J01: Approach to Chemical or Toxic Exposures

Robert MacMillan and Mike Sugimoto (ed)

Updated: May 27, 2021
Reviewed: March 01, 2021

Introduction

This clinical practice guideline is intended to provide general advice for paramedics and EMRs/FRs managing toxic or chemical exposures where the substances involved can be classified as irritants, asphyxiants, acids, or alkalis.

Separate practice guidelines exist for specific agents:

- → J02: Carbon Monoxide
- → J03: Cyanide
- → J04: Hydrogen Sulfide
- → J05: Organophosphates and Carbamates

Essentials

- The safety of paramedics and EMRs/FRs is paramount. Every patient's need for decontamination must be carefully evaluated, and measures taken to control or eliminate ongoing contamination hazards. Do not attempt to convey patients who have been insufficiently decontaminated.
  - CliniCall consultation required prior to undertaking decontamination procedures.
- Under no circumstances will BCEHS paramedics or EMRs/FRs provide care in hot or warm zones.
- Avoid contaminating ambulances or downstream health care providers and facilities: ensure that an appropriate decontamination is completed prior to loading a patient for conveyance. Downstream contamination can cause significant systemic disruption and must be avoided.
- Chemical exposures require a collaborative approach between agencies. Paramedics and EMRs/FRs will need to rely on the expertise of other responders at these scenes.
- The vast majority of toxic or chemical exposures can be managed symptomatically. Very few agents have specific out-of-hospital treatments. Supportive basic care (airway control, support for oxygenation and ventilation, and management of hypotension) are often more important than antidote administration.
- Apply a staged approach to all aspects of management, including airway control.

General Information

In all cases of chemical exposure, consultation with CliniCall for decontamination requirements and care planning is required. If there is uncertainty over the need for decontamination, the patient should undergo dry decontamination and then be re-assessed. Collaborate with other providers on scene.

Pulmonary Irritants

These agents primarily affect the respiratory tract and mucosal membranes. They include industrial chemicals like chlorine, ammonia, and phosgene. Mixing of household cleaning products, such as bleach and toilet bowl cleaners, can result in the production of irritant gases. Their effects vary depending upon their solubility in water. Upon contact with the mucosal membranes, they tend to dissociate into associated acids or bases, producing irritation. Bronchospasm is common, and severe exposures can result in non-cardiogenic pulmonary edema.

Asphyxiants

Asphyxiants are primarily gases whose dangerous properties relate to their ability to displace oxygen from a space. As the oxygen concentration falls, mental acuity among affected individuals begin to decrease while coordination and balance also decline. Loss of consciousness occurs at concentrations below 10%, and death can occur quickly where oxygen concentrations are below 6%. Patients who are removed from oxygen-deficient environments can be confused, agitated, combative, or comatose – all related to hypoxia.

Many asphyxiants have no warning properties such as taste, odour, or colour. Some of these gases are flammable

or explosive. Examples include hydrogen, helium, ethane, ethylene, nitrogen, neon, carbon dioxide, argon, acetylene, methane, propane, and propylene. Exposure to an asphyxiant does not generally require decontamination; where decontamination is required, dry decontamination and removal of clothing will generally suffice. If patients are swiftly removed from an oxygen-deficient environment, recovery can be rapid; prolonged exposure to hypoxic environments can lead to irreversible end-organ damage.

Acids

Widely used in both household and industrial applications, acids can be found in products as diverse as toilet bowl cleaners, drain cleaners, metal polishes, electroplating solutions, descaling solutions, and battery fluid. Exposure to acids generally involves splashes to the skin or into the eyes resulting in corrosive burns; ingestion of acid solutions or inhalation of acid fumes occurs occasionally.

All patients who have been exposed to acids must be decontaminated. The most effective method is to remove clothing and flush with copious amounts of running water. Acids attack proteins in tissue, causing a coagulation necrosis and inflammation; airway compromise may occur and should be managed conservatively. Bronchospasm should be treated as required.

Alkalis

Like their acid counterparts, alkaline corrosives are found in numerous household and industrial products and processes. Common examples include drain and oven cleaners, detergents, bleaches, and hair care products. "Lye" and "caustic soda" both refer to any strong alkali, generally either sodium hydroxide, potassium hydroxide, or a carbonate compound. Alkaline corrosives disrupt the lipid membranes of tissues, causing significant damage. As with other substances discussed in this guideline, the degree of damage depends heavily on the concentration of the substance, the duration of contact, and the total time of exposure.

These patients must be decontaminated. Brush off powdered material before removing clothes and flushing with water. Skin may feel "soapy" during flushing; continue flushing until the soapiness subsides. In cases of ingestion, and in consultation with CliniCall, consider giving 100-200 mL of milk or water to dilute the substance, but do not give in cases of nausea or vomiting.

Supraglottic airway devices are contraindicated due to potential ingestion of caustic substances.

Interventions

First Responder

Scene control:

- Protect responders and the public; isolate large-scale incidents, such as transportation accidents, according to the initial protective distances in the Emergency Response Guidebook (orange book)
- Stage in a safe environment until the scene is sufficiently controlled
- Conduct ongoing assessment and gather collateral information, such as medications and identification documents
- Establish ingress and egress routes from the patient's location
- Communicate patient deterioration to follow-on responders
- Assess and communicate the need for additional resources; identify the number of patients affected
- Ensure appropriate decontamination is performed prior to any patient assessment
  - → PR05: Patient Decontamination
- Apply a staged approach to the management of the airway, oxygenation, and ventilation
  - → B01: Airway Management
- Apply supplemental oxygen as required
  - → A07: Oxygen Administration
- Ventilate using bag-valve mask if respirations are inadequate or absent
- Do not attempt to remove clothing that has frozen to the skin; thaw first with warm water
- Flush skin and mucous membranes exposed to chemical agents with copious amounts of warm water; flushing should be done for at least 15 minutes and may take place concurrently with decontamination

- If eye exposure has taken place, gently remove contact lenses if not adherent to the cornea; flush eyes with water; do not attempt to open eyelids frozen shut by exposure to cryogenic liquids

Emergency Medical Responder – All FR interventions, plus:

CliniCall consultation required to discuss initial steps and plan for care of affected individuals.

General patient management:

- Use appropriate pharyngeal adjuncts (NPA/OPA) where required
  - → PR07: Nasopharyngeal Airways
- Administer high flow oxygen
  - → A07: Oxygen Administration

Primary Care Paramedic – All FR and EMR interventions, plus:

- Continue to apply a staged approach to airway management
  - → PR09: Continuous Positive Airway Pressure
  - → PR10: Positive End Expiratory Pressure
- Do not use supraglottic airway devices in cases of caustic (acid or alkaline) ingestion
- Consider the use of CPAP for patients who meet the appropriate criteria
- Consider the use of PEEP with a bag-valve mask to support oxygenation in patients whose respirations are inadequate
- Consider treating bronchospasm as required
  - → B03: Bronchospasm and Asthma
- Consider pain management
  - → E08: Pain Management
- For ongoing hypotension, consider vascular access and fluid administration to a systolic blood pressure of 90 mmHg
  - → D03: Vascular Access

Advanced Care Paramedic – All FR, EMR, and PCP interventions, plus:

- Continue to apply a staged approach to airway management
  - Endotracheal intubation is unlikely to be required in most cases; graded application of interventions will support oxygenation and ventilation in the majority of patients

J02: Carbon Monoxide

Robert MacMillan

Updated: May 27, 2021
Reviewed: March 01, 2021

Introduction

Carbon monoxide (CO) is a colourless, odourless, tasteless, non-irritating gas produced by the incomplete combustion of carbon-containing material such as gasoline, heating fuels, propane, oil, wood, and coal. Unintentional CO poisoning commonly results from inhalation of smoke from house fires, automobile exhaust, flue gas from furnaces or stoves, exhaust gas produced by outboard motors, ice-resurfacing machines, and barbecues used in poorly ventilated areas. Fatalities have been reported in those swimming near the engine exhaust outlets of boats and where gas-powered stoves and generators are misused as an indoor heat source.

Natural gas and propane do not contain CO, but can produce CO if burned without enough air. Methylene chloride, a common ingredient in paint stripper, can be metabolized to CO in the body after exposure.

Tobacco smokers have chronically elevated carboxyhemoglobin levels (see Toxic Dose).

Essentials

- Paramedics and EMRs must contact CliniCall regarding care planning for any suspected or confirmed CO poisoning.
- Remove patients to ambient air prior to assessment.
- Decontamination is not required for patients exposed to CO only. Patients who are removed from a house fire require a dry decontamination (i.e., removal of clothing) as a minimum measure before being loaded in an ambulance.
- High flow oxygen and supportive care is the treatment for all patients exposed to CO.
- ACP resources should be requested, if available, to measure carboxyhemoglobin (COHb) levels.
- Fire departments can measure CO levels inside buildings and this should be accomplished where possible.
- Standard pulse oximetry can be misleading in patients who have been exposed to CO; these devices are only able to detect oxygenated and deoxygenated hemoglobin and not any other form (such as carboxyhemoglobin or methemoglobin).

