



Phosphine

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Introduction

Phosphine, PH_3 , also known as phosphane, hydrogen phosphide or phosphorus hydride, is the phosphorus analog to ammonia. It is a natural product of swamps and sewers as well as a product of combustion that contributes to air pollution.

Phosphine – Overview

Phosphine is a colorless gas that is heavier than air and may cause asphyxiation in enclosed, poorly ventilated, or low-lying areas. Pure phosphine is nearly odorless, but commercially available phosphine has an odor of garlic or decaying fish. However, the odor threshold of 0.15 parts per million may not provide adequate warning of hazardous concentrations. It is extremely flammable and explosive and may ignite spontaneously on contact with air. Upon burning, it produces a dense white cloud of phosphorus pentoxide, which is also a severe pulmonary irritant due to the rapid formation of orthophosphoric acid on contact with the moist respiratory tract.

Industrially, phosphine is produced during acetylene production by the action of water on calcium carbide which is commonly contaminated with calcium phosphide. Phosphine is used in the semiconductor industry as a fumigant and as a polymerization initiator. Its metallic salts, aluminum phosphide and zinc phosphide, are solids used as grain fumigants and rodenticides.

From the perspective of terrorist use, its toxicity is such that it can effectively disable a group of people, but death is unlikely. However, the extreme flammability of this gas limits its potential use by these groups since it cannot be reliably dispersed without the risk of ignition.

Phosphine – Toxicity

Most phosphine exposures occur by inhalation of the gas. As a gas, phosphine produces no adverse effects on the skin or eyes, and such contact does not result in systemic toxicity. Keep in mind, though, that frostbite may occur from contact with the liquefied or compressed gas. The ingestion of phosphine is also unlikely because it is a gas at room temperature. However, the ingestion of metallic phosphides can also cause systemic effects. Upon contact with gastric acid these phosphides produce phosphine gas, which is systemically absorbed through the GI tract. It is also known that prolonged dermal exposure to the metallic phosphides can lead to systemic effects, although this route of exposure is less likely to cause major toxicity.

It must also be noted that children are at higher risk for toxicity because of their greater lung surface area to body weight ratio, increased minute volume to weight ratio, and shorter stature, which places them in higher concentrations of the gas since phosphine is heavier than air.

Phosphine – Toxicity (2)

OSHA's permissible exposure limit, averaged over an 8-hour work shift, is 0.3 parts per million. The NIOSH IDLH, the level considered immediately dangerous to life or health, is 50 parts per million. A level of 0.5 parts per million has been set as the maximum airborne concentration below which one can be exposed for up to 1 hour without experiencing or developing serious adverse health effects that could impair the individual's ability to take protective action.

Protective Equipment

Use of positive-pressure, self-contained breathing apparatus (SCBA) is advised in situations that involve exposure to potentially unsafe levels of phosphine. Chemical-protective clothing is not generally required because the gas is not absorbed through the skin, and skin irritation is unlikely. However, use rubber gloves and aprons with victims exposed to phosphides.

Detection

The detection of phosphine cannot be based upon its artificially instilled odor, as olfactory fatigue occurs rapidly. There are a number of commercially available gas sensors and monitors which can be used to detect phosphine.

Decontamination

In order to decontaminate, remove exposed individuals from the contaminated area as soon as possible. Remove and double-bag contaminated clothing and personal belongings. For eye exposure, flush the eyes with cool water for at least 15 minutes. For skin exposure, especially to the metallic phosphides, brush all visible particles from the skin and hair, flush with water for 5 to 10 minutes, and

then wash well with soap and water, followed by a thorough rinsing. Use caution to avoid hypothermia in children and the elderly.

Persons exposed only to phosphine gas do not pose substantial risks of secondary contamination. On the other hand, metallic phosphides on clothes, skin, or hair can off-gas phosphine after contact with water or moisture, so a risk of secondary contamination may be present. Vomitus containing phosphides can also off-gas phosphine.

Signs and Symptoms

The diagnosis of phosphine exposure is mainly a clinical one confirmed by the detection of the gas at the scene. Phosphine is a respiratory tract irritant that attacks primarily the cardiovascular and respiratory systems causing peripheral vascular collapse, cardiac arrest and failure, and pulmonary edema. Phosphine acts by interfering with enzymes and protein synthesis, primarily in the mitochondria of heart and lung cells. In the heart, metabolic changes cause excitation disturbances that alter transmembrane potentials, and laboratory tests may reveal abnormal myocardial enzymes. Its clinical manifestations may include hypotension, reduced cardiac output, tachycardia, and irregular heart beat. Phosphine also affects the small peripheral vessels, causing a profound decrease in systemic vascular resistance that can lead to severe hypotension that does not respond well to pressor agents. Ultimately, cardiac arrest and peripheral vascular collapse can occur. Most deaths occur within 12 to 24 hours after exposure and are cardiovascular in nature. If the patient survives the initial 24 hours, the ECG typically returns to normal, indicating that heart damage is reversible.

Signs and Symptoms

Respiratory toxicity is characterized by chest tightness, a cough productive of fluorescent green sputum, and shortness of breath. Severe exposure can cause pulmonary edema, which may have a delayed onset of up to 72 hours after exposure. Pulmonary edema and pneumonitis are believed to result from direct cytotoxicity to the pulmonary cells.

