Inorganic Acids and Related Compounds

SUBSTANCE IDENTIFICATION
Found as crystalline solids or colorless to yellow liquids. Some products may fume in air. Products range from odorless to strongly irritating odors. Used in batteries, as cleaners, in many chemical reactions, and in numerous manufacturing processes. Products are highly corrosive to tissue and metals. Reactions with most metals may generate hydrogen gas. Reaction with water may generate heat (exothermic reaction) or in the case of sulfuric acid, violent reaction and spattering.

ROUTES OF EXPOSURE
Skin and eye contact
Inhalation
Ingestion

TARGET ORGANS
Primary
Skin
Eyes
Respiratory system
Gastrointestinal system
Metabolism
Secondary
Central nervous system
Cardiovascular system
Renal
Blood

LIFE THREAT
Pulmonary edema, bronchospasm, circulatory collapse, laryngeal spasm and edema, GI tract perforation, hemorrhage, and peritonitis. Corrosive to skin, mucous membranes, and internal organs.

SIGNS AND SYMPTOMS BY SYSTEM
Cardiovascular: Hypovolemic shock, tachycardia with weak pulse, and circulatory collapse.
Respiratory: Coughing, chest pain, sneezing, rhinitis, dyspnea, bronchospasm, asphyxia, chemical pneumonitis, acute pulmonary fibrosis, reactive airways disease syndrome (RADS), acute pulmonary edema, upper airway obstruction from laryngeal spasm or glottic edema with stridor and pain, and aspiration pneumonia.
CNS: Symptoms of hypoxia, weakness, dizziness, stupor, lethargy, and coma.
Gastrointestinal: Acute toxicity results in burns to the mouth, esophagus, stomach, and lower GI tract. Nausea, vomiting, dysphagia, and diarrhea, possibly hemorrhagic.
Eye: Conjunctivitis, spasm of eyelids (blepharospasm), photophobia, opacification of the corneas, corneal perforation, and blindness.
Skin: Irritant dermatitis, hypersensitivity, and skin discoloration, full- and partial-thickness burns with coagulation necrosis, eschar formation, and scarring. Concomitant exothermic reaction may produce thermal burns.
Renal: Kidney damage.
Metabolism: Metabolic acidosis and hyponatremia.
Blood: Hemolysis, coagulopathy.
Other: Refer to guideline 16 for hydrofluoric acid and related compounds.

**SYMPTOM ONSET FOR ACUTE EXPOSURE**
Immediate
Respiratory symptoms may be delayed
Water solubility determines whether initial symptoms are directed at upper or lower airways; highly water-soluble products affect the upper airway; less water-soluble agents are more likely to cause lower airway symptoms; systemic effects may be seen.

**CO-EXPOSURE CONCERNS**
Other acids
Alkalis (exothermic reactions possible)

**THERMAL DECOMPOSITION PRODUCTS INCLUDE**
Arsenic: Arsenic acid
Bromine: Hydrobromic acid
Chromium: Chromic acid
Hydrogen chloride/hydrogen/chlorine: Hydrochloric and hypochlorous acid
Nitrogen oxides: Nitric and nitrous acid
Sulfur oxides: Sulfuric and sulfurous acid

**MEDICAL CONDITIONS POSSIBLY AGGRAVATED BY EXPOSURE**
Respiratory disorders
Skin disorders

**DECONTAMINATION**
- Wear positive-pressure SCBA and protective equipment specified by references such as the DOT Emergency Response Guidebook or the CANUTEC Initial Emergency Response Guide. If special chemical protective clothing is required, consult the chemical manufacturer or specific protective clothing compatibility charts.
- Delay entry until trained personnel and proper protective equipment are available.
- Remove patient from contaminated area.
- Quickly remove and isolate patient's clothing, jewelry, and shoes.
- Gently brush away dry particles and blot excess liquids with absorbent material.
- Rinse patient with copious amounts of warm water, 30°C/86°F, if possible.
- Wash patient with Tincture of Green soap or a mild liquid soap and large quantities of water.
- Speed in removing product from skin is essential in limiting tissue damage.
- Refer to decontamination protocol in Section Three.

**IMMEDIATE FIRST AID**
- Ensure that adequate decontamination has been carried out.
- If victim is not breathing, start artificial respiration, preferably with a demand-valve resuscitator, bag-valve-mask device, or pocket mask as trained. Perform CPR as necessary.
- Immediately flush contaminated eyes with gently flowing water.
- Do not induce vomiting. If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain an open airway and prevent aspiration.
- Keep victim quiet and maintain normal body temperature.
- Obtain medical attention.
BASIC TREATMENT
- Establish a patent airway. Suction if necessary.
- Watch for signs of respiratory insufficiency and assist respirations if needed.
- Administer oxygen by nonrebreather mask at 10 to 15 L/min.
- Monitor for pulmonary edema and treat if necessary (refer to pulmonary edema protocol in Section Three).
- Monitor for shock and treat if necessary (refer to shock protocol in Section Three).
- For eye contamination, flush eyes immediately with water. Irrigate each eye continuously with normal saline during transport (refer to eye irrigation protocol in Section Three).
- Do not use emetics. Activated charcoal is not effective. For ingestion, rinse mouth and administer 5 ml/kg up to 200 ml of water for dilution if the patient can swallow, has a strong gag reflex, and does not drool (refer to ingestion protocol in Section Three).
- Do not attempt to neutralize because of exothermic reaction.
- Cover skin burns with dry, sterile dressings after decontamination (refer to chemical burn protocol in Section Three).