Additional Treatment Information

- Clinical effects of CO:
  - An elevated COHb level is diagnostic of exposure but may not reflect severity of poisoning or potential for development of delayed neurological sequelae. A more useful method of assessing the level of exposure *may be* to divide clinical signs into categories of severity. The category should be based on early symptoms.
    - Mild: throbbing temporal headache; dizziness; nausea and vomiting; blurred vision
    - Moderate: impaired thinking; confusion; severe headache; syncope or brief loss of consciousness; tachycardia; chest pain; dyspnea; tachypnea; weakness
    - Severe: myocardial ischemia; dysrhythmias; hypotension; cardiac arrest; respiratory failure; seizures; coma

Referral Information

Patients who have COHb levels between 3-10%, and who are symptomatic, require conveyance to hospital. Asymptomatic patients with COHb levels between 3-10% may not need conveyance, provided:

- The patient is with a responsible adult
- There is no history of ischemic heart disease
- The building is cleared of CO
- The patient is not pregnant
- There is no history of syncope

Exposures > 10% require conveyance.

NB: these criteria depend on the ability to measure COHb, which is only available to ACP/CCP units in British Columbia. Point-of-care CO-oximetry can be unreliable, and in-hospital arterial blood gas sampling can reveal significant discrepancies in the amount of COHb in a patient's blood. Paramedics and EMRs/FRs should, on the whole, be biased in favour of conveying patients with potential CO exposure to hospital for observation and additional evaluation.

General Information

- CO is readily absorbed after inhalation and crosses the placenta. The elimination half-life of CO is 4-5 hours breathing room air, 1-2 hours breathing 100% oxygen, and approximately 20 minutes with hyperbaric oxygen (2.5 atm).
- Hyperbaric oxygen (HBO) is a therapeutic option for treatment of CO poisoning. HBO produces a 10-fold increase in the amount of oxygen dissolved in blood, increases oxygen delivery to hypoxic tissues, and enhances CO elimination. HBO may also inhibit secondary cell damage (lipid peroxidation, mitochondrial dysfunction).
  - Complications of HBO therapy are infrequent. Ear and sinus pain (common) are managed with decongestants and surgical myringotomy in a small number of patients. Confinement anxiety, pulmonary barotrauma, and oxygen toxicity seizures occur rarely.
  - Despite several prospective trials examining HBO in preventing delayed neurologic sequelae, it remains unclear which patients clearly benefit from HBO or which clearly have no potential for benefit. Each patient must be assessed individually, evaluating potential benefits and risks. The following must be considered: current clinical status; time since exposure; acute vs. chronic exposure; risk of conveyance, travel time to HBO chamber; concomitant diseases; and pregnancy status. Decision may be made in consultation with the poison control centre or the hyperbaric unit.
  - Patients who **may likely** benefit from HBO treatment for CO poisoning include those with:
    - Neurologic signs: altered mental status; coma; cerebellar dysfunction; seizures
    - History of loss of consciousness
    - Pregnant patient with COHb level > 20%
  - Patients who may possibly benefit from HBO treatment for CO poisoning include those with:
    - Myocardial ischemia or cardiac dysrhythmias
    - Metabolic acidosis
    - Older patients
    - Asymptomatic patients with COHb levels > 25%
  - CO poisoning can result in permanent neurologic damage and death. CO poisoning is one of the leading causes of death worldwide; however, death is uncommon in patients who reach medical care. The major goal of treatment is prevention of delayed neurologic sequelae in survivors.
  - The mechanism of toxicity is complex and not fully understood. Toxicity is a result of hypoxia, ischemia, and direct cellular damage. CO binds to heme proteins, impairing normal oxygen function. CO-associated nitric oxide release may enhance oxidative and inflammatory injury to the brain. CO also has a direct effect on cellular respiration by inhibiting the activity of cytochrome oxidase and can provoke a metabolic acidosis.
  - Hemoglobin has an affinity for CO that is 200-250 times greater than its affinity for oxygen. COHb is formed, displacing oxygen from hemoglobin and producing a leftward shift in the oxyhemoglobin dissociation curve resulting in decreased oxygen delivery to tissues and causing hypoxia. Myoglobin is affected similarly, with its CO affinity being 60 times greater. Impaired oxygen delivery may cause myocardial ischemia, resulting in dysrhythmias and systemic hypotension.
  - The toxic dose of CO is highly variable. The degree of poisoning is dependent on the concentration of CO in the inspired air, the duration of exposure, the level of activity among those exposed, and underlying patient health. Infants, patients with pre-existing cardiovascular or lung disease, anemia, and in utero fetuses are more susceptible to CO.
  - Normal COHb blood levels in nonsmokers can be up to 2%. Smokers of 2-3 cigarette packages per day may have COHb levels as high as 7-9%.
  - Exposure to 545 ppm for 10 minutes has produced headache; inhalation of 5000 ppm for 5 minutes has been reported to be fatal.
  - Automobile exhaust may contain up to 100,000 ppm (10%) CO.

Interventions

First Responder

- Have patient(s) removed from exposure
- Decontaminate as required if fire was source of CO
  - → PR05: Patient Decontamination
- Keep the patient warm and protect from further heat loss
- Place the patient in a position of comfort, as permitted by clinical condition
- All patients should receive 100% oxygen through a non-rebreather face mask
  - → A07: Oxygen Administration
- Protect airway and assist ventilations as needed
  - → B01: Airway Management

Emergency Medical Responder – All FR interventions, plus:

CliniCall consultation required to discuss care planning for all patients suspected of carbon monoxide poisoning.

- Monitor vital signs; pulse oximetry is not reliable in patients with CO poisoning

Primary Care Paramedic – All FR and EMR interventions, plus:

- Consider IV access
  - → D03: Vascular Access and Fluid Administration
  - Consider fluid bolus to correct hypoperfusion or hypotension if clinically indicated

Advanced Care Paramedic – All FR, EMR, and PCP interventions, plus:

- Maintain fluid and electrolytes; treat severe acidosis; a slight acidosis may be beneficial by shifting the oxyhemoglobin dissociation curve to the right
  - → D03: Vascular Access
- Avoid alkalosis
- Administer glucose as prophylaxis against hypoglycemia (intracellular glucose may be decreased even in presence of normal or elevated blood glucose)
  - → E01: Hypoglycemia and Hyperglycemia
- Obtain COHb level if patient was exposed to smoke from fire; consider concurrent cyanide toxicity
  - In *mild* exposures, 100% oxygen should continue until patient is asymptomatic and COHb levels are < 5-6%
  - To protect fetus, pregnant patients not receiving HBO may need to continue 100% oxygen 5 times longer than time required to reduce maternal COHb to < 5-6%
  - In *moderate and severely* poisoned patients who are not candidates for HBO, 100% oxygen may need to be continued for 24 hours after symptoms resolve
- Hypotension unresponsive to IV fluids may require vasopressors
- Seizures should be treated with IV benzodiazepines
  - → F02: Seizures
- Treat cerebral and pulmonary edema supportively

Critical Care Paramedic – All FR, EMR, PCP, and ACP interventions, plus:

- Monitor electrolytes, glucose, renal and liver function, creatine kinase
- Monitor ECG, troponin, blood gases and serum lactate levels in symptomatic patients
- Obtain methemoglobin level if patient was exposed to smoke from fire; consider concurrent cyanide toxicity
- Consider transport to a hyperbaric unit.

Evidence Based Practice

Carbon Monoxide

Supportive

- Oxygen

Neutral

- Direct Transport To Hyperbaric Facility

Against

References

1. Alberta Health Services. AHS Medical Control Protocols: Carbon Monoxide Poisoning. 2020. [Link]

J03: Cyanide

Robert MacMillan

Updated: May 27, 2021
Reviewed: March 01, 2021

Introduction

Cyanide is a molecule consisting of a carbon atom triply bonded to a single nitrogen atom. It can form compounds which are also known as cyanides. It is both naturally occurring and synthetic with many cyanide-containing compounds resulting in powerful, fast acting poisons.

Hydrogen cyanide is a colourless gas with a faint, bitter, almond-like odor. Sodium cyanide and potassium cyanide are both white solids with similar odours in damp air. Cyanide salts and hydrogen cyanide are used in electroplating, metallurgy, the production of organic chemicals, photography, plastics manufacturing, the fumigation of ships, and some mining processes. Fires involving modern building materials, plastics, and furnishings can also produce large amounts of cyanide, and individuals exposed to the smoke from these fires can have significant cyanide exposures.

