



Fluorine / Hydrogen Fluoride September 2003

Fluorine and Hydrogen Fluoride – Introduction

Fluorine is a gaseous element, designated as 'F' on the periodic table, that readily combines with hydrogen to create hydrogen fluoride gas. Rarely found in nature, it is used in the manufacturing of fluorochemicals, glass, enamel, bricks and plastics, and as a rocket propellant. Hydrogen fluoride gas is used to make aluminum and chlorofluorocarbons (CFCs), as a cracking catalyst in oil refineries, and for etching glass and enamel, removing rust, and cleaning brass and crystal. It also is used in manufacturing silicon semiconductor chips and as a laboratory reagent.

Fluorine – Overview

As a gas, fluorine has a yellow-green color and highly irritating odor, which might be confused with the more commonly used chlorine gas by inexperienced individuals. It has a low odor threshold, 0.035 to 0.14 ppm, and is extremely corrosive, being the most highly oxidizing element known. It is heavier than air and will settle to low-lying areas. It readily dissolves in water, forming the weak hydrofluoric acid. Although not flammable, it supports combustion and may react explosively with many substances. Fluorine combines directly or indirectly to form fluorides with all of the elements except helium, neon, argon, and, under ordinary conditions, oxygen or oxides of sodium, potassium or calcium. In contact with organic compounds, it reacts violently, usually disintegrating the molecule. In industry it is available as a compressed liquid.

Hydrogen Fluoride – Overview

Hydrogen fluoride (HF), on the other hand, is a colorless, fuming liquid or gas with a strong, irritating odor that is discernable at concentrations of 0.04 ppm. Like elemental fluorine, HF readily dissolves in water to form colorless

hydrofluoric acid, which, in dilute solutions, is visibly indistinguishable from water. Although hydrofluoric acid is comparatively weak, because of the fluoride ion's aggressive, destructive tissue penetration it can produce serious health effects by any route of exposure. It is lighter than air and is available industrially as a compressed gas.

Toxicity

Both gases are classified as pulmonary irritant agents and cause severe eye, mucous membrane, and skin irritation. OSHA's permissible exposure limit to fluorine, averaged over an 8-hour work shift, is 0.1 ppm (0.2 mg/m³) and the short-term (15 minute) exposure limit is 2 ppm. The NIOSH IDHL, the level immediately dangerous to health and life, is 25 ppm/ 5 minutes and is based on the lowest published toxic concentration for fluorine inhalation in man. A plasma level 3 mg/L from any route of exposure is fatal. For HF, OSHA limits an 8-hour workday, 40-hour workweek to 2.5 mg/m³ air. The NIOSH IDHL for hydrogen fluoride is 30 ppm. The estimated lethal dose of HF is 5 to 10 gm (32-64 mg/kg) in adults and 500 mg in small children. In addition to the irritant effects, exposure to the liquid forms can produce frostbite injury and burns. Since fluorine is heavier than air, children and others of short stature may be at higher risk. Also, children are at higher risk in general with gas exposures because of their greater lung surface area to body weight ratio, increased minute volume to weight ratio, and larger surface area to body weight ratio.

Toxicity

Fluorine and hydrogen fluoride's toxic effects are due primarily to the fluoride ion, which aggressively penetrates tissues and binds intracellular calcium and magnesium. This results in cell destruction, local bone demineralization, and systemic hypocalcemia, hypomagnesemia, and hyperkalemia. Hypocalcemia disrupts oxidative phosphorylation, glycolysis, coagulation, and neurotransmission, and causes tetany, cardiotoxicity, and cardiovascular collapse. Hypocalcemia becomes a risk in all instances of inhalation or ingestion and whenever skin burns exceed 25 square inches, an area roughly the size of the palm. Fluoride also inhibits acetylcholinesterase. Severe fluoride toxicity produces multi-organ failure, and death results from respiratory paralysis, dysrhythmias, or cardiac failure.

Protective Equipment

Positive-pressure, self-contained breathing apparatus (SCBA) is recommended and chemical protective clothing that is specifically resistant to fluorides should be worn, although it may provide little or no thermal protection. Structural firefighters' protective clothing is recommended for fire situations only; it is not effective in spill situations. Fully encapsulating, vapor protective clothing should be worn for spills and leaks with no fire.

Detection

Although highly irritating, fluorine's noxious odor can't be relied upon for detection and the inexperienced may misidentify it as chlorine. In addition, hydrofluoric acid is visually indistinguishable from plain water and can cause serious toxicity if ingested. Reliable detection must depend upon commercially available monitors and gas sensors. Fortunately there are a variety of such monitors for both wide area and personal use.

