Nitrates, Nitrites, and Related Compounds

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SUBSTANCE IDENTIFICATION

Colorless-to–pale yellow liquid or solid. Used as fertilizers, pharmaceuticals, food preservatives; in metal treatment and finishing; as anticorrosion inhibitors; and in the manufacturing process of a variety of products. When mixed with organic compounds, many of these products may create flammable or explosive compounds.

ROUTES OF EXPOSURE

Skin and eye contact
Inhalation
Ingestion
Skin absorption

TARGET ORGANS

Primary
Skin
Eyes
Central nervous system
Cardiovascular system
Blood
Secondary
Respiratory system
Gastrointestinal system

LIFE THREAT

Products may cause methemoglobinemia, hypotension, and/or circulatory collapse.

SIGNS AND SYMPTOMS BY SYSTEM

Cardiovascular: Cardiovascular collapse secondary to vasodilation and arrhythmias, tachycardia followed by bradycardia, rapid fall in blood pressure and orthostatic hypotension.

Respiratory: Tachypnea and dyspnea. A slowed respiratory rate may be observed in late stages of toxicity.

CNS: Headache, dizziness, visual disturbances, roaring in the ears, seizures, syncope, coma, and a generalized tingling sensation (paresthesia).

Gastrointestinal: Nausea, vomiting, diarrhea and abdominal pain.

Eye: Chemical conjunctivitis.

Skin: Flushed or extreme cyanosis with profuse sweating.

Blood: Methemoglobinemia.

SYMPTOM ONSET FOR ACUTE EXPOSURE

Immediate

CO-EXPOSURE CONCERNS

Alcohols
Organophosphates/carbamates
Hydrocarbon solvents

THERMAL DECOMPOSITION PRODUCTS INCLUDE

Nitrites
Nitrogen oxides

MEDICAL CONDITIONS POSSIBLY AGGRAVATED BY EXPOSURE

Respiratory disorders
Cardiac arrhythmias
Trauma

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 any 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 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 ventilations if necessary.
- 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 as 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 or in respiratory arrest.
- Monitor cardiac rhythm and treat arrhythmias if necessary (refer to cardiac protocol in Section Three).

- Start an IV with D_5W TKO. Use lactated Ringer's if signs of hypovolemia are present.
- For hypotension with signs of hypovolemia, administer fluid cautiously. If unresponsive to these measures, vasopressors may be helpful. 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).
- Administer 1% solution methylene blue if patient is symptomatic with severe hypoxia, cyanosis, and cardiac compromise not responding to oxygen. DIRECT PHYSICIAN ORDER ONLY (refer to methylene blue 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. Arterial blood gases (ABGs), chest radiographs, and electrocardiogram may be required.
- Monitor blood methemoglobin levels and treat with methylene blue if patient is symptomatic and/or has a blood methemoglobin level greater than 30% (refer to methylene blue protocol in Section Four).
- Hyperbaric oxygen may be helpful.
- Obtain toxicological consultation as necessary.

SPECIAL CONSIDERATIONS

- Withdrawal from chronic occupational (usually longer than 1 year) nitrate/nitrite exposure may cause coronary vasospasm in the absence of atherosclerosis. This condition mimics ischemic heart disease. Therefore the use of epinephrine and other sympathomimetic compounds should be administered with caution.

48 Nitrogen Oxides (NO_x) and Related Compounds

SUBSTANCE IDENTIFICATION

Can be found in gas, liquid, or solid form. Most gases are colorless to brown with a sharp odor. Used as chemical warfare and personal protection agents, propellant fuels, and agricultural fumigants. Others are used in laboratory research and as solvents, bleaching agents, and refrigerants. They may be released from the combustion or decomposition of substances that contain nitrogen. Toxic exposure symptoms may result from working in grain silos ("Silo Filler's Disease"). Some products may ignite other combustible materials. Nitrous oxide (N_2O) is used as an anesthetic gas.

ROUTES OF EXPOSURE

Skin and eye contact
Inhalation
Ingestion
Skin absorption (rare)

TARGET ORGANS

Primary
Skin
Eyes
Cardiovascular system
Respiratory system
Blood
Secondary
Central nervous system
Gastrointestinal system

LIFE THREAT

As a result of poor water solubility, lower respiratory tract symptoms predominate: pulmonary edema, laryngospasm, bronchospasm, and asphyxiation.