Essentials

- OlniCall consultation required when attending cases of suspected cyanide exposure.
- Rescue of unconscious victims exposed to cyanide gas must only be done by trained personnel equipped with self-contained breathing apparatuses and protective clothing.
- Patients must be decontaminated. Remove clothing to limit off-gassing and secondary contamination.
- Severe, acute cyanide poisoning is usually associated with rapid onset of central nervous system symptoms, including unconsciousness and seizures. Cardiovascular effects, such as hypotension and tachycardia, and metabolic acidosis, are common.
- Hydrogen cyanide and the inorganic cyanide salts rapidly produce symptoms following acute exposure. Death may occur within minutes. Exposure to cyanide-containing compounds may result in a delayed onset of symptoms.
- Hydroxocobalamin is the first-line antidote to cyanide poisoning.

Additional Treatment Information

- Topical exposure to concentrated solutions of cyanide salts can cause skin burns as well as systemic toxicity. Skin flushing may be observed from systemic effects. Remove and dispose of contaminated clothing. Flush skin and eyes thoroughly with soap and water and treat symptomatically as for ingestion.
- Inhalation of cyanide-containing gases produce respiratory tract irritation. Massive exposure may cause a sudden loss of consciousness and death from respiratory arrest within minutes. Cyanogen chloride can cause delayed pulmonary edema.

Referral Information

Asymptomatic patients should be monitored for at least six hours following acute exposure. The monitoring period should be extended to at least 24 hours following exposure to nitriles or cyanide-releasing compounds. Conveyance is mandatory.

General Information

- Signs and symptoms of cyanide toxicity include:
  - Tachycardia, mild transient hypertension progressing to hypotension, bradycardia, and cardiovascular collapse
  - Tachypnea is common initially with progression to respiratory depression and respiratory arrest; pulmonary edema may develop
  - Headaches, anxiety, dizziness, agitation and confusion are common in early stages; patients may become obtunded or seize
  - Nausea and vomiting may develop; ingestion of caustic, alkaline cyanide salts may cause gastrointestinal

bleeding
    - Metabolic acidosis with hyperlactatemia is characteristic of severe cyanide poisoning; hyperglycemia may also occur
- Cyanide inhibits the activity of cytochrome oxidase A3 in the mitochondria, preventing aerobic respiration. The resulting shift to anaerobic metabolism produces an excess of lactate. Effects are most prominent in brain and cardiovascular tissues.
- Cyanides are rapidly absorbed by ingestion, inhalation, and through contact with mucosal membranes. Symptoms may be seen within seconds to minutes of exposure.
- Air concentrations of 200-300 ppm of hydrogen cyanide may be rapidly fatal.
- The "bitter almond" odour of hydrogen cyanide is not a reliable indicator of danger – many individuals are unable to detect this odour.
- The estimated lethal dose to an adult is 50 mg of hydrocyanic acid and 200-300 mg of an inorganic cyanide salt.
- Patients have survived > 1 g potassium cyanide ingestion with prompt antidote therapy.

Interventions

First Responder

- Remove patient and decontaminate as required
    - Remove and dispose of clothing
    - Flush exposed skin and mucosal membranes with soap and water
    - → PR05: Patient Decontamination
    - If eyes are involved, flush with a gentle stream of water for at least 15 minutes
- Keep the patient warm and protect from further heat loss
- Place the patient in a position of comfort, as permitted by clinical condition
- Protect the airway and assist ventilations as necessary
    - → B01: Airway Management
- Provide supplemental oxygen via non-rebreather face mask
    - → A07: Oxygen Administration

Emergency Medical Responder – All FR interventions, plus:

CliniCall consultation required when attending cases of suspected cyanide exposure.

- Convey with notification

Primary Care Paramedic – All FR and EMR interventions, plus:

- Consider vascular access and treatment of hypotension
    - → D03: Vascular Access

Advanced Care Paramedic – All FR, EMR, and PCP interventions, plus:

- Consider push-dose EPINEPHrine for hypotension refractory to fluids
- Control seizures if necessary
    - → F02: Seizures

Critical Care Paramedic – All FR, EMR, PCP, and ACP interventions, plus:

- Hydroxocobalamin
- Correct metabolic acidosis
- Seizures refractory to benzodiazepines should be managed with barbiturates

References

1. Agency for Toxic Substances and Disease Registry (ATSDR). [Link]

2. ATSDR - Division of Toxicology and Human Health Sciences (DTHHS). 2018. [Link]
3. British Columbia Drug and Poison Information Centre. [Link]

J04: Hydrogen Sulfide

Robert MacMillan

Updated: June 02, 2021
Reviewed: March 01, 2021

Introduction

Hydrogen sulfide (H_2S) is a colourless gas with a characteristic odor of rotten eggs that is a product of the decomposition of sulfur-containing organic materials. It occurs naturally in fossil fuel deposits, sulfurous rocks, and is also released from hot tar and asphalt. Accidents involving H_2S have occurred in mines, caves, oil fields, petroleum refineries, sewers, liquid manure storage tanks, agricultural facilities, and the cargo holds of fishing boats.

Chemical suicides using common household products to create H_2S gas are becoming more common.

The toxicity of H_2S depends on the concentration and the duration of exposure. Most deaths occur at the scene as a result of respiratory paralysis, also known as "knockdown." Trauma may also occur as a result of falls following a loss of consciousness.

Essentials

- CliniCall consultation required when attending cases of suspected H2S exposure.
- Prompt rescue and treatment can save lives. Rescue of unconscious victims must only be undertaken by trained personnel equipped with self-contained breathing apparatuses and appropriate protective clothing. Atmospheric gas monitoring is mandatory.
- Decontamination is required. Remove and dispose of clothing.
- Inhalation of high concentrations of H_2S causes immediate respiratory paralysis and a rapid loss of consciousness, followed shortly after by death from asphyxia.
- Patients who are ventilated immediately following rescue often recover completely. Those who remain unconscious for longer periods of time are at risk for permanent hypoxic brain injuries.

Additional Treatment Information

- Early endotracheal intubation and mechanical ventilation with high concentrations of oxygen is recommended in patients with central nervous system depression or respiratory distress.
- Patients with respiratory paralysis may not begin breathing spontaneously for hours.
- Aspiration and pulmonary edema may develop in severe cases.

Referral Information

Patients who are asymptomatic should be observed for at least several hours following exposure.

General Information

- H_2S is highly toxic. The characteristic odour of the gas is an unreliable predictor of danger; prolonged exposure to low concentrations of H_2S, or brief exposures to higher concentrations, results in olfactory fatigue and renders individuals insensitive to the smell.
- At concentrations between 50-100 ppm, H_2S is irritating to lungs, mucosal membranes, and eyes. Prolonged exposure at this level may cause pulmonary edema.
- Concentrations > 500 ppm may produce severe toxicity within minutes. A single breath at concentrations between 800-1,000 ppm may be rapidly fatal.
- The toxicity of H_2S is due to its ability to paralyze respiratory muscles and produce profound hypoxia.

Interventions

First Responder

- Decontaminate patients in open air
  - → PR05: Patient Decontamination
  - Flush exposed skin and eyes with warm water
- Keep the patient warm and protect from further heat loss
- Place the patient in a position of comfort, as permitted by clinical condition
- Provide supplemental oxygen and ventilation as required; use high-flow devices; provide airway management as required
  - → B01: Airway Management
  - → A07: Oxygen Administration

Emergency Medical Responder – All FR interventions, plus:

- CliniCall consultation required when attending cases of suspected H2S exposure for hazard control and care planning purposes.

Primary Care Paramedic – All FR and EMR interventions, plus:

- Consider CPAP or PEEP for patients with developing pulmonary edema
  - → PR09: Continuous Positive Airway Pressure
  - → PR10: Positive End Expiratory Pressure
- Consider vascular access and fluid replacement for hypotension
  - → D03: Vascular Access

Advanced Care Paramedic – All FR, EMR, and PCP interventions, plus:

- Apply a staged approach to oxygenation and ventilation in cases of significant CNS depression
- Control seizures as required
  - → F02: Seizures

Critical Care Paramedic – All FR, EMR, PCP, and ACP interventions, plus:

- Consider sodium nitrite
- Contact DPIC (1-800-567-8911) or ETP for additional guidance

J05: Organophosphates and Carbamates

Robert MacMillan

Updated: June 02, 2021
Reviewed: March 01, 2021

Introduction

Organophosphates and carbamates are groups of related chemicals commonly used as pesticides to control insects. Some preparations are approved for veterinary use to control fleas and other parasites. They are commonly formulated as dusts, granules, emulsions, suspensions, or solutions. Concentrates are often formulated in a petroleum distillate base.

Clinically, poisonings from organophosphates and carbamates are similar to each other, resulting in the inhibition of acetylcholinesterase, causing stimulation of muscarinic and nicotinic receptors. Carbamate toxicity is generally of shorter duration as its effect on acetylcholinesterase is reversible.

Deaths from these products occur as a result of acute respiratory failure.