Decontamination

Rapid decontamination is critical. Victims should be removed from the contaminated area immediately. Thaw clothing frozen to the skin before removing. Otherwise, quickly remove and double-bag contaminated clothing while flushing exposed skin and hair with plain water or saline for at least 30 minutes. Cover exposed skin with a calcium-containing slurry or gel of 2.5 g of calcium gluconate in 100 mL of a water-soluble lubricant, such as K-Y Jelly, or 1 ampule of 10% calcium gluconate per ounce of K-Y Jelly. Exposed eyes should be flushed immediately with water for 15 minutes, and then continuously irrigated with normal saline during transport to the hospital, using an ophthalmic anesthetic, such as 0.5% tetracaine, as necessary to alleviate blepharospasm.

Signs and Symptoms

The adverse action of the fluoride ion may progress for several days before symptoms appear. As previously stated, hypocalcemia can disrupt oxidative phosphorylation, glycolysis, coagulation, and neurotransmission; and cause tetany; cardiotoxicity; and cardiovascular collapse. Hyperkalemia may cause ventricular fibrillation. Also, the inhibition of acetylcholinesterase will result in hypersalivation, vomiting, and diarrhea. The patient may also display seizures as the result of hypomagnesemia and/or hypocalcemia. Other systemic symptoms include coma, hypotension without a compensatory tachycardia, acidosis, paresthesias, and coagulation disturbances.

Signs and Symptoms

Acute exposure to fluorine produces eye, nasopharyngeal, skin, and respiratory irritation, followed by coughing, choking and chills that persist 1 to 2 hours after exposure. In severe exposures, there may be an asymptomatic period of 12 to 48 hours, after which fever, cough, cyanosis, rales, and tightness in the chest develop, before progressing to pulmonary edema. These symptoms intensify over 48 hours and then regress slowly over 2 to 4 weeks.

Inhaled hydrogen fluoride mist or vapor affects the nose, throat, and eyes initially, with mucous membrane irritation and inflammation, cough, and narrowing of the bronchi. In more severe exposures, upper airway obstruction can arise from a rapid narrowing and swelling of the throat. Lung injury may evolve rapidly or may be delayed up to 36 hours. As the injury progresses, pulmonary edema, bronchiolar constriction, and partial or complete lung collapse can occur. Pulmonary effects result from dermal exposure and ingestion as well as inhalation.

Signs and Symptoms

Although inhalation injury is the most dangerous and produces the most immediate symptoms, dermal and eye contact, and ingestions also result in toxicity. Depending on the concentration and duration of exposure, skin contact may produce pain, redness of the skin, and deep, slow-healing burns secondary to the formation of hydrofluoric acid on the skin. Acid concentrations of more than 50%, including anhydrous hydrogen fluoride, cause immediate severe, throbbing pain and a whitish discoloration of the skin, which usually evolves into blisters. HF solutions from 20% to 50% may produce pain and swelling, which may be delayed up to 8 hours, while solutions of less than 20% cause almost no pain on contact but may produce serious injury 12 to 24 hours later.

Eye exposure also results in the formation of hydrofluoric acid in the conjunctival sac. While mild exposure rapidly produces eye irritation, more severe effects include sloughing of the surface of the eye, swelling of various structures of the eye, and ischemic cell death. Potentially permanent corneal clouding may develop.

Finally, toxicity can occur from an overdose ingestion of fluorides, which react with saliva and gastric fluids to form hydrofluoric acid. Small ingestions produce local gastrointestinal upset, salivation and a metallic taste that may last 48 hours. More serious ingestions result in corrosive injury to the mouth, throat, and esophagus, inflammation of the stomach with bleeding, nausea, vomiting, diarrhea, and abdominal pain, as well as systemic symptoms.

Treatment

After decontamination, follow the ABC's of advanced life support. Establish intravenous access and administer glucose in saline vigorously to maintain a high urine output. Anticipate seizures and treat as indicated. Monitor electrolytes, calcium, and magnesium hourly and correct as necessary, using 10% calcium gluconate IV for tetany or dysrhythmias related to hypocalcemia. Treat

hypomagnesemia with 2 to 4 mL of 50% of magnesium sulfate IV over 40 minutes.

Securing and maintaining an open airway is critical in victims showing pulmonary symptoms. If the victim is not breathing, start artificial respiration using a demand-valve resuscitator, bag-valve-mask device, or pocket mask as trained. Do not initiate mouth-to-mouth resuscitation, which can lead to secondary exposure to the rescuer. Watch for signs of respiratory insufficiency and consider assisted ventilation at the first indication of trouble. For hypoxia, administer oxygen by a non-rebreather mask at 10 to 15 L/min and monitor the patient's oxygenation status with arterial blood gases or pulse oximetry. Also observe closely for signs of pulmonary edema, which may be delayed, and treat accordingly. The early use of positive airway pressure intermittent positive pressure breathing (IPPB), a positive end-expiratory pressure (PEEP) mask or, intubation (with or without a ventilator) may delay and/or minimize pulmonary edema and reduce the degree of hypoxia.