SIGNS AND SYMPTOMS BY SYSTEM

Cardiovascular: Cardiovascular collapse with a rapid and weak pulse, reflex bradycardia.

Respiratory: Dyspnea, cough, laryngospasm, bronchospasm, wheezing, chest pain, chemical pneumonitis, pulmonary edema, and upper airway obstruction from edema of the glottis. With most agents, a mild and transient cough is the only symptom at the time of exposure. Onset of dyspnea, rapid respirations, violent coughing, and pulmonary edema usually are delayed. Some agents work immediately on the upper airway, resulting in chest pain, choking, and spasm of the glottis, which may result in a temporary apneic period.

CNS: Fatigue, restlessness, and decreasing level of consciousness are usually delayed signs.

Gastrointestinal: Burning/irritation of the mucous membranes, nausea, vomiting, and abdominal pain.

Eye: Chemical conjunctivitis.

Skin: Irritation of moist skin areas, pallor, and cyanosis.

Blood: Methemoglobinemia (usually mild).
Other: Olfactory fatigue. Some products may present a human carcinogenic risk.

SYMPTOM ONSET FOR ACUTE EXPOSURE

Immediate
Pulmonary edema symptoms possibly delayed for 4 to 12 hours
Other respiratory symptoms possibly delayed
CNS symptoms possibly delayed

CO-EXPOSURE CONCERNS

Carbon monoxide
Simple asphyxiants
Nitrates/nitrites

THERMAL DECOMPOSITION PRODUCTS INCLUDE

Nitrogen oxides

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 any 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.
- Aggressive airway management may be needed.
- Encourage patient to take deep breaths.
- 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).

- 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 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 with D_5W TKO.
- Consider drug therapy for pulmonary edema (refer to pulmonary edema protocol in Section Three).
- Consider the use of vasopressors to treat hypotension without signs of hypovolemia (refer to shock protocol in Section Three).
- Administer 1% solution methylene blue if patient is symptomatic with severe hypoxia, cyanosis, and cardiac compromise not responding to oxygen. DIRECT PHYSICIAN ORDER ONLY (refer to methylene blue 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. Arterial blood gases (ABGs), chest radiograph, and electrocardiogram may be required.
- Monitor blood methemoglobin levels and treat with methylene blue if patient is symptomatic and/or has a blood methemoglobin level greater than 30% (refer to methylene blue protocol in Section Four).
- 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.
- Obtain toxicological consultation as necessary.

SPECIAL CONSIDERATIONS

- In most cases of mild exposure, symptoms are self-limited and require supportive management only. Use of medications such as atropine, epinephrine, expectorants, and sedatives are not indicated and may cause further damage.
- Treat severe symptomatic exposures as required.
- Besides its role as an environmental poison, nitric oxide (NO) is also synthesized in the body by two enzyme systems to function as a physiologic messenger in the immune, cardiovascular, and neurologic systems. It is metabolized to nitrite after its messenger function is complete. The exact role of nitric oxide in health and disease requires further study.

Organophosphates and Related Compounds

49

SUBSTANCE IDENTIFICATION

Found as liquids, dusts, wettable powders, concentrates, and aerosols with a garlic-type odor. Used as insecticides. Products are among the most poisonous commonly used for pest control. Related to chemical warfare agents soman, sarin, and tabun.

ROUTES OF EXPOSURE

Skin and eye contact
Inhalation
Ingestion
Skin absorption

TARGET ORGANS

Primary
Central nervous system
Cardiovascular system
Respiratory system
Secondary
Skin
Eyes
Gastrointestinal system
Hepatic
Metabolism

LIFE THREAT

Respiratory failure caused by chemically mediated pulmonary edema and respiratory muscle paralysis. Inhibits acetylcholinesterase, causing over-stimulation of parasympathetic nervous system, striated muscle, sympathetic ganglia, and CNS Causes bradycardia, hypotension, and pulmonary edema.

SIGNS AND SYMPTOMS BY SYSTEM

Cardiovascular: Bradycardia (tachycardia possible), ventricular arrhythmias, A-V blocks, and hypotension.