Essentials

- Paramedics and EMRs must contact CliniCall to discuss the case, ideally prior to arrival on scene.
- Decontamination requirements can be complex and must be completed prior to moving the patient to the ambulance for treatment.
  - → PR05: Patient Decontamination
- Attempt to identify the name and amount of the substance the patient was exposed to, including the pesticide control number, WHMIS information, or photo of the label. This can be relayed to CliniCall for additional information.
- Health care staff, including paramedics and EMRs/FRs, should wear protective clothing when handling contaminated clothing and grossly contaminated patients.
- Treatment should be directed at decontamination, administration of antidotes where available, and support for oxygenation and ventilation.

Additional Treatment Information

- Activated charcoal may be considered in hospital for up to one hour post-ingestion.
- High doses of atropine, in conjunction with pralidoxime, may be required in cases of severe organophosphate poisoning.

General Information

- Organophosphate pesticides include: acephate; azinphos-methyl; chlorpyrifos; diazinon; dichlorvos; dimethoate; fenthion; malathion; methamidophos; naled; phorate; propetamphos; terbufos; tetrachlorvinphos; and trichlorfon.
- Carbamates include: bendiocarb; carbaryl; carbofuran; formetanate; methomyl; oxamyl; and propoxur.
- Products may be labeled as "systemic" or "contact" – this refers to the action of the pesticide and whether it is taken into plant tissues. It has no bearing on human toxicity.
- The onset of symptoms is usually within minutes to hours after exposure. Symptoms may be delayed in the case of skin exposure.
- Symptoms can be divided into muscarinic effects (miosis, excessive sweating and bronchial secretions, bradycardia, hypotension) and nicotinic effects (mydriasis, tachycardia, fasciculations, muscle weakness, paralysis). Central nervous system effects can include headache, drowsiness, seizures, and unconsciousness.
  - Muscarinic receptors are predominantly in the parasympathetic nervous system whereas nicotinic receptors are primarily in the sympathetic nervous system.
- A useful mnemonic for organophosphate toxicity is SLUDGEM/BBB:
  - Salivation
  - Lacrimation

- Urination
- Defecation
- GI upset
- Emesis
- Miosis
- Bronchorrhea
- Bronchospasm
- Bradycardia

- The three "Bs" – bronchorrhea, bronchospasm, and bradycardia – are the most common causes of death in organophosphate and carbamate poisoning.

Interventions

First Responder

- Maintain a safe working environment:
- Decontaminate as per CliniCall, fire department, or hazardous materials specialist on scene
  - → PR05: Patient Decontamination
- Keep the patient warm and protect from further heat loss
- Place the patient in a position of comfort, as permitted by clinical condition
- Provide airway management and support for oxygenation and ventilation as required
  - → B01: Airway Management
  - → A07: Oxygen Administration

Emergency Medical Responder – All FR interventions, plus:

- CliniCall consultation required for guidance, ideally prior to arrival on scene.

Primary Care Paramedic – All FR and EMR interventions, plus:

- Consider extraglottic device for profoundly unconscious patients (may require removal for suctioning purposes)
  - → PR08: Supraglottic Airways
- Obtain vascular access
  - → D03: Vascular Access

Advanced Care Paramedic – All FR, EMR, and PCP interventions, plus:

- Manage seizures
  - → F02: Seizures
- Treat dysrhythmia and chest discomfort as per CPGs
  - → C01: Acute Coronary Syndromes
  - → C02: Bradycardia
  - → C03: Narrow Complex Tachycardia
  - → C04: Wide Complex Tachycardia
- Consider magnesium sulfate for management of ventricular tachycardia (likely caused by QTc prolongation from organophosphates)
- Atropine:
  - Double the dose every five minutes until effect is seen
  - The goal is to control secretions and correction of significant bradycardia and hypotension; secretions respond more slowly than bradycardia
  - Atropine will reverse muscarinic symptoms but will not alter nicotinic effects

Critical Care Paramedic – All FR, EMR, PCP, and ACP interventions, plus:

- Intubate if necessary
- Provide mechanical ventilation as required
- Consider obtaining pralidoxime from a hospital while en route to call:
  - Call ETP prior to pralidoxime
  - Do not administer pralidoxime without concurrently giving atropine
    - Adults:
      - Loading dose: 1 – 2 g IV infused over 30 minutes
      - May be given by direct IV injection over 5 minutes in severely poisoned patients
      - First dose may be given IM if IV access is not possible
      - Maintenance dose: continuous infusions preferred; optimal dose not established; 500 mg/hr is often recommended; consider 8-10 mg/kg/hr (as per WHO guidance); lower rates may be adequate; titrate to response
    - Children:
      - Loading dose: 20-50 mg/kg
      - May also be given by direct IV injection over 5 minutes, or IM if IV access cannot be obtained
      - Maintenance dose: 10-20 mg/kg/hr
    - Therapeutic endpoint: control of nicotinic symptoms
    - Treatment for 24-48 hours is usually sufficient in many cases; prolonged treatment (i.e., several weeks) may be required in severe poisoning
    - Discontinue when patients no longer require ventilatory support
  - Pralidoxime use in carbamate poisoning is controversial; indicated for use in mixed organophosphate/carbamate poisoning
  - Risk of serious adverse effects (laryngospasm, hypertension) increases with rapid IV administration
  - May precipitate myasthenic crisis in patients with myasthenia gravis; may cause toxicity of carbaryl, a carbamate insecticide
- May consider glycopyrrolate for organophosphate symptoms if atropine is ineffective or unavailable

Evidence Based Practice

Pesticide Poisoning

Supportive

Neutral

- Atropine

Against

J06: Radionuclear Incidents

Michelle Haig and Mike Sugimoto

Updated: May 27, 2021
Reviewed: March 01, 2021

Introduction

Radiological and nuclear incidents are related, but separate events.

In radiological events or accidents, individuals are exposed to radiation, or contaminated by radioactive material. This can occur as a deliberate act, as in the use of an explosive radiological dispersal device – which is any device that is designed to spread radioactive material around an area using either explosives or a compressed gas – or through exposure to radioactive material or generating devices, such as sealed sources, x-ray devices, or accelerators. Individuals exposed in these incidents may have no knowledge of their exposure until some time later.

Critical assemblies, or criticality events, occur when sub-critical masses of fissile material are brought together, inadvertently starting a chain reaction. This results in the creation of an unshielded nuclear reactor and produces significant amounts of radiation. There is no explosion, although substantial heat may be produced.

Nuclear incidents involve a chain reaction (fission) which can be accidental, or result from the intentional detonation of a nuclear weapon. Detonations are accompanied by widespread blast and heat. Exposed individuals will generally notice when this happens.

With the exception of deliberate weapons-related events (i.e., the bombings of Hiroshima and Nagasaki and national nuclear weapons tests), every radiation-related incident involving members of the public has been a radiological event. Critical assemblies have been limited to research and industrial sites.

Essentials

- Paramedics and EMRs must contact CliniCall to discuss the case, ideally prior to arrival on scene.
- Skin or wound contamination is rarely life threatening for patients or health care personnel.
- Removal of the outer layer of clothing and shoes typically reduces external contamination by 90%.
- The goal of decontamination is to remove as much contaminated material as possible without damaging the skin or creating adverse effects.
- Decontamination can be accomplished without radiological monitoring if necessary.
- Subsequent decontamination cycles may be necessary. There is no single target value for decontamination appropriateness for all circumstances. Generally, decontamination is "successful" when survey meters show less than 2 or 3 times the normal background radiation or when further efforts stop an increase in the count rate significantly.
- Internal contamination, and incorporation of radioactive materials into body tissues, may occur and require additional treatment.

General Information

Radiation Precautions

- Individuals who have been exposed to ionizing radiation, but who have not come into contact with radioactive material, are neither contaminated nor radioactive. They do not require radiation precautions.
- In patients with known or suspected external or internal contamination, paramedics and EMRs/FRs should don gown, masks, cap, boots, and gloves. The patient should be isolated to the maximal extent possible. Avoid touching surfaces or items unnecessarily.

Burns

- Both thermal and radiation burns can occur in radiological incidents. Thermal burns with radiation exposure are a "combined injury," for which the prognosis is worse than burn or radiation exposure alone.
- Cool burns as required. Be aware of the risk of hypothermia.
- Radiation burns may occur in patients undergoing radiation therapy or who have had extensive fluoroscopy

procedures. These do not benefit from cooling and have complex wound care requirements.

Acute Radiation Syndrome

- Individuals exposed to radiation will develop Acute Radiation Syndrome (ARS) only if all of the following requirements are met:
  - The radiation dose was high
  - The radiation dose was penetrating (i.e., it was able to reach internal organs, such as x-rays or gamma rays)
  - The person's entire body, or most of it, received the dose
  - The radiation was received in a short time, usually minutes; this is most common in industrial accidents and therapeutic misadventures
- There are four subsyndromes of ARS – hematopoietic, gastrointestinal, cutaneous, and neurovascular – and their severity will vary with dose and individual factors.
- High dose whole-body radiation exposure also produces clinically detectable effects in the lungs, liver, and kidneys. Usually, injury to these organs is detected long after ARS manifests itself; individuals would need to survive the earlier injuries for these types of injuries to become life threatening.
- Immune dysfunction, as part of the injury to the hematopoietic system, is also clinically significant if the dose and exposure is severe enough.