Treatment

In addition to securing and maintaining the airway and ventilation, other aspects of pulmonary care must be considered. The use 2.5% calcium gluconate (2.5 g of calcium gluconate in 100 mL of water or 25 mL of 10% calcium gluconate diluted to 100 mL with water) administered by nebulizer with oxygen has been recommended, but the success of this therapy is not proven. If the victim displays acute bronchospasm, administer aerosolized bronchodilators, taking into consideration the health of the myocardium in choosing the type of bronchodilator to be used. Fluoride poisoning is not known to add additional risk during the use of bronchial or cardiac sensitizing agents. In children, consider epinephrine aerosol for those who develop stridor. A dose of 0.25 to 75 mL of 2.25% racemic epinephrine solution in water, repeated every 20 minutes as needed, should be used.

For ingestions, do not induce vomiting or administer activated charcoal. Rinse the mouth well and administer 5 mL/kg, up to 200 mL, of water or milk for dilution if the patient can safely swallow, has a strong gag reflex, and does not drool. If available, give a one-time dose of 2 to 4 ounces of an antacid containing magnesium (Maalox, milk of magnesia) or calcium (Tums). If less than 1 hour after exposure, consider gastric lavage with lime water (0.15% calcium hydroxide solution) or other Ca^{+2} salts that will precipitate the fluoride. Due to the corrosive effects of hydrofluoric acid on the esophagus, take great care in placing the naso- or orogastric tube for use in the lavage.

Treatment

A burn specialist or plastic surgeon should be consulted early in the treatment of fluoride burns. Initial care of the burned area includes the use of a calcium

gluconate gel (2.5 grams in 100 mL water-soluble lubricant, such as K-Y Jelly) until the pain is relieved. If used as definitive treatment, the gel should be applied 4 to 6 times daily for 3 to 4 days. Large or deeply penetrating burns may require injections of sterile aqueous calcium gluconate into and around the burned area. The recommended dose is to inject up to 0.5 mL of 10% calcium gluconate solution per cm² of affected skin surface using a small-gauge needle. Intra-arterial calcium gluconate (10 mL of 10% calcium gluconate diluted with 40 mL D₅W, over 4 hours) is effective for the treatment of burned digits and upper extremities. The radial artery is used, unless there is incomplete anastomotic flow between the radial and ulnar circulations in such cases use the brachial artery. If pain is unrelieved, try a 20% concentration. Once the patient has been pain-free for 4 hours, the arterial catheter can be removed. Do not inject or use calcium chloride, as it can cause extreme pain and additional injury.

For ocular burns, immediately consult an ophthalmologist. Do not use oils, salves, or ointments for injured eyes. Do not use the gel form of calcium gluconate in eyes, as described for skin treatment.

Long-term Medical Sequelae

All routes of exposure can lead to long-term problems for the victim of fluorine exposure. Chronic lung disease may result from severe inhalation exposure, and skin burns can take months to heal with the potential for extensive scarring. Fingertip injuries with chronic pain, bone loss, and nail-bed injury can be debilitating. With eye contact, prolonged or permanent visual defects, blindness, or complete eye destruction can occur. Finally, ingestion may damage the esophagus and stomach progressively for weeks, leaving a persistent narrowing of the esophagus.

The EPA has determined that fluorine and HF are not classifiable (Group D) as to their carcinogenicity, and their reproductive and teratogenic effects are not known.

Environmental Sequelae

Fluorine is so highly reactive that it does not persist in the environment but will rapidly form fluoride salts in soil and dissolve in water, forming hydrofluoric acid, oxygen fluoride, hydrogen peroxide, oxygen and ozone.

Summary

Fluorine and hydrogen fluoride are toxic gases classed as pulmonary irritants because of their severe eye, mucous membrane, and skin effects. Fluorine and hydrogen fluoride's toxic effects are due primarily to the fluoride ion, which aggressively penetrates tissues and binds intracellular calcium and magnesium and results in cell destruction, local bone demineralization, and systemic

hypocalcemia, hypomagnesemia, and hyperkalemia. Hypocalcemia, in turn, disrupts oxidative phosphorylation, glycolysis, coagulation, and neurotransmission, and causes tetany, cardiotoxicity, and cardiovascular collapse. Treatment is largely supportive with an emphasis on pulmonary care. Securing and maintaining the airway is critical. In addition, the early use of positive airway pressure intermittent positive pressure breathing (IPPB), a positive end-expiratory pressure (PEEP) mask or, intubation (with or without a ventilator) may delay and/or minimize pulmonary edema and reduce the degree of hypoxia. Unfortunately, long-term medical problems are often seen following fluorine and fluoride poisoning.