ADVANCED TREATMENT
- Consider orotracheal or nasotracheal intubation for airway control in the patient who is unconscious, has severe pulmonary edema, or is in respiratory arrest. Early intubation, at the first sign of upper airway obstruction, may be necessary.
- Positive-pressure ventilation techniques with a bag-valve-mask device may be beneficial.
- Monitor cardiac rhythm and treat arrhythmias as necessary (refer to cardiac protocol in Section Three).
- Start an IV with D5W TKO. Use lactated Ringer’s if signs of hypovolemia are present. Watch for signs of fluid overload.
- Consider drug therapy for pulmonary edema (refer to pulmonary edema protocol in Section Three).
- For hypotension with signs of hypovolemia, administer fluid cautiously. Consider vasopressors if patient is hypotensive with a normal fluid volume. Watch for signs of fluid overload (refer to shock protocol in Section Three).
- Use proparacaine hydrochloride to assist eye irrigation (refer to proparacaine hydrochloride protocol in Section Four).

INITIAL EMERGENCY DEPARTMENT CONSIDERATIONS
- Useful initial laboratory studies include complete blood count, platelet count, coagulation profile, serum electrolytes, blood urea nitrogen (BUN), creatinine, glucose, urinalysis, and baseline biochemical profile, including serum aminotransferases (ALT and AST), calcium, phosphorus, and magnesium. Determination of anion and osmolar gaps may be helpful. Arterial blood gases (ABGs), chest radiograph, and electrocardiogram may be required.
- Products may cause acidosis; hyperventilation and sodium bicarbonate may be beneficial. Bicarbonate therapy should be guided by clinical presentation, ABG determinations, and serum electrolyte considerations.
- Positive end-expiratory pressure (PEEP)-assisted ventilation may be necessary in patients with acute parenchymal injury who develop pulmonary edema or adult respiratory distress syndrome.
Inorganic Acids and Related Compounds

- Bronchospastic symptoms should be treated with an inhalation medication regime similar to that used for reactive airways disease. Inhaled corticosteroids may be of value in severe bronchospasm.
- Oral exposures may require endoscopy.
- Obtain toxicological consultation as necessary.
Organic Acids and Related Compounds

SUBSTANCE IDENTIFICATION
Short-chained organic acids are usually found as colorless liquids. Some products may fume in air, presenting an increased inhalation exposure hazard. Most products have pungent, penetrating odors. Longer-chained organic acids tend to be waxy solids. Used in food preservatives, dyes, insecticides, photographic chemicals, fumigants, leather tanning processes, and in the manufacture of pharmaceuticals, rubber, and plastics. Products are highly corrosive to tissue and reactive with metals.

ROUTES OF EXPOSURE
Skin and eye contact
Inhalation
Ingestion
Skin absorption

TARGET ORGANS
Primary
Skin
Eyes
Respiratory system
Gastrointestinal system
Metabolism
Secondary
Central nervous system
Cardiovascular system

LIFE THREAT
Pulmonary edema, circulatory collapse, laryngeal edema, spasm, GI tract perforation, hemorrhage, and peritonitis. Corrosive to skin, mucous membranes, and internal organs.

SIGNS AND SYMPTOMS BY SYSTEM
Cardiovascular: Hypovolemic shock, circulatory collapse, and tachycardia with weak pulse.
Respiratory: Mucosal edema, erythema, coughing, sneezing, dyspnea, upper airway obstruction from laryngeal spasm or glottic edema with stridor and pain, acute pulmonary edema, asphyxia, and chemical pneumonitis.
CNS: Symptoms of hypoxia, stupor, lethargy, and coma.
Gastrointestinal: Ingestion can result in burns to the mouth, esophagus, stomach, and lower GI tract. Salivation, nausea, vomiting, and diarrhea, hematemesis, melena, and abdominal muscle spasm.
Eye: Conjunctivitis, spasm of the eyelid (blepharospasm), blurred vision, corneal destruction, and possibly blindness.
Skin: Full- or partial-thickness burns with blistering and tissue damage. Dermal ulceration with eschar formation secondary to coagulation of superficial tissue proteins. This coagulation necrosis generally limits further tissue destruction and acid penetration.
Metabolism: Metabolic acidosis.
SYMPTOM ONSET FOR ACUTE EXPOSURE
Immediate; respiratory symptoms may be delayed

CO-EXPOSURE CONCERNS
Other acids
Alkalies (exothermic reaction possible)

THERMAL DECOMPOSITION PRODUCTS INCLUDE
Carbon dioxide
Carbon monoxide

MEDICAL CONDITIONS POSSIBLY AGGRAVATED BY EXPOSURE
Respiratory system disorders

DECONTAMINATION
- Wear positive-pressure SCBA and protective equipment specified by references such as the DOT Emergency Response Guidebook or the CANUTEC Initial Emergency Response Guide. If special chemical protective clothing is required, consult the chemical manufacturer or specific protective clothing compatibility charts.
- Delay entry until trained personnel and proper protective equipment are available.
- Remove patient from contaminated area.
- Quickly remove and isolate patient's clothing, jewelry, and shoes.
- Gently brush away dry particles and blot excess liquids with absorbent material.
- Rinse patient with warm water, 30°C/86°F, if possible.
- Wash patient with Tincture of Green soap or a mild liquid soap and large quantities of water.
- Speed in removing product from skin is essential in limiting tissue damage.
- Refer to decontamination protocol in Section Three.

