spasms and emphysema.

CAUSES AND RISK FACTORS

- Accumulation of high nitrogenous waste (NH₃).
- Heaters operated in poor ventilated areas (CO).
- Accumulation of protein/sulfur containing organic matter and agitation of slurry (H₂S, CO₂, NH₃, CH₄).
- Ensiled forages in silo produce NO₂ which can move into adjoining areas.
- Animals in enclosed, non-ventilated areas (CO₂ and CH₄).
- Industrial pollution (SO₂, SO₃).
- Low temperature, overcrowding of animals especially in closed structures, faulty heaters/furnaces, poor facility design, improper ventilation, and poor waste management.



DIAGNOSIS

DIFFERENTIAL DIAGNOSIS

- ABPE—onset of clinical signs usually 1–4 days after an abrupt change to lush pasture, pathological evaluation of lung
- Infectious respiratory diseases

- Nonprotein nitrogen toxicosis—exposure to NPN supplements, toxic levels of ammonia in rumen content and blood
- Organophosphorus or carbamate insecticide exposure—gastrointestinal irritation and neurological signs, evaluation of cholinesterase activity, detection of pesticides
- Cardiotoxic plants (oleander, milkweeds, and azaleas)—analysis for plant toxins in gastrointestinal contents, history of exposure

CBC/BIOCHEMISTRY/URINALYSIS

Changes are limited; chronic hypoxia can lead to increased PCV (increased Hgb).

OTHER LABORATORY TESTS

- Blood gases (pCO₂, pO₂)
- Blood color—bright red with CO poisoning
- COHb in blood (not routinely available)

IMAGING

N/A

OTHER DIAGNOSTIC PROCEDURES

History, field observation, and clinical signs; “rapid tests” provided by local fire department for analysis of toxic gases in the air.

- NH₃ is lighter than air and has a characteristic pungent odor detectable around 10 ppm.
- CO₂ is a colorless, odorless gas normally present in the air at concentrations around 400 ppm (0.04%); it is heavier than air.
- CO is a colorless, odorless gas present in fresh air at round 0.02 ppm but concentrations of around 40 ppm may be present in high traffic areas.
- H₂S is heavier than air and has a characteristic “rotten egg” smell. The odor threshold is 0.025-0.2 ppm but at higher concentrations (>150 ppm) it becomes undetectable due to the paralyzing effect it has on the olfactory sensory apparatus.
- CH₄ is a colorless, odorless gas. At concentrations >5-15% by volume, it is explosive.
- NO₂ is heavier than air, and has a pungent, sweetish, “bleach-like” odor (odor threshold 0.1-0.2 ppm). NO₂ is only visible as yellowish-brown gas when concentrations reach a dangerous level (75-150 ppm).
- Sulfur oxides such as SO₂ and SO₃ are gases present in industrial polluted air.

PATHOLOGIC FINDINGS

Gross lesions include

increased mucous secretions within the respiratory tract.

- Pulmonary congestion and edema.
- Bright red mucous membranes and pink coloration of tissues and non-pigmented skin (CO).
- Greenish discoloration of grey matter, viscera and bronchial secretions (H₂S).
- General congestion of tissue (result of hypoxia).

Histopathological lesions include

- Hyperplasia of bronchiolar and alveolar epithelium.
- Pulmonary congestion, edema, and emphysema.
- Necrosis in the brain and heart (result of hypoxia).



TREATMENT

THERAPEUTIC APPROACH

- Restore adequate oxygen supply
- Remove from exposure to toxic gas.
- Corticosteroids to alleviate pulmonary edema and clinical signs.
- Broad spectrum antibiotics to control secondary bacterial pathogens.
- Electrolyte and fluid therapy as needed.



MEDICATIONS

DRUGS OF CHOICE

- No antidote
- Broad spectrum antibiotics (secondary infections).

CONTRAINDICATIONS

- IV fluids should be administered carefully in animals with cardiovascular problems.



FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS

- Most often, animals exposed to high concentrations of toxic gases are found dead.
- In surviving animals, the prognosis is fair if there is prompt medical treatment of clinical signs and no further exposure.

POSSIBLE COMPLICATIONS

Secondary bacterial infections.

PATIENT CARE

- Monitor cardiac, pulmonary and neurologic function.
- Avoid excessive stress and exercise.

PREVENTION

- Provide good ventilation around the base of silo during the fermentation process so that gases will be carried away. In addition, animals should not be allowed in areas near the silo for at least 48 hours after filling.
- Allow at least one foot of space between the highest manure level and slats upon which animals are housed (prevents inhalation of gases that accumulate at surface of the pit).
- Provide as much ventilation of the pit and building during agitation of manure or waste as possible.



MISCELLANEOUS

PRODUCTION MANAGEMENT

Ensure adequate ventilation of all animal facilities.

ABBREVIATIONS

- ABPE = acute bovine pulmonary edema and emphysema
- CH₄ - methane
- CO - carbon monoxide
- COHb - carboxyhemoglobin
- CO₂ - carbon dioxide
- H₂S - hydrogen sulfide
- HCN - hydrogen cyanide
- IV = intravenous
- NH₃ – ammonia
- NO₂ - nitrogen dioxide
- NPN = nonprotein nitrogen
- ppm - parts per million
- SO₂ - Sulfur dioxide
- SO₃ - Sulfur trioxide

SEE ALSO

- Organophosphate Toxicity
- Cardiotoxic Plants
- Oleander and Cardiotoxic Plant Toxicity
- Milkweed (*Asclepias spp.*) Toxicosis

Suggested Reading

Hooser, S.B., Van Alstine W., Kiupe, M., Sojka J. Acute pit gas (hydrogen sulfide) poisoning in confinement cattle. JVDI 2000; 12(3): 272-5.

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