

INTRODUCTION

Poisoning from drugs, chemicals, or exposure is a major cause of morbidity and mortality worldwide, and is the leading cause of non-traumatic cardiac arrest under the age of 35. Poisoning may occur by accidental exposure or by intentional use as is the case with self-harm or recreational drug abuse.

Initial assessment and management during a toxicologic emergency should ensure there is no ongoing exposure to the toxin, as well as ensuring the patient has a patent airway, adequate breathing and hemodynamic status. The majority of toxic exposures in the pre-hospital environment can be successfully managed by providing supportive care.

SAFETY

Toxicologic emergencies require comprehensive scene safety assessments that may reveal potential risks to clinicians, patients, and members of the general public. Many potent toxins are capable of directly threatening the health of clinicians when proper protection is not used (e.g. pesticide absorbed through the skin).

Taking protective measures is important due to the variety of possible modes of transmission including ingestion, inhalation, injection, and absorption of toxic substances. Toxic exposures can result in multiple and mass casualty incidents, and a single patient contaminated with a highly potent toxin (e.g. radioactive isotopes, nerve agents, or pesticides) can easily disable emergency systems.

- Confirm that the scene is declared safe by the proper authority before you enter.
- Wear appropriate PPE. CAUTION: N95 masks are not respirators and will not protect you from inhaled toxins.
- In the case of a HAZMAT situation maintain a safe distance from the scene. Have the patient properly decontaminated and brought to a safe, uncontaminated location before you begin treatment. Refer to the appropriate operational policies.
- Ensure that you inform receiving facilities of the event as early as possible. This will allow time to mobilize the proper resources to ensure the safety of the facility and staff.

ASSESSMENT

Though the majority of toxicologic emergencies require only supportive pre-hospital care, it is important for the clinician to have a systematic approach to assessing these patients. Once scene safety is established, the approach to the patient begins with an assessment of the level of consciousness and ABCs.

Level of Consciousness and ABCs

A number of pharmacologic agents or toxins can cause an altered mental status. This may range from somnolence or unconsciousness to severe agitation. A decreased level of consciousness will mandate airway assessment and management. Also consider whether there may be co-existing trauma and consider spinal immobilization as appropriate (refer to relevant Guidelines pertaining to airway, the agitated patient, and spinal immobilization).

Look for signs of respiratory distress on initial assessment. Inhaled gases may irritate mucous membranes of the eyes, nose, and upper airway resulting in lacrimation, nasal burning, and cough. This typically results in the patient immediately removing themselves from the source and therefore limiting the exposure. Assess and monitor such patients for signs of laryngeal edema or laryngospasm (e.g. stridor).

Inhaled toxins may also reach the lower respiratory tract causing bronchospasm, acute lung injury (previously referred to as non-cardiogenic pulmonary edema), and sometimes fulminant adult respiratory distress syndrome (ARDS). The lungs should be auscultated to assess air movement and listen for any adventitious sounds such as wheezes or crackles. Certain ingested drugs, such as ASA and opiates, can also cause acute lung injury via a systemic effect. Other toxins, such as organophosphate pesticides absorbed through the skin may lead to copious amounts of fluid being excreted into the lungs.

Inhaled toxins such as carbon dioxide or cyanide will have significant systemic effects; most fatalities resulting from fires are due to smoke inhalation as opposed to burns. These patients may have oxygen saturations of 100% with clear lung sounds, but are actually hypoxemic due to the systemic effect of the

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toxin therefore 100% oxygen should be applied (PEP 2 supportive).

When assessing circulation, be aware that many toxins can cause rate or rhythm abnormalities that can result in hemodynamic instability. The clinician should be sure to check the pulse and blood pressure early, and initiate continuous cardiac monitoring.

History

When assessing the scene, the surrounding area should be searched for prescription bottles, open medications or chemicals, signs of drug use (e.g. syringes), or other evidence of toxins in the environment (e.g. poorly ventilated building, room odours). Items related to the source of the exposure (e.g. pill bottles, chemical containers, plant leaves) should be transported if possible and safe to do so. If time allows, pills may be counted in an attempt to better quantify an overdose.