Respiratory: Respiratory failure, bronchoconstriction, profuse pulmonary secretions (bronchorrhea), acute pulmonary edema, dyspnea, and tightness of the chest.

CNS: CNS depression, coma, anxiety, headache, dizziness, weakness, loss of muscle coordination, muscle fasciculations, seizures, disorientation, confusion, drowsiness, and slurred speech.

Gastrointestinal: Nausea, vomiting, diarrhea, abdominal cramps, excessive salivation, urination, and defecation.

Eye: Lacrimation, blurred vision. Constricted pupils are common; however, dilated pupils may be present.

Skin: Pale, cyanotic skin with excessive diaphoresis.

Hepatic: Liver damage.

Metabolism: Hypoglycemia or hyperglycemia, and acidosis.

Other: Hypothermia may occur. Classic SLUDGE syndrome (salivation, lacrimation, urination, defecation, GI pain, and emesis). May range from flu-type symptoms,

anxiety, seizures and coma. Symptoms may be broken down into muscarinic effects (classic SLUDGE syndrome, cardiac effects, constricted pupils, bronchoconstriction, pulmonary edema) and nicotinic symptoms (muscle fasciculations, tachycardia, hypertension, respiratory paralysis). If dermal absorption and nicotinic receptors are the primary sites of stimulation, the SLUDGE syndrome may not be clinically present. Instead, cardiovascular collapse may be the primary clinical manifestation. Cholinesterase inhibitor exposures may be cumulative. Symptoms in children may differ from those found in adults. Pediatric signs and symptoms include CNS depression, flaccid muscle tone, dyspnea, and coma. Many of these products use a hydrocarbon as a solvent vehicle. Identify the solvent and refer to the appropriate guideline for related toxic effects.

SYMPTOM ONSET FOR ACUTE EXPOSURE

Immediate
Some symptoms possibly delayed
After initial treatment, possible recurrence of symptoms several days to a week later

CO-EXPOSURE CONCERNS

Carbamates
Insecticide solvent

THERMAL DECOMPOSITION PRODUCTS INCLUDE

Carbon monoxide
Nitrogen oxides
Phosphorus oxides
Sulfur oxides

MEDICAL CONDITIONS POSSIBLY AGGRAVATED BY EXPOSURE

Respiratory disorders
Neurological 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 any dry particles and blot any 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.
- Products are highly absorbable, and decontamination is critical.
- Discard all exposed leather products.
- 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.
- Aggressive airway control may be needed.
- 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).
- 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 or has severe pulmonary edema.
- Positive-pressure ventilation techniques with a bag-valve-mask device may be beneficial.
- Monitor cardiac rhythm and treat arrhythmias if necessary (refer to cardiac protocol in Section Three).
- Start an IV with D_5W TKO. Use lactated Ringer's if signs of hypovolemia are present.
- Administer atropine. Correct hypoxia before giving atropine (refer to atropine protocol in Section Four).
- Administer pralidoxime chloride (2 PAM). USE UNDER DIRECT PHYSICIAN ORDERS ONLY (refer to pralidoxime chloride protocol in Section Four).
- Treat seizures with adequate atropinization and correction of hypoxia. Rarely is diazepam necessary (refer to seizure protocol in Section Three).
- For hypotension with signs of hypovolemia, administer fluid cautiously and consider vasopressors for hypotension 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, and magnesium. Arterial blood gases (ABGs), chest radiograph, and electrocardiogram may be required.

- Both plasma and red blood cell acetylcholinesterase levels should be obtained. Do not delay therapeutic interventions pending laboratory results. Treat symptomatically.
- 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.
- In cases of skin absorption and primary nicotinic stimulation, atropine may not reverse the respiratory paralysis. Be prepared to assist ventilations and administer 2 PAM. Initial symptoms may be diaphoresis, muscle fasciculations, and respiratory arrest.
- Depending on clinical symptoms, 2-PAM will be most effective if administered within 24 to 48 hours after exposure. After this time the organophosphate-acetylcholinesterase complex may "age" (develop an irreversible covalent bond) (refer to pralidoxime chloride protocol in Section Four).
- Obtain toxicological consultation as necessary.