Interventions

First Responder

- Decontaminate patient
- → PR05: Patient Decontamination
- Provide supplemental oxygen as required
  - → A07: Oxygen Administration
- Treat life and limb threatening injuries
- Place patient in position of comfort
- Cool burns
- Provide wound care

Emergency Medical Responder – All FR interventions, plus:

- Paramedics and EMRs must contact CliniCall to discuss the case, ideally prior to arrival on scene.
- Provide supplemental oxygen to maintain $SpO_2 \geq 94\%$
  - → B01: Airway Management
  - → A07: Oxygen Administration
- Manage pain
  - → E08: Pain Management

Primary Care Paramedic – All FR and EMR interventions, plus:

- Consider vascular access
  - → D03: Vascular Access
- Manage nausea
  - → E07: Nausea and Vomiting

References

1. US Department of Health & Human Services. REMM - Radiation Emergency Medical Management. 2020. [Link]

J07: Beta Blocker Toxicity

Mike Sugimoto

Updated: May 01, 2024
Reviewed: May 01, 2024

Introduction

Beta blockers are widely used in the management of an extensive range of clinical problems, including hypertension, heart failure, migraine headaches, tremors, and aortic dissection. Although overdoses of these medications, either accidental or otherwise, occur infrequently, beta blocker toxicity is associated with significant morbidity and mortality. The primary mechanism for beta blocker toxicity is through the adrenergic blocking action of these medications. Some beta blockers, such as sotalol, propranolol, and acebutolol, have significant pro-arrhythmic tendencies.

Essentials

- As with all poisoning or overdoses, manage the airway and ensure adequate oxygenation and ventilation while a more comprehensive history is obtained.
- Search for and treat reversible causes: do not overlook other causes of the patient's symptoms.
- Out-of-hospital management of beta blocker overdose is limited and specific therapies should only be undertaken in consultation with CliniCall (see ACP interventions below). Rapid conveyance is indicated for virtually all patients.

General Information

- Consider the possibility of co-ingestion of other drugs in patients who are suspected of beta blocker toxicity, particularly calcium channel blockers, digoxin, clonidine, and cholinergic agents.
- Beta blocker toxicity is generally more severe in individuals with a pre-existing cardiovascular history.
- Patients who have overdosed on beta blocking drugs typically become symptomatic within two hours, and virtually all becoming symptomatic within six hours.
- The most common symptoms are bradycardia and hypotension. Myocardial depression and cardiogenic shock can develop in severe cases. Ventricular dysrhythmias are more common with propranolol and sotalol. Mental status changes, such as confusion, delirium, seizures, and unconsciousness, can occur at virtually any point.
- Respiratory depression has been reported. Bronchospasm and hypoglycemia, produced by the beta blockade, can complicate management.
- Possible electrocardiogram changes include PR elongation, QRS prolongation, and any bradydysrhythmia.

Interventions

First Responder

- Keep the patient warm and protect from further heat loss
- Place the patient in a position of comfort, as permitted by clinical condition
- Protect the airway and ensure adequate oxygenation and ventilation
  - → B01: Airway Management
- Provide supplemental oxygen as required
  - → A07: Oxygen Administration

Emergency Medical Responder – All FR interventions, plus:

- Provide supplemental oxygen to maintain SpO_2 ≥ 94%
  - → A07: Oxygen Administration
- Measure capillary blood glucose levels
- Initiate conveyance; consider intercept with additional resources

Primary Care Paramedic – All FR and EMR interventions, plus:

- Consider vascular access and fluid bolus; target systolic blood pressure > 90 mmHg; do not give more that 2 L of fluid
  - → D03: Vascular Access
- Correct hypoglycemia if present:
  - → E01: Hypoglycemia and Hyperglycemia

Advanced Care Paramedic – All FR, EMR, and PCP interventions, plus:

- Obtain and interpret 12-lead ECG
  - → PR16: 12-Lead ECG
- Treat bradycardia:
  - → C02: Bradycardia
  - Atropine; note that atropine may not reverse bradycardia or offer only partial recovery
  - Transcutaneous pacing may be required, though may be ineffective
- Manage seizures:
  - MIDAZOLam
  - See F02: Seizures for additional details
- If airway management is required, *aggressively* attempt to limit peri-procedural hypotension
- → PR18: Anesthesia Induction
- → PR23: Awake Intubation

CliniCall consultation required prior to initiation and to discuss suitability of any of the following therapies:

- Correct ventricular arrhythmias:
  - Consider calcium chloride.
  - Consider sodium bicarbonate in wide complex dysrhythmias
  - Consider magnesium sulfate (particularly in cases of sotalol-induced ventricular dysrhythmia)
  - Consider EPINEPHrine infusion, escalating in consultation with CliniCall; note that higher dose rates may be required to overcome competitive inhibition

Critical Care Paramedic – All FR, EMR, PCP, and ACP interventions, plus:

- Bradycardia
  - TVP
- Consider IV glucagon
- Consider IV calcium salts
- Consider IV vasopressor (epinephrine)
- Consider IV high-dose insulin and glucose
- Consider IV lipid emulsion therapy

Evidence Based Practice

Overdose-Poisoning

Supportive

- Activated Charcoal
- Naloxone-IM (Opiate OD)
- Naloxone-IN (Opiate OD)

- Naloxone-IV (Opiate OD)
- Naloxone-SQ (Opiate OD)
- Capnography
- Naloxone-Nebulized (Opiate OD)
- Oxygen
- Oxymetry Monitoring
- Sodium Bicarb (TCA OD)

Neutral

- Glucagon (Beta-Blocker OD)
- Treat & Release (Opiate OD)

Against

- Benzodiazepine antagonist (Benzo OD)

References

1. Barrueto F. Beta blocker poisoning. In UptoDate. 2020. [Link]

J08: Tricyclic Antidepressant Toxicity

Mike Sugimoto

Updated: June 02, 2021
Reviewed: March 01, 2021

Introduction

Although not as commonly used for their original purpose, tricyclic antidepressants (TCAs) remain in use for the treatment of depression and other conditions.

Essentials

- TCA overdose produces sedation, unconsciousness, and seizures. Tachycardias, including wide complex tachycardias, and hypotension are common.
- Patients who have overdosed on TCAs can deteriorate rapidly. Urgent conveyance with appropriate preparation should be undertaken.
- As with most poisonings or overdoses, care for TCA toxicity is primarily supportive. Protect the airway, provide supplemental oxygen, maintain effective ventilation, and support blood pressure as necessary.
- ECG monitoring can be helpful in identifying cardiac rhythm disturbances common to TCA overdose. Consider ACP intercept where available.
- Consider the possibility of co-ingestion of other medications or substances. Care more generally for the patient than for any particular poison.

Additional Treatment Information

- Patients who have overdosed on TCAs are frequently hypotensive. Fluid resuscitation should be initiated in patients who are significantly hypotensive; unmanaged hypotension is a primary cause of mortality in these patients.
- Sodium bicarbonate should be considered, in consultation with CliniCall, when the QRS interval exceeds 100 ms or the QRS morphology is grossly distorted.
- As a general rule, antiarrhythmics should be avoided in TCA overdose: their interactions with a disordered heart are unpredictable and most have been poorly studied. Magnesium *may* be an acceptable antiarrhythmic in the context of cardiac arrest, but should only be given in consultation with CliniCall.