IMMEDIATE FIRST AID
- Ensure that adequate decontamination has been carried out.
- If victim is not breathing, start artificial respiration, preferably with a demand-valve resuscitator, bag-valve-mask device, or pocket mask as trained. Perform CPR if necessary.
- Immediately flush contaminated eyes with gently flowing water.
- Do not induce vomiting. If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain an open airway and prevent aspiration.
- Keep victim quiet and maintain normal body temperature.
- Obtain medical attention.

BASIC TREATMENT
- Establish a patent airway. Suction if necessary.
- Watch for signs of respiratory insufficiency and assist respirations if necessary.
- Administer oxygen by nonrebreather mask at 10 to 15 L/min.
- Monitor for pulmonary edema and treat if necessary (refer to pulmonary edema protocol in Section Three).
- Monitor for shock and treat if necessary (refer to shock protocol in Section Three).
- For eye contamination, flush eyes immediately with water. Irrigate each eye continuously with normal saline during transport (refer to eye irrigation protocol in Section Three).
- Do not use emetics. For ingestion, rinse mouth and administer 5 ml/kg up to 200 ml of water for dilution if the patient can swallow, has a strong gag reflex, and does not drool. Activated charcoal is not effective (refer to ingestion protocol in Section Three).
- Do not attempt to neutralize because of exothermic reaction.
- Cover skin burns with dry, sterile dressings after decontamination (refer to chemical burn protocol in Section Three).

**ADVANCED TREATMENT**
- Consider orotracheal or nasotracheal intubation for airway control in the patient who is unconscious, has severe pulmonary edema, or is in respiratory arrest. Early intubation, at the first sign of upper airway obstruction, may be necessary.
- Positive-pressure ventilation techniques with a bag-valve-mask device may be beneficial.
- Monitor cardiac rhythm and treat arrhythmias as necessary (refer to cardiac protocol in Section Three).
- Start an IV with D$_5$W TKO. Use lactated Ringer's if signs of hypovolemia are present. Watch for signs of fluid overload.
- Consider drug therapy for pulmonary edema (refer to pulmonary edema protocol in Section Three).
- For hypotension with signs of hypovolemia, administer fluid cautiously. Consider vasopressors if hypotensive with a normal fluid volume. Watch for signs of fluid overload (refer to shock protocol in Section Three).
- Use proparacaine hydrochloride to assist eye irrigation (refer to proparacaine hydrochloride protocol in Section Four).

**INITIAL EMERGENCY DEPARTMENT CONSIDERATIONS**
- Useful initial laboratory studies include complete blood count, serum electrolytes, blood urea nitrogen (BUN), creatinine, glucose, urinalysis, baseline biochemical profile, including serum aminotransferases (ALT and AST), calcium, phosphorus, magnesium, determination of anion and osmolar gaps, arterial blood gases (ABGs), chest radiograph, and electrocardiogram.
- Products may cause acidosis; hyperventilation and sodium bicarbonate may be beneficial. Bicarbonate therapy should be guided by clinical presentation, ABG determinations, and serum electrolyte considerations.
- Positive end-expiratory pressure (PEEP)–assisted ventilation may be necessary in patients with acute parenchymal injury who develop pulmonary edema or adult respiratory distress syndrome.
- Bronchospastic symptoms should be treated with an inhalation medication regime similar to that used for reactive airways disease. Inhaled corticosteroids may be of value in severe bronchospasm.
- Oral exposures may require endoscopy.
- Obtain toxicological consultation as necessary.

**SPECIAL CONSIDERATIONS**
- Formic acid is a toxic metabolite of methanol. Formic acid and formaldehyde are the agents responsible for the anion gap acidosis and ocular toxicity of methanol poisoning. Refer to methanol (guideline 31).
Hydrofluoric Acid (HF) and Related Compounds

SUBSTANCE IDENTIFICATION
An extremely volatile, colorless corrosive liquid or a colorless gas with a sharp, irritating odor. Used in etching, manufacturing of fluorinated chemicals, electropolishing of metals, and the semiconductor industry. May react with metal to generate hydrogen gas. The liquid product evaporates and produces large amounts of vapors and white fumes.

ROUTES OF EXPOSURE
Skin and eye contact
Inhalation
Ingestion
Skin absorption

TARGET ORGANS
Primary
Eyes
Skin
Respiratory system
Gastrointestinal system
Metabolism
Secondary
Central nervous system
Cardiovascular system

LIFE THREAT
Pulmonary edema, laryngeal edema, circulatory collapse, and severe skin burns. May cause perforation of GI tract. Systemic fluoride poisoning may result.