Though gathering a history from the patient themselves may be difficult, coworkers, family, friends and bystanders may all be able to provide information. A pertinent history should include the following (if possible):

- What was the patient exposed to?
- What was the route of exposure?
- Are there particular substances the patient is routinely exposed to?
- How much were they exposed to?
- How long were they exposed to it?
- Were others also exposed?
- What symptoms have occurred since the exposure?
- What were the circumstances leading to the exposure? (accidental or intentional)
- Was any treatment given/decontamination performed prior to arrival?
- What is the patient's past medical history (including any mental health concerns)?
- What allergies does the patient have?
- What medications is the patient on?
- Does the patient have a history of substance abuse?
- What other medications/toxins would the patient have access to? (e.g. family/friend's medications)

Physical Examination

A patient history may be unavailable or unreliable, due to an altered level of consciousness or lack of patient cooperation. In this setting a comprehensive physical examination becomes an even more important part of the patient assessment.

Once the ABCs and level of consciousness are assessed, the vital signs should be considered in more detail as they possess valuable information in terms of clues regarding the presence of a particular toxin or drug. Various drugs or toxins can cause a wide variety of abnormal vital signs (including thermoregulation). Refer to Figure 1 for examples of vital sign changes produced by particular substances.

Patients with a toxicologic emergency should have a cardiac monitor applied and the clinician should observe for arrhythmias. A 12-lead ECG should also be performed. A variety of substances can cause ECG changes such as bradycardia, AV block of varying degrees, tachycardia, widened QRS, prolonged QT interval, and/or ST segment changes. Tricyclic antidepressants for example can abruptly cause QRS widening and hemodynamic instability.

While assessing the patient's overall presentation, take note of whether there is any odour on the patient's breath, as some toxins produce distinct smells. Determine if the patient is responding to cues that aren't present, which would suggest visual hallucinations. Assess the patient's skin for colour and condition, evidence of track marks, skin popping, blisters or rashes.

A neurological exam with a focus on toxin mediated changes may reveal an abnormal gait, presence of tremors, or ataxia. Pupil changes can also occur in the presence of toxins; constriction with substances such as opioids and dilation with substances such as anticholinergics, sympathomimetics, and sedative-hypnotic/opioid drug withdrawal. Examine the patient's general limb tone, looking for flaccidity, rigidity, hyperactive reflexes, or myoclonus. Antidepressant and antipsychotic medications for example can cause acute syndromes that present with myoclonus and severe limb rigidity.

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Gastrointestinal symptoms are also common with a variety of toxicologic exposures. Assess for nausea, vomiting, diarrhea, or abdominal pain.

Toxidromes

Toxidromes are defined as vital sign and end organ changes that classically occur with a particular type of toxin. Refer to Figure 2 for a list of toxidromes, their causative agents, and typical presentations. Toxidromes are divided into general categories, such as sympathomimetic, anticholinergic, cholinergic, and opioid/hypnotic. If the toxin is unknown, identifying the presence of a particular toxidrome may help identify the type of substance involved. If multiple types of substances are involved (e.g. cocaine and ethanol), this may obscure the picture of any one classic toxidrome. It is perhaps most important to keep in mind that most drugs/toxins produce other unique clinical presentations that usually do not fit into one of the above categories.

MANAGEMENT

Limiting Exposure & Decontamination

The initial goal during a toxicologic emergency is to limit the patient's exposure to the substance as much as possible thereby limiting absorption. Determine quickly whether following HAZMAT policy is applicable, in which case there are other personnel who will perform decontamination prior to treatment and/or transport. Outside of HAZMAT scenarios, assess for whether the patient requires flushing of a dermal, eye or mucous membrane exposure on scene. Typically clinicians are unable to decontaminate ingested toxins in the field. There are options available during the subsequent ED phase of care to allow decontamination or enhanced elimination (e.g. activated charcoal, whole bowel irrigation, or hemodialysis) therefore rapid transport to an appropriate facility is important.

Airway Management

Assessment for patency and early intervention with basic or advanced airway management may be indicated for the patient with decreased level of consciousness or hypoxia. Aggressive airway management (e.g. intubation) should be avoided when the cause of the altered mental status is deemed reversible (e.g. opioid intoxication, hypoglycemia, or arrhythmia). For more information

refer to the Adult Airway Management Clinical Practice Guideline.