General Information

- TCA overdoses carry several important clinical consequences; the most significant is the blockade of fast sodium ion channels in the heart.
- The clinical course of a TCA poisoning is unpredictable due to complexities with uptake from the gastrointestinal tract, bioavailability, and drug metabolism. Patients may initially appear well, but deteriorate rapidly and without warning.
- Signs of TCA poisoning typically include sedation, but may also feature confusion, delirium, and hallucinations. Anticholinergic effects, such as hyperthermia, flushing, and dilated pupils are common. Hypotension is the most ominous finding; the majority of patients who die from TCA overdose do so as a result of refractory, uncorrectable hypotension.
- ECG findings in TCA overdose include:
  - QRS > 100 ms
  - Deep S waves in leads I, aVL
  - Tall R waves in lead aVR
  - Tachycardias, including sinus tachycardia

Interventions

First Responder

- Keep the patient warm and protect from further heat loss
- Place the patient in a position of comfort, as permitted by clinical condition
- Protect the airway and ensure adequate oxygenation and ventilation
  - → B01: Airway Management
- Provide supplemental oxygen as required
  - → A07: Oxygen Administration

Emergency Medical Responder – All FR interventions, plus:

- Provide supplemental oxygen to maintain SpO_2 ≥ 94%
  - → A07: Oxygen Administration
- Support ventilation as required
- Obtain and measure capillary blood glucose
- Initiate conveyance; prepare for acute deterioration en route
- Consider intercept with additional resources

Primary Care Paramedic – All FR and EMR interventions, plus:

- Obtain vascular access and correct hypotension and hypoglycemia
  - → D03: Vascular Access
  - → E01: Hypoglycemia and Hyperglycemia

Advanced Care Paramedic – All FR, EMR, and PCP interventions, plus:

- Obtain and interpret 12-lead ECG
  - → PR16: 12-Lead ECG
- CliniCall consultation required prior to initiation and to discuss suitability of any of the following therapies:
  - Consider sodium bicarbonate; assess for QRS narrowing following administration
  - Consider push-dose EPINEPHrine for hypotension refractory to fluid bolus
  - Magnesium sulfate *may* be an acceptable antiarrhythmic in the context of cardiac arrest

Critical Care Paramedic – All FR, EMR, PCP, and ACP interventions, plus:

- Consider sodium bicarbonate infusion (37.5 mEq/hr) if initial bolus dose of sodium bicarbonate was effective at narrowing QRS complex
- Consider norepinephrine for refractory hypotension

Evidence Based Practice

Overdose-Poisoning

Supportive

- Activated Charcoal
- Naloxone-IM (Opiate OD)
- Naloxone-IN (Opiate OD)
- Naloxone-IV (Opiate OD)
- Naloxone-SQ (Opiate OD)
- Capnography
- Naloxone-Nebulized (Opiate OD)
- Oxygen

- Oxymetry Monitoring
- Sodium Bicarb (TCA OD)

Neutral

- Glucagon (Beta-Blocker OD)
- Treat & Release (Opiate OD)

Against

- Benzodiazepine antagonist (Benzo OD)

J09: Calcium Channel Blocker Toxicity

Mike Sugimoto

Updated: May 24, 2024
Reviewed: March 01, 2021

Introduction

Calcium channel blockers, commonly used to treat hypertension and cardiac dysrhythmias, have a significant risk of toxicity if used inappropriately.

Essentials

- As with most poisonings, out-of-hospital management options are limited. Protect the airway, ensure optimal oxygenation, support ventilation as necessary, and attempt to correct hypotension. Care more generally for the patient than for the specific suspected poison.
- Hypotension and bradycardia are common findings.
- Be aware of the possibility of co-ingestion of other medications or substances.
- Pre-existing heart disease and myocardial ischemia can cause symptoms similar to calcium channel blocker overdose and must be excluded.

Additional Treatment Information

- As a first-line treatment, a fluid bolus of 500 mL should be given to any patient suspected of having overdosed on calcium channel blockers who is hypotensive, and may be repeated as necessary up to 1 L.
- Atropine should be considered in patients who are bradycardic, repeated as necessary, up to a total dose of 3 mg.
- Intravenous calcium (either calcium chloride or calcium gluconate) may overcome the cardiovascular effects of calcium channel blockers. Intravenous administration of 1-2 grams can be provided over 10 minutes.

General Information

- Calcium channel blockers can be divided into two categories: the dihydropyridines, which block L-type calcium channels in the vasculature, and the non-dihydropyridines, which act on calcium channels in the myocardium.
  - The dihydropyridines include nifedipine, amlodipine, and felodipine. They are potent vasodilators and have limited effect on cardiac contractility or conduction. The non-dihydropyridines, diltiazem and verapamil, act more centrally and are more likely to directly affect cardiac output.
- In general, dihydropyridine drugs are more likely to cause arterial vasodilation and tachycardia, whereas diltiazem and verapamil tend to produce bradycardia and poor contractility.
- The changes in myocardial contractility may induce symptoms of heart failure. Carefully evaluate patients for signs of myocardial dysfunction, including shortness of breath and pulmonary edema.
- Patients who have overdosed on calcium channel blockers may have significant hyperglycemia. This is clinically insignificant, but may assist in diagnosis. Obtain and record a capillary blood glucose measurement.
- Epinephrine infusions may be required for patients whose hypotension and bradycardia are refractory to atropine and calcium. Profound calcium channel blocker toxicity may require significantly higher doses and dose rates than might otherwise be expected. Titrate drug doses to effect; be aware of arrhythmogenic potential.

Interventions

First Responder

- Keep the patient warm and protect from further heat loss
- Place the patient in a position of comfort, as permitted by clinical condition
- Protect the airway and ensure adequate oxygenation and ventilation
  - → B01: Airway Management

- Provide supplemental oxygen as required
  - → A07: Oxygen Administration

Emergency Medical Responder – All FR interventions, plus:

- Provide supplemental oxygen to maintain SpO_2 ≥ 94%
  - → A07: Oxygen Administration
- Obtain capillary blood glucose measurement
- Initiate conveyance; consider intercept with additional resources

Primary Care Paramedic – All FR and EMR interventions, plus:

- Obtain vascular access and correct hypotension
  - → D03: Vascular Access

Advanced Care Paramedic – All FR, EMR, and PCP interventions, plus:

- For bradycardia:
  - Atropine; repeated as necessary
  - → C02: Bradycardia
- CliniCall consultation required prior to initiating any of the following therapies.
  - Calcium chloride 1-2 g IV over 10 minutes
  - Consider push-dose EPINEPHrine or infusion for hypotension refractory to calcium chloride

Critical Care Paramedic – All FR, EMR, PCP, and ACP interventions, plus:

- Consider Norepinephrine
- Consider Glucagon IV 1-5 mg IVP (up to 15 mg)
- Consider lipid emulsion therapy 20% solution
  - Call ETP prior to lipid emulsion therapy
  - Bolus 1.5 ml/kg over 2 minutes
  - Infusion 1.5ml/kg over 60 minutes
- For bradycardia
  - Call ETP prior to TVP therapy
  - TVP
- Consider high dose insulin and glucose therapy

Evidence Based Practice

Overdose-Poisoning

Supportive

- Activated Charcoal
- Naloxone-IM (Opiate OD)
- Naloxone-IN (Opiate OD)
- Naloxone-IV (Opiate OD)
- Naloxone-SQ (Opiate OD)
- Capnography
- Naloxone-Nebulized (Opiate OD)
- Oxygen

- Oxymetry Monitoring
- Sodium Bicarb (TCA OD)

Neutral

- Glucagon (Beta-Blocker OD)
- Treat & Release (Opiate OD)

Against

- Benzodiazepine antagonist (Benzo OD)

References

1. Barrueto F. Calcium channel blocker poisoning. In UpToDate. 2020. [Link]

J10: Acetaminophen Toxicity

Mike Sugimoto

Updated: July 26, 2021
Reviewed: March 01, 2021

Introduction

Acetaminophen is the most widely used analgesic and antipyretic in the world and is found in a wide range of over-the-counter products. It is a generally safe drug, but overconsumption can lead to significant harm, particularly to the liver. It is the most common cause of acute liver failure and responsible for a significant fraction of all liver transplants.

Accidental acetaminophen overdose is more common among individuals who have low levels of health literacy and who do not recognize its prevalence in multiple products.

Essentials

- In early stages, acetaminophen has no readily observable toxidrome. In the out-of-hospital environment, acetaminophen overdose is most likely diagnosed through history taking, both from the patient and collaterally. Always consider the possibility of co-ingestion of other drugs or substances.
- To the extent that acetaminophen toxicity offers signs and symptoms, they are generally non-specific: nausea and vomiting; malaise; lethargy; pallor; and diaphoresis, associated with right upper quadrant abdominal pain. Patients with significant liver injury may remain asymptomatic for hours prior to their deterioration.
- Single ingestions greater than 250 mg/kg (or more than 12 g in 24 hours) are likely to cause toxicity, but injury can occur at lower doses.
- Individuals with pre-existing liver disease are at increased risk of acetaminophen toxicity and can experience significant liver dysfunction, even with doses of acetaminophen that are generally considered safe.
- Provide supportive care for patients.

Additional Treatment Information

- The specific antidote to acetaminophen, N-acetylcysteine, is a hospital-based therapy that requires diagnostic testing not available in the out-of-hospital environment.
- Administration of activated charcoal within four hours of ingestion may help reduce the need for N-acetylcysteine treatment and limit the degree of liver injury. Paramedics and EMRs/FRs identifying an acetaminophen overdose should consider the timing of ingestion and strive to deliver these patients to a hospital in a timely fashion.

Referral Information

Because of its high potential for toxicity, as well as its delayed onset of symptoms, patients suspected of acetaminophen overconsumption should be conveyed to hospital.

General Information

- There are essentially no signs or symptoms that are unique to acetaminophen overdose. Diagnosis is made on the basis of a history of ingestion combined with serum acetaminophen.