SIGNS AND SYMPTOMS BY SYSTEM
Cardiovascular: Cardiac arrhythmias, tachycardia with weak pulse, asystole, hypovolemic shock, and circulatory collapse.
Respiratory: Acute pulmonary edema, asphyxia, chemical pneumonitis. Upper airway obstruction with stridor, pain, and cough secondary to airway edema.
CNS: Symptoms of hypoxia, stupor, lethargy, altered sensorium, and coma.
Gastrointestinal: Acute toxicity results in burns to the mouth, esophagus, stomach, lower GI tract; nausea; vomiting; diarrhea; and possibly hemorrhage.
Eye: Conjunctivitis, opacification of the cornea, blindness.
Skin: Severe pain and normal-looking skin with small, total-surface-area burns. As concentration and surface area of burn increase, skin may look whitish (blanched). Burn is in lower skin layers. Damage may be severe with no outward signs or symptoms, except intense pain. In severe cases, demineralization of bone may occur.
Metabolism: Hypocalcemia and hypomagnesemia may be seen. Even dilute solutions (less than 3% hydrofluoric acid) may cause serious or fatal injury in cases of large skin surface area involvement or oral ingestion. Hyperkalemia may also be observed.
Other: Hydrofluoric acid is a systemic poison, as well as a primary irritant. Burns over
Hydrofluoric Acid (HF) and Related Compounds

20% of body surface may have a fatal outcome as a result of systemic fluoride poisoning. Any skin contamination in a hydrofluoric acid use area should be considered a hydrofluoric acid exposure.

**SYMPTOM ONSET FOR ACUTE EXPOSURE**
Immediate
Skin and systemic effects possibly delayed; onset depends on product concentration, route, and duration of exposure

**THERMAL DECOMPOSITION PRODUCTS INCLUDE**
Hydrogen
Hydrogen fluoride

**MEDICAL CONDITIONS POSSIBLY AGGRAVATED BY EXPOSURE**
Respiratory system disorders
Cardiac disorders
Kidney disorders

**DECONTAMINATION**
- Wear positive-pressure SCBA and protective equipment specified by references such as the DOT Emergency Response Guidebook or the CANUTEC Initial Emergency Response Guide. If special chemical protective clothing is required, consult the chemical manufacturer or specific protective clothing compatibility charts.
- Delay entry until trained personnel and proper protective equipment are available.
- Remove patient from contaminated area.
- Quickly remove and isolate patient’s clothing, jewelry, and shoes.
- Gently brush away dry particles and blot excess liquids with absorbent material.
- Rinse patient with warm water, 30°C/86°F, if possible.
- Wash patient with Tincture of Green soap or a mild liquid soap and large quantities of water.
- A 0.13% benzalkonium chloride (Zephiran chloride) solution may be used for decontamination, if available. Do not delay decontamination to obtain benzalkonium chloride solution.
- Refer to decontamination protocol in Section Three.

**IMMEDIATE FIRST AID**
- Ensure that adequate decontamination has been carried out.
- If victim is not breathing, start artificial respiration, preferably with a demand-valve resuscitator, bag-valve-mask device, or pocket mask as trained. Perform CPR if necessary.
- Immediately flush contaminated eyes with gently flowing water.
- Do not induce vomiting. If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain an open airway and prevent aspiration.
- Keep victim quiet and maintain normal body temperature.
- Effects may be delayed. Obtain medical attention for any exposure.

**BASIC TREATMENT**
- Establish a patent airway. Suction if necessary.
- Watch for signs of respiratory insufficiency and assist ventilations if necessary.
- Administer oxygen by nonrebreather mask at 10 to 15 L/min.
- Monitor for pulmonary edema and treat if necessary (refer to pulmonary edema protocol in Section Three).
- Monitor for shock and treat if necessary (refer to shock protocol in Section Three).
Hydrofluoric Acid (HF) and Related Compounds

- For eye contamination, flush eyes immediately with water. Irrigate each eye continuously with normal saline during transport (refer to ingestion protocol in Section Three).
- Do not use emetics. For ingestion, rinse mouth and administer 5 ml/kg up to 200 ml of water if the patient can swallow, has a strong gag reflex, and does not drool (refer to ingestion protocol in Section Three).
- Do not attempt to neutralize because of exothermic reaction.
- Cover skin burns with dry, sterile dressings after decontamination (refer to chemical burn protocol in Section Three).

ADVANCED TREATMENT

- Consider orotracheal or nasotracheal intubation for airway control in the patient who is unconscious, has severe pulmonary edema, or is in respiratory arrest. Early intubation at the first sign of upper airway obstruction may be necessary.
- Positive-pressure ventilation with a bag-valve-mask device may be beneficial.
- Monitor cardiac rhythm and treat arrhythmias as necessary.
- Development of QT interval prolongation may signal hypocalcemia and need for IV calcium gluconate administration (refer to cardiac protocol in Section Three and calcium gluconate protocol in Section Four).
- Start an IV with D5W TKO. Use lactated Ringer's if signs of hypovolemia are present. Watch for signs of fluid overload.
- Consider drug therapy for pulmonary edema (refer to pulmonary edema protocol in Section Three).
- For hypotension with signs of hypovolemia, administer fluid cautiously. Consider vasopressors if patient is hypotensive with a normal fluid volume. Watch for signs of fluid overload (refer to shock protocol in Section Three).
- Use 2.5% calcium gluconate gel for skin burns over painful areas after thorough decontamination (refer to calcium gluconate gel protocol in Section Four). Iced 0.13% benzalkonium chloride (Zephiran chloride) solution soak or compresses may be used. Compresses should be changed every 2 min.
- Use proparacaine hydrochloride to assist eye irrigation (refer to proparacaine hydrochloride protocol in Section Four).