SPECIAL CONSIDERATIONS

- Three clinical syndromes of organophosphate toxicity have been described: immediate, intermediate (1 to 4 days), and delayed (8 to 14 days) after exposure.
- Succinylcholine, other cholinergic agents, and aminophylline are contraindicated.
- Preservative-free atropine should be used to avoid toxicity from preservative agents.
- Mydriasis may occur early in the administration of atropine; however the endpoint for atropine administration is the drying of pulmonary secretions.
- Immediate or delayed ascending paralysis (dying back axonopathy) starting in the lower extremities may occur. This may be confused with Guillain-Barré syndrome
- Parathion and possibly other organophosphate insecticide residues may persist in clothing, despite repeated laundering.

Carbamates and Related Compounds

50

SUBSTANCE IDENTIFICATION

Found in solid, powder, or liquid form with a white-to-gray color and a weak odor. Reversible cholinesterase inhibitors, found in insecticides and herbicides. Products may be dissolved in hydrocarbon solvents and have concomitant hydrocarbon exposure effects.

ROUTES OF EXPOSURE

Skin and eye contact
Inhalation
Ingestion
Skin absorption

TARGET ORGANS

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

LIFE THREAT

Reversible inhibitor of acetylcholinesterase. Causes bradycardia, hypotension, paralysis of the respiratory muscles, respiratory arrest, and chemically mediated pulmonary edema.

SIGNS AND SYMPTOMS BY SYSTEM

Cardiovascular: Bradycardia, ventricular arrhythmias, hypotension. Tachycardia and disseminated intravascular coagulation may occur.

Respiratory: Bronchoconstriction, profuse bronchial secretions (bronchorrhea) with dyspnea, upper airway irritation, tightness of the chest, and paralysis of respiratory muscles. Chemically mediated pulmonary edema.

CNS: CNS depression and coma. Headache, slurred speech, dizziness, weakness, loss of muscle coordination, muscle fasciculations, and seizures.

Gastrointestinal: Nausea, vomiting, diarrhea, abdominal cramps, excessive salivation, urination, and defecation.

Eye: Lacrimation, blurred vision, and constricted pupils.

Skin: Pale, cyanotic skin with excessive diaphoresis.

Other: Classic SLUDGE syndrome (salivation, lacrimation, urination, defecation, GI pain, and emesis). May range from flu-type symptoms to anxiety, seizures, and coma. Symptoms may be broken down into muscarinic effects (classic SLUDGE syndrome, cardiac effects, constricted pupils, bronchoconstriction, pulmonary edema) and nicotinic symptoms (muscle fasciculations, tachycardia, hypertension, respiratory paralysis). If dermal absorption and nicotinic receptor sites are the primary site of stimulation, the SLUDGE syndrome may not be clinically present; instead, cardiovascular collapse may

be the primary clinical manifestation. Cholinesterase inhibitor exposures may be accumulative. Symptoms in children may differ from those found in adults. Pediatric signs and symptoms include CNS depression, flaccid muscle tone, dyspnea, and coma. Many of these products use a hydrocarbon as a solvent vehicle, identify the solvent, and refer to the appropriate guideline for related toxic effects.