Interventions

First Responder

- Keep the patient warm and protect from further heat loss
- Place the patient in a position of comfort, as permitted by clinical condition
- Provide supplemental oxygen where indicated
  - → A07: Oxygen Administration

Critical Care Paramedic – All FR, EMR, PCP, and ACP interventions, plus:

- Consider N-acetylcysteine
  - Call ETP prior to N-acetylcysteine
  - Administer an initial loading dose of 150 mg/kg IV over 60 minutes.
  - Next, administer a dose of 50 mg/kg over four hours (infusion at 12.5 mg/kg **per hour** IV for four hours).
  - Finally, administer a dose of 100 mg/kg over 16 hours (infusion at 6.25 mg/kg **per hour** IV for 16 hours).
- Significant Troponin levels may occur. This is an ominous late sign of cardiogenic disfunction.
  - Consider Inotropic support if required
    - Dobutamine
    - Milrinone
  - Consider vasopressor support if required
    - Epinephrine
    - Levophed
    - Dopamine
    - Vasopressin

References

1. Burns M, et al. Acetaminophen (paracetamol) poisoning in adults: Pathophysiology, presentation, and evaluation. In UpToDate. 2020. [Link]
2. Heard K, et al. Acetaminophen (paracetamol) poisoning in adults: Treatment. In UpToDate. 2020. [Link]
3. Heard K, et al. Management of acetaminophen (paracetamol) poisoning in children and adolescents. In UpToDate. 2020. [Link]

J11: Marijuana and Cannabis Products

Mike Sugimoto

Updated: May 25, 2021
Reviewed: March 01, 2021

Introduction

With the advent of legalization in Canada, marijuana and marijuana-containing products are increasingly available to adult consumers.

Essentials

- Although marijuana is generally considered to be low risk, adverse effects can still occur. The most common signs and symptoms of overconsumption include anxiety, paranoia, panic, tachycardia, confusion, dry mouth, and nausea and vomiting.
- Because of their delayed onset, individuals are far more likely to over-consume edible marijuana products.
- Children are particularly at risk from ingested cannabis products.
- Cannabinoid hyperemesis syndrome is the most significant acute complication of marijuana use.

General Information

- The majority of individuals who experience adverse reactions to cannabis can be managed with gentle, supportive care and reassurance only. Symptoms are generally self-limiting and resolve gradually over a period of hours.
- Children who have consumed cannabis-containing products may develop significant and profound central nervous system depression. Hyperkinesis may occur despite apparent coma. Provide supportive care to these patients, ensuring a patent airway and effective oxygenation and ventilation.
- Cannabinoid hyperemesis syndrome is a cyclical vomiting syndrome that occurs primarily in individuals who use significant quantities of cannabis, generally on a daily basis. It involves a prodromal phase, where individuals feel vaguely unwell and mildly nauseated, followed by a hyperemetic phase with persistent nausea and vomiting. People suffering from cannabinoid hyperemesis syndrome often report that hot water (bathing or showering) improves their symptoms; some evidence suggests that use of a capsicum cream rubbed on the abdomen may also attenuate the nausea. Supportive care, including fluid replacement and anti-emetic medications, can be helpful; ultimately, cessation of cannabis use and time will allow symptoms to resolve.

Interventions

First Responder

- Keep the patient warm and protect from further heat loss
- Place the patient in a position of comfort, as permitted by clinical condition
- Provide airway management and supplemental oxygen as required
  - → B01: Airway Management
  - → A07: Oxygen Administration

Emergency Medical Responder – All FR interventions, plus:

- Measure capillary blood glucose and manage hypoglycemia as required
  - → E01: Hypoglycemia and Hyperglycemia

Primary Care Paramedic – All FR and EMR interventions, plus:

- Consider vascular access in cases of persistent vomiting
  - → D03: Vascular Access
- Consider antiemetic:
  - → E07: Nausea and Vomiting

References

1. Wang GS. Cannabis (marijuana): Acute intoxication. In UpToDate. 2020. [Link]

J12: Opioids

Mike Sugimoto

Updated: September 29, 2023
Reviewed: September 29, 2023

Introduction

Opioid overdose is the most commonly seen toxidrome in out-of-hospital practice in British Columbia, which is, as of 2020, in its fourth year of a public health emergency. Contamination of the illicit drug supply with powerful, synthetic opioids, such as fentanyl, is largely responsible for the crisis. This contamination makes consumption of any illicit drug extremely dangerous.

In 2018, 1,510 overdose deaths were recorded in the province, which represents more than four times the number of fatalities from motor vehicle collisions.

Essentials

- Opioid toxicity should be suspected in any individual with a decreased level of consciousness and depressed respirations or apnea.
- Assisted ventilation is the cornerstone of management. Paramedics and EMRs/FRs must ensure that proper airway management, including effective ventilations, continue until symptoms have resolved; this must supersede any pharmaceutical interventions. Consider the use of airway adjuncts to facilitate ventilation. Monitor oxygenation at all times.
- Assess for and treat hypoglycemia.
- The goal of naloxone administration is the restoration of adequate respirations – a return of full consciousness is not necessary.

Additional Treatment Information

- Cardiac arrests related to opioid use are primarily hypoxic. Naloxone is unlikely to benefit these patients and its routine use is unsupported by current evidence. Paramedics and EMRs/FRs must focus instead on effective ventilation, oxygenation, and chest compressions. In rare cases, patients may present with pulses that are difficult to palpate. If unsure whether a patient has a pulse, begin compressions and ventilations and evaluate the response to these treatments (e.g., oxygen saturation, heart rate, presence of central or peripheral pulses) before considering the use of naloxone.
- Effective ventilation and oxygenation are key to the successful management of opioid toxicity. A well-perfused, well-oxygenated brain that receives naloxone will be more likely to recover gracefully.
- Paramedics and EMRs/FRs must differentiate between overdoses of recreational opioids and overdoses of prescribed medication. In the case of opioid overdose from a patient's prescribed medication, a careful clinical history of opioid use must be elicited and naloxone should be administered judiciously to avoid precipitating a pain crisis or significant withdrawals.
  - In patients with palliative needs, who are presenting with respiratory depression due to opioids, there is a need to ensure adequate oxygenation while maintaining analgesia and avoiding rapid opioid withdrawal. In these patients, the administration of naloxone as a bolus, rather than titrating to respiratory effect, may result in refractory reversal of the opioid analgesia and provoke withdrawal symptoms. *As such it is recommended that the administration of naloxone, at a rate of 0.1 mg IM/IV every two minutes, be titrated to respiratory function, not to the pain or level of consciousness.*
  - See the appropriate palliative care clinical practice guidelines for altered mental status in the context of palliative care. CliniCall consultation is strongly encouraged and collaboration with the rest of the patient's care team is required to manage these cases (1-833-829-4099):
    - → P01: Palliative Care: General
    - → P02: Palliative Care: Delirium
    - → P03: Palliative Care: Pain
- Titrate naloxone to effect. Do not administer subsequent doses of naloxone without allowing the medication time to work and without assessing ventilations. Some substances, particularly the fentanyl analogues such as carfentanil, may require significantly larger doses of naloxone to resolve. Early consultation with CliniCall is

recommended in cases where patients do not improve following two doses of naloxone (see FR interventions below).

- Consider the possibility of co-intoxication when assessing patients. Other substances, such as benzodiazepines, gamma hydroxybutyrate, and alcohol can prolong unconsciousness despite resolution of opioid toxicity. Once adequate spontaneous respirations have been re-established, make preparations to convey the patient.
- Pulmonary edema is a known, but rare, complication of naloxone use. If respiratory distress develops following recovery from opioid intoxication, consider the use of CPAP to support oxygenation.
- Patients who wake up following naloxone administration can be confused and violent. Calm reassurance is more helpful in these cases than confrontation. Violence and combativeness can be reduced by ensuring patients are optimally oxygenated prior to receiving naloxone.

Referral Information

Refusal of care instructions and guidelines must be followed for patients who decline to be conveyed to hospital.

General Information

- Beyond a decreased level of consciousness and depressed respiratory drive, as demonstrated by both decreased rate and limited tidal volume, signs and symptoms of an opioid overdose can include:
  - Pinpoint pupils (miosis)
  - Hypotension
  - Hypothermia
  - Tachycardia
- Intranasal drug administration is of limited benefit in opioid overdoses, as the distribution and uptake of the medication requires ongoing respirations. It may be an acceptable option if parenteral delivery routes are unavailable.
- Patients need not have specifically ingested or otherwise consumed what they believe to be opioids to develop opioid toxicity – many recreational drugs are contaminated with synthetic opioids, and users frequently have no way to establish the safety of their substances. Black-market prescription medications, cocaine, methamphetamine, and GHB, have all been associated with opioid contamination and users of these substances have died as a result of consumption. Paramedics and EMRs/FRs should rely on the clinical signs and symptoms of opioid toxicity and manage patients accordingly, regardless of the history available at the scene.
- Drug supply contamination can be caused by multiple agents, of which fentanyl is the most common. Other fentanyl analogues, of varying potency, have been found in the supply of illicit drugs. Contaminated supply "outbreaks" occur randomly and can produce waves of overdoses and overdose fatalities.
- Questioning patients about specific quantities of substances used is unlikely to be helpful.
- Patients should be screened for the risk of additional opioid intoxication and they (or their friends and family members) educated on the use of naloxone kits. Distribute kits to patients and families in accordance with BCEHS policy. Referral pathways for treatment may be available in some regions of British Columbia and these should be utilized wherever and whenever possible.
- Refer cases of children with opioid toxicity to the Ministry of Children and Family Development in accordance with BCEHS policy.