INITIAL EMERGENCY DEPARTMENT CONSIDERATIONS

- Useful initial laboratory studies include complete blood count, serum electrolytes, blood urea nitrogen (BUN), glucose, creatinine, urinalysis and baseline biochemical profile, including serum aminotransferases (ALT and AST), calcium, phosphorus, and magnesium. Determination of anion and osmolar gaps may be helpful. Arterial blood gases (ABGs), chest radiograph, and electrocardiogram may be required.
- Positive end-expiratory pressure (PEEP)-assisted ventilation may be necessary in patients with acute parenchymal injury who develop pulmonary edema or adult respiratory distress syndrome.
- Subcutaneous or intraarterial 5% calcium gluconate may be needed for dermal exposure.
- IV calcium gluconate may be needed for severe systemic hypocalcemia (refer to calcium gluconate protocol in Section Three).
- Massive hypocalcemia/hypomagnesemia from concentrated hydrofluoric acid exposure demonstrates high morbidity/mortality. Rapid, aggressive treatment is required, with close monitoring of electrocardiogram, serum electrolytes, calcium, and magnesium.
· Monitor and treat for hyperkalemia as necessary.
· Obtain toxicological consultation as necessary.

SPECIAL CONSIDERATIONS

· Dermal injury is proportional to the concentration, duration of skin contact, and release of hydrogen and fluoride ions into soft tissue, blood, and bones. Fluoride ions bind with calcium and magnesium, forming insoluble salts. This reaction produces liquefaction necrosis in deep tissue/bone and interferes with cellular metabolism, such as in myocardial tissue, producing cell death.
Oxalate and Related Compounds

SUBSTANCE IDENTIFICATION
Found as a colorless, odorless solid or liquid. Used as a metal cleaner/polisher in textile cleaning, flameproofing, rust removal, anti-corrosion coating and as a chemical intermediate/catalyst in the photography, ceramics, and rubber industry. Can be found in certain plants such as rhubarb leaves and dieffenbachia.

ROUTES OF EXPOSURE
Skin and eye contact
Inhalation
Ingestion
Slow skin absorption

TARGET ORGANS
Primary
Skin
Eyes
Respiratory system
Renal metabolism
Secondary
Central nervous system
Cardiovascular system
Gastrointestinal tract

LIFE THREAT
Cardiovascular collapse, arrhythmias, and seizures.

SIGNS AND SYMPTOMS BY SYSTEM
Cardiovascular: Cardiovascular collapse, hypotension, and arrhythmias.
Respiratory: Irritation of respiratory tract.
CNS: Headache, muscle cramps, tetany, fasciculations, seizures, CNS depression, coma, increased deep tendon reflexes.
Gastrointestinal: Nausea, vomiting (hematemesis) and burning pain in mouth, esophagus, and stomach. Exposed mucous membranes turn white in color.
Eye: Chemical conjunctivitis, corneal damage, and burns.
Skin: Irritant dermatitis, full- or partial-thickness burns.
Renal: Kidney damage and oxaluria.
Metabolism: Hypocalcemia.

SYMPTOM ONSET FOR ACUTE EXPOSURE
Immediate
Systemic symptoms possibly delayed

THERMAL DECOMPOSITION PRODUCTS INCLUDE
Carbon dioxide
Carbon monoxide
Depending on product, may release:
Ammonia
Nitrogen oxides
Oxalic acid

**MEDICAL CONDITIONS POSSIBLY AGGRAVATED BY EXPOSURE**

Respiratory system disorders
Kidney disorders
Dermatitis

**DECONTAMINATION**

- Wear positive-pressure SCBA and protective equipment specified by references such as the *DOT Emergency Response Guidebook* or the *CANUTEC Initial Emergency Response Guide*. If special chemical protective clothing is required, consult the chemical manufacturer or specific protective clothing compatibility charts.
- Delay entry until trained personnel and proper protective equipment are available.
- Remove patient from contaminated area.
- Quickly remove and isolate patient’s clothing, jewelry, and shoes.
- Gently brush away dry particles and blot excess liquids with absorbent material.
- Rinse patient with warm water, 30° C/86° F, if possible.
- Wash patient with Tincture of Green soap or a mild liquid soap and large quantities of water.
- Refer to decontamination protocol in Section Three.

**IMMEDIATE FIRST AID**

- Ensure that adequate decontamination has been carried out.
- If victim is not breathing, start artificial respiration, preferably with a demand-valve resuscitator, bag-valve-mask device, or pocket mask as trained. Perform CPR if necessary.
- Immediately flush contaminated eyes with gently flowing water.
- Do not induce vomiting. If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain an open airway and prevent aspiration.
- Keep victim quiet and maintain normal body temperature.
- Obtain medical attention.