Hemodynamic Management

Patients experiencing hypotension should be initially managed with IV fluid. Vasopressors (e.g. dopamine) may be indicated if the patient remains unresponsive to fluid therapy. For more information refer to the Shock Clinical Practice Guideline.

Cardiac Arrhythmias

For brady- or tachyarrhythmias of toxic origin, consider whether there is an antidote or specific therapy (see below). Management otherwise includes atropine, transcutaneous pacing, amiodarone, and/or synchronized cardioversion as indicated. Refer to the Adult Cardiac Arrhythmia Clinical Practice Guideline.

Seizures

Treatment of seizures should include appropriate administration of benzodiazepines, adequate airway protection, and oxygenation. Blood glucose should also be reassessed.

Agitation

Many patients suffering from an intentional or unintentional toxicologic exposure may exhibit violent or agitated behavior. "Agitated delirium" (also known as excited delirium or autonomic hyperarousal state) is a syndrome commonly encountered in the setting of drug use. This may necessitate physical or chemical restraint to prevent the patient from harming themselves or care providers. Refer to the Behavioural Emergencies Clinical Practice Guideline for treatment recommendations.

IWK Regional Poison Centre

It is recommended the clinician contact the IWK Regional Poison Centre (1-800-565-8161 or through the Medical Communications Centre) when managing a patient who has been exposed to a substance in order to discuss the potential severity of the exposure as well as any treatment recommendations. Any specific management suggestions falling outside of your scope of practice should be reviewed with OLMC prior to implementation.

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Specific Management

Toxic exposures frequently encountered in the pre-hospital setting, as well as exposures with antidotes or specific therapies that can be administered outside of hospital are outlined here. A more complete list of toxins and their antidotes are outlined in Figure 3.

Ethanol

Ethanol (drinking alcohol) intoxication is common in patients seeking emergency care, whether it reflects the primary complaint or an incidental finding. Signs and symptoms of ethanol intoxication include slurred speech, nystagmus, disinhibited behavior, incoordination, unsteady gait, memory impairment, decreased level of consciousness, or coma. It may also cause bradypnea, hypoxia, and hypotension in the patient with decreased level of consciousness.

It is important that clinicians ensure that ethanol intoxication is a diagnosis of exclusion, and that other serious alternative or coexisting etiologies have been explored. Co-ingestants, head trauma, hypoxia, hypoglycemia, hypothermia, and other metabolic and physiologic conditions must also be considered.

Ethanol intoxication usually requires supportive care and serial observation. In the absence of signs of volume depletion, IV catheterization and volume replacement are not usually necessary. Severe ethanol intoxication often requires aggressive care including close monitoring of airway and respiratory status, as well as IV rehydration. Patients who abuse ethanol may also develop acute complications such as alcoholic ketoacidosis, electrolyte abnormalities, and Wernicke's encephalopathy requiring a prolonged stay in the ED or hospital admission.

Acute ethanol withdrawal and its more extreme presentation "delirium tremens" can be life threatening. Withdrawal presents with coarse tremors, agitation, tachycardia, diaphoresis, delirium, hallucinations, fever, and in some instances seizures. Ethanol withdrawal requires acute management with benzodiazepines.

Benzodiazepines

Benzodiazepines are widely prescribed, and overdose is therefore common in the setting of both recreational use and with gestures of self-harm. They have a primarily depressive effect on the body, causing slurred speech, ataxia, and depressed mental status. Isolated overdose often presents with an altered mental status with other vital signs within normal limits.

Respiratory compromise is possible with isolated oral ingestion, but with intentional overdose they are often taken with a second agent, most commonly ethanol. The synergistic effect of these substances places the patient at increased risk of respiratory depression. Clinicians must carefully monitor all of these patients for signs of respiratory depression. Benzodiazepine overdose rarely requires more than supportive care, and close observation of a patient's airway status and respiratory drive.