Signs and Symptoms

Phosphine also produces serious central nervous system (CNS), gastrointestinal (GI), and renal effects. Phosphine is a CNS depressant, producing headache, restlessness, dizziness, loss of feeling, impaired gait, trembling of the extremities during movement, and double vision upon initial exposure. Severe exposure can cause seizures and coma.

Signs and Symptoms

Gastrointestinal symptoms are usually the first to occur after exposure and may include nausea, vomiting, abdominal pain, and diarrhea. Liver damage can also be prominent, although liver injury does not usually become evident until 48 to 72 hours after exposure. Jaundice, hepatomegaly, elevated serum transaminases, and increased serum bilirubin may be evident, and centrilobular necrosis has been reported. Liver failure is the prime cause of death after 24 hours. In addition, phosphine has systemic renal effects, producing hematuria and proteinuria, as well as acute kidney failure.

Signs and Symptoms

Laboratory findings may include blood gas analysis revealing a combined respiratory and metabolic acidosis. In addition, expect to see abnormal cardiac isoenzymes, liver enzymes, and urine findings. There have also been reports of significant hypomagnesemia and hypermagnesemia associated with massive focal myocardial damage.

Signs and Symptoms

Chronic exposure to very low concentrations over several days may result in anemia, bronchitis, nausea and other gastrointestinal disturbances, inflammation of the nasal cavity and throat, weakness, dizziness, jaundice and other liver effects, increased bone density, and visual, speech, and motor disturbances. Chronic exposure may be more serious for children because of their potential longer latency period.

Treatment

At the scene, basic first aid and ventilatory support should be administered to all victims. There is no antidote for phosphine poisoning and treatment consists of support of respiratory and cardiovascular functions. Steroids have no proven efficacy. Hemodialysis is recommended only if renal failure develops, and the effectiveness of exchange transfusions is questionable.

Follow the ABCs of evaluating and supporting the airway, breathing, and circulation. Patients who are comatose, hypotensive, or seizing should be provided supportive care with intravenous fluids, pressor agents, sodium bicarbonate, or anticonvulsants as indicated.

Treatment

For adult victims in shock or with severe hypotension, defined as a systolic pressure under 80 mmHg, give a 1,000 mL bolus of intravenous saline or lactated Ringer's solution over one hour, followed by an infusion at 150 to 200 mL/hour.

If the systolic pressure is over 90 mmHg, begin fluid infusion without the initial bolus. For children, administer a 20 mL/kg bolus of normal saline over 10 to 20 minutes, followed by an infusion at 2 to 3 mL/kg/hour. For significant acidosis, administer sodium bicarbonate intravenously (adult dose = 1 ampule; pediatric dose = 1 mEq/kg), with additional therapy guided by arterial blood gas measurements.

Treatment

Symptomatic patients should receive supplemental oxygen for dyspnea and/or hypoxia, and all victims should be observed for at least 72 hours with repeated chest examinations and other appropriate studies. 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) may delay and/or minimize the 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 which type of bronchodilator should be used.

For children who develop stridor, consider racemic epinephrine aerosol. A dose of 0.25–0.75 mL of 2.25% racemic epinephrine solution in water, repeated every 20 minutes as needed, should be used.

Treatment

If phosphides have been ingested, it is important to remove them as quickly as possible from the GI tract, but do not induce emesis. Gastric lavage with a potassium permanganate solution (1:10,000) is recommended. Permanganate oxidizes phosphine in the stomach to form phosphate, thus reducing the available phosphine. Follow lavage with a slurry of activated charcoal at 1 gm/kg. The usual adult dose is 60 to 90 gm and for children, 25 to 50 gm. A mineral oil cathartic (100 mL) is recommended rather than a saline cathartic.

Routine laboratory studies for all victims include a CBC, basic metabolic panel, renal function tests, liver-function tests and serial cardiac isoenzymes. Diagnostic studies include ECG, chest radiography, pulse oximetry, blood gases, and PF13.

Long-term Medical Sequelae

Most survivors of acute phosphine exposure show no permanent disabilities. Secondary sequelae, such as myocardial infarct or stroke, are possible, however, when there has been insufficient blood supply to the heart and brain. Subacute poisoning, resulting from exposure over a few days, has been reported to cause reactive airways dysfunction syndrome months later. The EPA has determined

that phosphine is not classifiable (Group D) as to its carcinogenicity and no teratogenic effects are known.

Environmental Sequelae

From an environmental perspective, phosphine in air is changed to less harmful chemicals in less than a day. Phosphine is also removed from the air by contact with moist soil which promotes oxidation to orthophosphate. Zinc phosphide disappears from soils of 50% or more water content in less than 5 weeks, and the majority is recoverable as orthophosphate.

Summary

In summary, phosphine's use by terrorists would be primarily one of disruption and fear, with minimal mortalities. Phosphine, as a gas, is a pulmonary irritant that systemically affects the cardiovascular and respiratory systems. In addition, metallic phosphides can also produce systemic effects when ingested. The clinical manifestations of exposure may include severe hypotension that does not respond well to pressors, pulmonary edema, reduced cardiac output, tachycardia, and irregular heart beat, potentially leading to cardiac arrest and peripheral vascular collapse. There is no specific treatment and care is primarily supportive, yet most survivors show no permanent disabilities.