**BASIC TREATMENT**

- Establish a patent airway. Suction if necessary.
- Watch for signs of respiratory insufficiency and assist ventilations as needed.
- Administer oxygen by nonrebreather mask at 10 to 15 L/min.
- Monitor for shock and treat if necessary (refer to shock protocol in Section Three).
- Anticipate seizures and treat if necessary (refer to seizure protocol in Section Three).
- For eye contamination, flush eyes immediately with water. Irrigate each eye continuously with normal saline during transport (refer to eye irrigation protocol in Section Three).
- Do not use emetics. For ingestion, rinse mouth and administer 5 ml/kg up to 200 ml of water for dilution if the patient can swallow, has a strong gag reflex, and does not drool (refer to ingestion protocol in Section Three).
- Cover chemical burns with dry, sterile dressings after decontamination (refer to chemical burn protocol in Section Three).

**ADVANCED TREATMENT**

- Consider orotracheal or nasotracheal intubation for airway control in the unconscious patient.
- Monitor cardiac rhythm and treat arrhythmias as necessary (refer to cardiac protocol in Section Three).
Oxalate and Related Compounds

- Start an IV with lactated Ringer's TKO. Watch for signs of fluid overload.
- For hypotension with signs of hypovolemia, administer fluid cautiously. Watch for signs of fluid overload (refer to shock protocol in Section Three).
- Treat seizures with diazepam (Valium) (refer to diazepam protocol in Section Four).
- Use proparacaine hydrochloride to assist eye irrigation (refer to proparacaine hydrochloride protocol in Section Four).

INITIAL EMERGENCY DEPARTMENT CONSIDERATIONS

- Useful initial laboratory studies include complete blood count, serum electrolytes, blood urea nitrogen (BUN), creatinine, glucose, urinalysis, and baseline biochemical profile, including serum aminotransferases (ALT and AST), calcium, phosphorus, and magnesium. Determination of anion and osmolar gaps may be helpful. Arterial blood gases (ABGs), chest radiograph, and electrocardiogram may be required.
- Hemodialysis may be indicated in the presence of renal failure.
- IV calcium gluconate may be needed for hypocalcemia. Treatment should be guided by clinical presentation and laboratory values.
- Obtain toxicological consultation as necessary.

SPECIAL CONSIDERATIONS

Oxalic acid is produced by the metabolism of ethylene glycol. Refer to ethylene glycol, glycols, and related compounds (guideline 40).
Sodium Azide (NaN₃) and Related Compounds

SUBSTANCE IDENTIFICATION
Found as a white, odorless crystalline solid. Used in the manufacture of explosives and pharmaceuticals; as a laboratory reagent; as a herbicide, fungicide, nematocide, and soil fumigant; in the preparation of hydrazolic acid (HN₃), lead azide, and pure sodium; and as an intermediate in organic synthesis. Sodium azide is also used as a propellant for inflating automobile airbags. The hermetically sealed gas generator in the airbag unit may contain 400 to 600 g of propellant that usually contains only 30% to 40% of sodium azide. On ignition, nitrogen gas, trace amounts of sodium metal, and sodium hydroxide are produced. The nitrogen gas inflates the air bag. Automobile manufacturer data show that there is complete combustion of the sodium azide on airbag inflation. Therefore sodium azide exposure risk after inflation is not expected. Skin or eye irritation, if present, may be the result of sodium hydroxide or direct trauma from airbag contact.

ROUTES OF EXPOSURE
Skin and eye contact
Inhalation
Ingestion
Skin absorption

TARGET ORGANS
Primary
Skin
Eyes
Central nervous system
Cardiovascular system
Respiratory system
Blood
Secondary
Gastrointestinal system
Metabolism

LIFE THREAT
Hypotension, cardiac arrhythmias, asystole, seizures, and coma.

SIGNS AND SYMPTOMS BY SYSTEM
Cardiovascular: Bradycardia, hypotension are common. Tachycardia, ventricular arrhythmias, asystole and vasodilation are possible.
Respiratory: Irritation of respiratory tract, chest pain, pulmonary edema, and respiratory failure.
CNS: Headache, apprehension, dizziness, muscle weakness, blurred vision, hyperreflexia, decreased level of consciousness, syncope, coma, and seizures.
Gastrointestinal: Irritation of the GI tract, nausea, vomiting, diarrhea, and polydipsia.
Eye: Conjunctivitis.
Skin: Irritation and cyanosis.
Metabolism: Metabolic acidosis.
Blood: Increased white blood cell count (leukocytosis) and platelet aggregation inhibition

Other: Body temperature regulation disturbances.