SYMPTOM ONSET FOR ACUTE EXPOSURE

Immediate

Depending on absorption rate, some symptoms possibly delayed

CO-EXPOSURE CONCERNS

Organophosphates

Other carbamates

Insecticide solvent vehicle

THERMAL DECOMPOSITION PRODUCTS INCLUDE

Carbon monoxide

Methylamine

Methyl isocyanate

Nitrogen oxides

MEDICAL CONDITIONS POSSIBLY AGGRAVATED BY EXPOSURE

Respiratory disorders

Neurological 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.
- Products are highly absorbable, decontamination is critical.
- Discard all leather products.
- 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.
- Aggressive airway management may be needed.
- Watch for signs of respiratory insufficiency and assist ventilations if necessary.
- 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).
- Monitor for pulmonary edema and treat if necessary (refer to pulmonary edema 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 and 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 unconscious patient.
- Positive-pressure ventilation techniques with a bag-valve-mask device may be beneficial.
- Monitor cardiac rhythm and treat arrhythmias if necessary (refer to cardiac protocol in Section Three).
- Start an IV with D_5W TKO. Use lactated Ringer's if signs of hypovolemia are present.
- For hypotension if signs of hypovolemia are present, administer fluid cautiously. Watch for pulmonary edema (refer to shock protocol in Section Three).
- Administer atropine. Correct hypoxia before administration (refer to atropine protocol in Section Four).
- In severely poisoned patients, administer pralidoxime chloride (2-PAM). DIRECT PHYSICIAN ORDERS ONLY (refer to pralidoxime chloride protocol in Section Four).
- Treat seizures with adequate atropinization and correction of hypoxia. Rarely is diazepam necessary (refer to seizure 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.
- Both plasma and red blood cell acetylcholinesterase levels should be obtained. Do not delay therapeutic interventions pending laboratory results.
- 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.
- Depending on clinical symptoms, 2-PAM is most effective if administered within 24 to 48 hours after exposure. Use of 2-PAM is controversial because of self-release of carbamate-acetylcholinesterase bond within 48 hours of poisoning. (Refer to pralidoxime chloride protocol in Section Four).
- Obtain toxicological consultation as necessary.

SPECIAL CONSIDERATIONS

- Succinylcholine, other cholinergic agents, and aminophylline are contraindicated.
- Preservative-free atropine should be used to avoid toxicity from preservative agents.
- Mydriasis may occur early in the administration of atropine; however, the end point for atropine administration is the drying of pulmonary secretions.

Nicotine and Related Compounds

51

SUBSTANCE IDENTIFICATION

Found as a colorless-to-brown liquid plant alkaloid with a tobacco-like odor. Found in tobacco products, pesticides, and insecticides. Used in tanning processes.

ROUTES OF EXPOSURE

Skin and eye contact
Inhalation
Ingestion
Skin absorption

TARGET ORGANS

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

LIFE THREAT

Respiratory arrest and cardiac standstill. The alkaloid demonstrates stimulant and depressive action effects.

SIGNS AND SYMPTOMS BY SYSTEM

Cardiovascular: Initial hypertension and bradycardia, followed by tachycardia and hypotension. Cardiac standstill and paroxysmal atrial fibrillation.
Respiratory: Rapid, deep respirations followed by dyspnea, increased bronchial secretions, and paralysis of the respiratory muscles leading to respiratory arrest.
CNS: Agitation followed by headache, dizziness, confusion, muscle tremors/weakness, fasciculations, hyporeflexia, incoordination, auditory, and visual disturbances. Seizures.
Gastrointestinal: Nausea, vomiting, diarrhea, abdominal pain, salivation, and burning pain in the throat/mouth.
Eye: Chemical conjunctivitis and lacrimation. Pupils generally constricted (miotic) at first, then dilated (mydriatic).
Skin: Dermatitis and cyanosis.
Metabolism: Respiratory acidosis.

SYMPTOM ONSET FOR ACUTE EXPOSURE

Immediate
Initial stimulation followed by depression

CO-EXPOSURE CONCERNS

Chlorinated hydrocarbon insecticides
Other insecticides, pesticides

THERMAL DECOMPOSITION PRODUCTS INCLUDE

Carbon dioxide
Carbon monoxide
Nitrogen oxides

MEDICAL CONDITIONS POSSIBLY AGGRAVATED BY EXPOSURE

Central nervous system disorders
Cardiovascular 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
- 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.
- Aggressive airway management may be needed.
- Watch for signs of respiratory insufficiency and assist ventilations if necessary.
- Administer oxygen by nonrebreather mask at 10 to 15 L/min.
- 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 or in respiratory arrest.
- Monitor cardiac rhythm and treat arrhythmias if necessary (refer to cardiac protocol in Section Three).

- Start an IV with D_5W TKO. Use lactated Ringer's if signs of hypovolemia are present. Watch for signs of fluid overload.
- 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.
- 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.
- Atropine may be administered for signs and symptoms of parasympathetic effects (respiratory tract secretions, bradycardia, bronchoconstriction). Adult dosage: 0.5 mg titrated to effect. Correct hypoxia before giving atropine (refer to atropine protocol in Section Four).
- Obtain toxicological consultation as necessary.