Interventions

First Responder

- Manage the airway and support ventilations with bag-valve mask as required; consider the use of 2-person BVM techniques with appropriate airway adjuncts
  - → B01: Airway Management
- Administer high flow oxygen
  - → A07: Oxygen Administration
- Obtain capillary blood sample and assess for hypoglycemia
  - → E01: Hypoglycemia and Hyperglycemia
- Reverse opioid toxicity:

- Naloxone

Emergency Medical Responder – All FR interventions, plus:

- Consider the use of nasopharyngeal airways in patients whose level of consciousness precludes an oropharyngeal airway
  - → PR07: Nasopharyngeal Airways

Primary Care Paramedic – All FR and EMR interventions, plus:

- Consider placement of supraglottic airway device
  - → PR08: Supraglottic Airways
- Consider intravenous dextrose or intramuscular glucagon for hypoglycemia
- In cases of continued unconsciousness and apnea, consider establishing vascular access and giving naloxone intravenously
  - → D03: Vascular Access

Advanced Care Paramedic – All FR, EMR, and PCP interventions, plus:

- Consider fifth dose (4 mg) of naloxone
- CliniCall consultation required prior to sixth dose (10 mg) of naloxone
  - Note that, depending on supplies and resources available, this intervention may not be feasible

Evidence Based Practice

Overdose-Poisoning

Supportive

- Activated Charcoal
- Naloxone-IM (Opiate OD)
- Naloxone-IN (Opiate OD)
- Naloxone-IV (Opiate OD)
- Naloxone-SQ (Opiate OD)
- Capnography
- Naloxone-Nebulized (Opiate OD)
- Oxygen
- Oxymetry Monitoring
- Sodium Bicarb (TCA OD)

Neutral

- Glucagon (Beta-Blocker OD)
- Treat & Release (Opiate OD)

Against

- Benzodiazepine antagonist (Benzo OD)

References

1. Kolinsky D, et al. Is a prehospital treat and release protocol for opioid overdose safe? 2017. [Link]

2. Levine M, et al. Assessing the risk of prehospital administration of naloxone with subsequent refusal of care. 2016. [Link]
3. Rudolph SS, et al. Prehospital treatment of opioid overdose in Copenhagen—Is it safe to discharge on-scene? 2011. [Link]
4. Wampler DA, et al. No deaths associated with patient refusal of transport after naloxone-reversed opioid overdose. 2011. [Link]
5. Willman MW, et al. Do heroin overdose patients require observation after receiving naloxone? 2017. [Link]

Practice Updates

- 2023-09-29: updated FR interventions

J13: Button Battery Ingestion

Stuart Woolley

Updated: October 06, 2022
Reviewed: September 29, 2022

Introduction

To understand the pathophysiology of button battery ingestion (BBI), it is important to understand the basic principles of batteries. Batteries produce electricity using two different metals in a chemical substance known as an "electrolyte". An electrolyte simply means a substance that can be broken down by electrolysis. Each battery contains two different metals which create a chemical reaction. During this reaction, one metal will lose more electrons than the other.

When a button battery becomes impacted within the esophagus, or the digestive tract, the body's mucosa serves as a circuit between the two electrical terminals (+/-) of the battery. This circuit allows for the the electrons freed in the one metal to flow into the other metal, balancing the electrical charge.

The electrical current that is now flowing through the tissues results in the generation of hydroxide radicals in the body's tissues. This presents a serious risk to patients as hydroxide radicals are associated with a rapid rise in pH far outside normal physiological parameters. The result is caustic injury and subsequent coagulative necrosis. This can weaken the esophageal wall in a short amount of time. As the injuries are caustic in nature, there is significant probability that they will not be isolated to the point of contact and can extend to adjacent tissue, such as the trachea or great vessels.

Risk Factors

First Responders, Emergency Medical Responders, and Paramedics should be aware of the risk of battery ingestion, the presentations, and subsequent management.

There has been an increase in battery ingestion incidents, specifically in children. Over the last 10 years, there has been a 10-fold increase in complications with the likelihood of rapid damage to the body being catastrophic, with 75% of all foreign body ingestions occurring in children, specifically between the ages of 6 months and 3 years of age. Approximately 90% of BBI occurrences resulted in adverse outcomes due to a BB size greater than 20mm in diameter.

A BB size of 20-25mm in diameter carries an increased risk of the BB becoming impacted in the pediatric esophagus compared to the previously standard 15mm and under alkaline BB. The increased voltage in newer lithium cells (3.0 V) is a major contributing factor in the type and degree of harm sustained from ingestion when compared to alkaline cells (1.5 V).

This guideline provides clinicians with the knowledge necessary to quickly recognize this specific emergency, identify environmental and population-based risk factors, and to perform necessary treatment. It is focused on BBI ***only***. Paramedics and EMRs/FRs should refer to other guidelines for the management of airway obstruction, croup, epiglottitis, or anaphylaxis as required:

- → B02: Airway Obstruction
- → B04: Croup and Epiglottitis
- → E09: Anaphylaxis

Essentials

WARNING

A battery lodged in the esophagus is a medical emergency even if asymptomatic.

Presentation can vary and be non-specific – from the completely asymptomatic patient to a clinically unstable patient.

Non-specific symptoms can include nausea, pain, coughing, fever, and tachycardia. Misdiagnosis and mistreatment can be high, therefore thorough history gathering is key.

- Urgent assessment of airway patency **must** be completed prior to any other treatment

- Do not induce vomiting
- Do not delay conveyance to nearest emergency department to administer honey

Unconscious patients should have their breathing and circulation assessed concurrently. If the patient is found to be pulseless, there is no change in patient management – compressions and defibrillation continue to be prioritized above ventilations. In the event of a pediatric cardiac arrest, emphasis must be placed on early airway management and ventilatory support.

Additional Treatment Information

With the exception of honey, patients should be given nothing by mouth until the ingestion is confirmed through radiography. Honey is administered in an attempt to coat the battery poles and delay the progression of chemical burns to the adjacent tissues.

Referral Information

There is no current referral pathway for these patients. Conveyance to an Urgent and Primary Care Centre is not appropriate.

General Information

BBI can lead to significant morbidity and mortality in the pediatric population due to the creation of a local pH environment from 10-13 leading to liquefactive necrosis at the negative pole.

There is often little out-of-hospital treatment available beyond supportive care and conveyance to the nearest emergency department.

Patients who have ingested a button battery can present with vague symptoms similar to a viral illness. Any suspicion of ingestion needs to be investigated and clinicians must maintain a high index of suspicion.

General clinical features include:

- Airway obstruction and associated drooling
- Acute Stridor
- Unexplained wheeze
- Dysphagia and cough
- Difficulty swallowing or feeding
- Decreased appetite
- Throat, chest, or abdominal pain
- Fever (usually indicates esophageal perforation)

Late Signs:

- Hematemesis
- Melena or hematochezia
- Epistaxis (unless the battery is impacted within the nares, this is likely the result of injury to the esophagus)

Interventions

First Responder

- Estimate time of BBI
- Monitor and maintain airway, breathing, and circulation
- Provide supplemental oxygen as required
  - → A07: Oxygen Administration
- Monitor and provide ongoing care until arrival of additional resources

Emergency Medical Responder – All FR interventions, plus:

For stable witnessed or suspected unwitnessed ingestion within 12 hours, and if there are no airway concerns and the patient's ability to swallow is intact:

- Children over 1 year old:
  - Commercial honey, when available, should be administered
    - 10 mL every 10 minutes to a maximum of 60 mL; *do not delay conveyance to find or administer honey*
  - Convey to nearest emergency department with ongoing monitoring
- Do not give honey to children under 1 year old

References

1. Zipursky et al. Button battery ingestions in children. 2021. [Link]
2. BC Emergency Medicine Network. Foreign body ingestion -- diagnosis and treatment. 2021. [Link]
3. Darr et al. Recognition and management of button battery ingestion amongst emergency practitioners. 2021. [Link]
4. Leinwand et al. Button battery ingestions in children: a paradigm for management of severe pediatric foreign body ingestions. 2016. [Link]
5. National Capital Poisons Centre. Battery ingestion triage and treatment guideline. 2021. [Link]
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Practice Updates

- 2022-09-29: guideline created