SYMPTOM ONSET FOR ACUTE EXPOSURE
Immediate
Some symptoms possibly delayed

CO-EXPOSURE CONCERNS
When mixed with acids, hydrazolic acid is produced
Nitrites/nitrates

THERMAL DECOMPOSITION PRODUCTS INCLUDE
Hydrazolic acid fumes (explosion hazard)
Nitrogen oxides
Sodium
Sodium hydroxide

MEDICAL CONDITIONS POSSIBLY AGGRAVATED BY EXPOSURE
Hypertensive cardiac disease

DECONTAMINATION
- Wear positive-pressure SCBA and protective equipment specified by references such as the DOT Emergency Response Guidebook or the CANUTEC Initial Emergency Response Guide. If special chemical protective clothing is required, consult the chemical manufacturer or specific protective clothing compatibility charts.
- Delay entry until trained personnel and proper protective equipment are available.
- Remove patient from contaminated area.
- Quickly remove and isolate patient’s clothing, jewelry, and shoes
- Gently brush away dry particles and blot excess liquids with absorbent material.
- Rinse patient with warm water, 30°C/86°F, if possible.
- Wash patient with Tincture of Green soap or a mild liquid soap and large quantities of water.
- Refer to decontamination protocol in Section Three.

IMMEDIATE FIRST AID
- Ensure that adequate decontamination has been carried out.
- If victim is not breathing, start artificial respiration, preferably with a demand-valve resuscitator, bag-valve-mask device, or pocket mask as trained. Perform CPR if necessary.
- Immediately flush contaminated eyes with gently flowing water.
- Do not induce vomiting. If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain an open airway and prevent aspiration.
- Keep victim quiet and maintain normal body temperature.
- Obtain medical attention.

BASIC TREATMENT
- Establish a patent airway. Suction if necessary.
- Watch for signs of respiratory insufficiency and assist respirations if necessary.
- Administer oxygen by nonrebreather mask at 10 to 15 L/min.
- Monitor for pulmonary edema and treat if necessary (refer to pulmonary edema protocol in Section Three).
- Monitor for shock and treat if necessary (refer to shock protocol in Section Three).
- Anticipate seizures and treat if necessary (refer to seizure protocol in Section Three).
For eye contamination, flush eyes immediately with water. Irrigate each eye continuously with normal saline during transport (refer to eye irrigation protocol in Section Three).

Do not use emetics. For ingestion, rinse mouth and administer 5 ml/kg up to 200 ml of water for dilution if the patient can swallow, has a strong gag reflex, and does not drool. Administer activated charcoal (refer to ingestion protocol in Section Three and activated charcoal protocol in Section Four).

ADVANCED TREATMENT

- Consider orotracheal or nasotracheal intubation for airway control in the patient who is unconscious, has severe pulmonary edema, or is in respiratory arrest. Early intubation, at the first sign of upper airway obstruction, may be necessary.
- Positive-pressure ventilation techniques with a bag-valve-mask device may be beneficial.
- Monitor cardiac rhythm and treat arrhythmias as necessary (refer to cardiac protocol in Section Three).
- Start an IV with D₅W TKO. Use lactated Ringer's if signs of hypovolemia are present. Watch for signs of fluid overload.
- Consider drug therapy for pulmonary edema (refer to pulmonary edema protocol in Section Three).
- Treat seizures with diazepam (Valium) (refer to diazepam protocol in Section Four).
- For hypotension with signs of hypovolemia, administer fluid cautiously. Consider vasopressors if patient is hypotensive with a normal fluid volume. Watch for signs of fluid overload (refer to shock protocol in Section Three).
- Use proparacaine hydrochloride to assist eye irrigation (refer to proparacaine hydrochloride protocol in Section Four).

INITIAL EMERGENCY DEPARTMENT CONSIDERATIONS

- Useful initial laboratory studies include complete blood count, serum electrolytes, blood urea nitrogen (BUN), creatinine, glucose, urinalysis, and baseline biochemical profile, including serum aminotransferases (ALT and AST), calcium, phosphorus, magnesium, and coagulation profiles. Arterial blood gases (ABGs), chest radiograph, and electrocardiogram may be required.
- Positive end-expiratory pressure (PEEP)—assisted ventilation may be necessary in patients with acute parenchymal injury who develop pulmonary edema or adult respiratory distress syndrome.
- Products may cause acidosis; hyperventilation and sodium bicarbonate may be beneficial. Bicarbonate therapy should be guided by patient presentation. ABG determination and serum electrolyte considerations.
- Obtain toxicological consultation as necessary.

SPECIAL CONSIDERATIONS

- Sodium azide may block oxidative phosphorylation and the cytochrome oxidase system. Symptoms may mimic cyanide poisoning. The use of the cyanide antidote kit has been recommended for sodium azide exposure. This treatment is controversial. There are not sufficient data at this time to recommend it.
Inorganic Bases/Alkaline Corrosives and Related Compounds

SUBSTANCE IDENTIFICATION
Found as solids in pellets, flakes, lumps, or sticks and liquids. Used as acid neutralizers; in petroleum refining; in cleaning agents, paint removers, solvents; and in water treatment processes. Part of the manufacturing process of cellulose, paper, textiles, and plastics.

ROUTES OF EXPOSURE
Skin and eye contact
Inhalation
Ingestion

TARGET ORGANS
Primary
Skin
Eyes
Respiratory system
Gastrointestinal system
Secondary
Central nervous system
Cardiovascular system

LIFE THREAT
Severe tissue irritant that may cause upper airway burns and edema, pulmonary edema, and skin burns. May cause GI perforation, hemorrhage, and peritonitis leading to circulatory collapse.

SIGNS AND SYMPTOMS BY SYSTEM
Cardiovascular: Tachycardia, hypotension, and shock.
Respiratory: Dyspnea, tachypnea, sneezing, coughing, stridor, burns, upper airway edema, and pulmonary edema.
CNS: Apathy, mental confusion, blurred vision, and tremors.
Gastrointestinal: Nausea; vomiting; hemorrhage; perforation; abdominal pain; painful swallowing; profuse salivation; and burns to the mouth, esophagus, stomach, and gastrointestinal tract may occur.
Eye: Chemical conjunctivitis, corneal ulceration, severe scarring, permanent blindness.
Skin: Deep-tissue chemical burns, skin rash (in milder cases), cold and clammy skin with cyanosis or pale color.

SYMPTOM ONSET FOR ACUTE EXPOSURE
Immediate
Some symptoms such as pulmonary edema, GI perforation, and cardiovascular collapse possibly delayed
CO-EXPOSURE CONCERNS
Other alkalis
Acids (exothermic reaction)

MEDICAL CONDITIONS POSSIBLY AGGRAVATED BY EXPOSURE
Respiratory system disorders
Gastrointestinal disorders

DECONTAMINATION
- Wear positive-pressure SCBA and protective equipment specified by references such as the DOT Emergency Response Guidebook or the CANUTEC Initial Emergency Response Guide. If special chemical protective clothing is required, consult the chemical manufacturer or specific protective clothing compatibility charts.
- Delay entry until trained personnel and proper protective equipment are available.
- Remove patient from contaminated area.
- Quickly remove and isolate patient’s clothing, jewelry, and shoes.
- Gently brush away dry particles and blot excess liquids with absorbent material.
- Rinse patient with warm water, 30°C/86°F, if possible.
- Wash patient with Tincture of Green soap or a mild liquid soap and large quantities of water.
- Speed in removing product from skin is essential in limiting tissue damage.
- Refer to decontamination protocol in Section Three.

IMMEDIATE FIRST AID
- Remove victim from contact with the material.
- Ensure that adequate decontamination has been carried out.
- If victim is not breathing, start artificial respiration, preferably with a demand valve resuscitator, bag-valve-mask device, or pocket mask as trained. Perform CPR if necessary.
- Immediately flush contaminated eyes with gently flowing water.
- Do not induce vomiting. If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain an open airway and prevent aspiration.
- Keep victim quiet and maintain normal body temperature.
- Obtain medical attention.

BASIC TREATMENT
- Establish a patent airway. Suction if necessary.
- Watch for signs of respiratory insufficiency and assist ventilations if necessary.
- Administer oxygen by nonrebreather mask at 6 to 12 L/min.
- Monitor for pulmonary edema and treat if necessary (refer to pulmonary edema protocol in Section Three).
- Monitor for shock and treat if necessary (refer to shock protocol in Section Three).
- For eye contamination, flush eyes immediately with water. Irrigate each eye continuously with normal saline during transport (refer to eye irrigation protocol in Section Three).
- Do not use emetics. For ingestion, rinse mouth and administer 5 ml/kg up to 200 ml of water for dilution if the patient can swallow, has a strong gag reflex, and does not drool (refer to ingestion protocol in Section Three).
- Do not attempt to neutralize.
- Cover skin burns with dry sterile dressings after decontamination (refer to chemical burn protocol in Section Three).
ADVANCED TREATMENT

- Consider orotracheal or nasotracheal intubation for airway control in the patient who is unconscious or in respiratory arrest. Early intubation, at the first signs of upper airway obstruction, may be necessary.
- Positive-pressure ventilation techniques with a bag-valve-mask device may be beneficial.
- Monitor cardiac rhythm and treat arrhythmias as necessary (refer to cardiac protocol in Section Three).
- Start an IV of D5W TKO. Use lactated Ringer's if signs of hypovolemia are present. Watch for signs of pulmonary edema.
- For hypotension with signs of hypovolemia, administer fluid cautiously. Watch for signs of fluid overload (refer to shock protocol in Section Three).
- Consider drug therapy for pulmonary edema (refer to pulmonary edema protocol in Section Three).
- Use proparacaine hydrochloride to assist eye irrigation (refer to proparacaine hydrochloride protocol in Section Four).

INITIAL EMERGENCY DEPARTMENT CONSIDERATIONS

- Useful initial laboratory studies include complete blood count, serum electrolytes, blood urea nitrogen (BUN), creatinine, glucose, urinalysis, and baseline biochemical profile, including serum aminotransferases (ALT and AST), calcium, phosphorus, and magnesium. Arterial blood gases (ABGs), chest radiograph, and electrocardiogram may be required.
- Positive end-expiratory pressure (PEEP)-assisted ventilation may be necessary in patients with acute parenchymal injury who develop pulmonary edema or adult respiratory distress syndrome.
- Endoscopy may be required for evaluation of oral ingestion.
- Obtain toxicological consultation as necessary.

SPECIAL CONSIDERATIONS

- Do not attempt to neutralize products because of exothermic reaction risk.
- Alkalies on contact with skin, mucous membranes, or conjunctival tissue produce a liquefaction necrosis that allows the substance to penetrate into deep tissue structures.