Anti-depressants

Anti-depressant medications are commonly prescribed, and commonly seen in overdose. Anti-depressants include a wide variety of drugs with a number of different mechanisms of action. Examples would include tricyclic antidepressants (TCAs), monoamine oxidase inhibitors (MAOIs), and selective serotonin reuptake inhibitors (SSRIs). SSRIs are typically associated with less mortality following overdose. Some anti-depressants, such as TCAs, venlafaxine and bupropion have a higher risk of neurologic or cardiovascular complications. With the exception of TCAs, the management of anti-depressant overdose is largely supportive.

Although uncommon, TCA overdose is a life-threatening condition because of their effect on several receptors and channels across multiple systems. This results in a variety of signs and symptoms including sedation, confusion, delirium, hallucinations, seizures, hypotension, and tachycardia or arrhythmias.

ECG monitoring of a patient with an anti-depressant overdose is important, as some can lead to cardiovascular complications. ECG monitoring of a patient with TCA poisoning is critical as patients can deteriorate rapidly even with initially stable appearing presentations. A prominent ECG

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manifestation of TCA toxicity is a wide QRS duration. Severe toxicity can result in ventricular fibrillation or ventricular tachycardia. Sodium bicarbonate is indicated in the pre-hospital setting for TCA poisoning (**PEP 3 neutral**), in the setting of a wide QRS, hemodynamic instability (e.g. hypotension), or cardiac arrest. A toxin-induced wide QRS is considered to be anything greater than 100msec, which is different from the normally-defined 120msec. OLMC may be contacted regarding additional doses beyond what is endorsed in the medication profile.

Opioids

Opioids have the ability to interact with a variety of receptor sites within and outside the central nervous system therefore these agents have a large range of effects on many physiologic systems. The classic signs of opioid intoxication include CNS depression, decreased respiratory rate and depth, and miotic (constricted) pupils. It is important to note that a normal pupil examination does not rule out a possible opioid intoxication. The most reliable sign of opioid intoxication is the presence of a depressed respiratory drive in conjunction with possible drug use.

The primary goal when treating the opioid intoxicated patient should focus on effective airway and breathing management. Often BLS maneuvers (jaw-thrust and bag-valve-mask ventilation) are sufficient to maintain adequate oxygenation and ventilation in the pre-hospital setting.

Naloxone may be indicated (**PEP 1-3 supportive** depending on route administered) if oxygenation cannot be accomplished with BLS maneuvers. **If administered, the goal of naloxone administration is improvement of the patient's respiratory drive, sufficient to allow adequate oxygenation and ventilation. The goal is not to achieve an increase in level of consciousness.** Begin with a very small dose of naloxone. Inadvertently administering too high a dose can precipitate acute narcotic withdrawal, or an agitated and uncooperative patient, which can be a danger to both the patient and care provider. If naloxone administration is unsuccessful in improving the patient's respirations and oxygenation, the clinician should explore alternative etiologies of the patient's hypoventilation and consider an advanced airway.

Patient's in opioid withdrawal present with symptoms opposite of those observed in the opioid toxidrome. Opiate withdrawal causes agitation, malaise, diaphoresis, nausea, diarrhea, and tachycardia. Pre-hospital care is supportive as opioid withdrawal is not acutely life-threatening, though it is very uncomfortable for the patient. Pre-hospital administration of an opioid (e.g. fentanyl, morphine) is not indicated in this setting.

Acetaminophen

Acetaminophen is one of the most common agents related to deaths due to medication poisoning. It is easily accessible and is a common co-ingestant. Signs and symptoms related to acetaminophen poisoning depend on the time since ingestion. Most patients presenting within 24 hours are asymptomatic. After 24 hours, patients present with nausea, vomiting, and abdominal pain due to liver injury. 3-4 days later liver failure may occur with encephalopathy and multi-organ failure.

Rapid identification of possible acetaminophen poisoning is important because treatment with N-acetylcysteine is most successful within 8 hours of ingestion. It is helpful for pre-hospital clinicians to determine the number and strength of tablets ingested, as well as the time they were ingested.

Calcium Channel Blockers and Beta-Blockers

These are common medications which can be highly toxic in overdose. Signs and symptoms of toxicity include hypotension and bradycardia. Patients will therefore require continuous cardiac monitoring, pacing pads should be readily available or applied to the chest, IV access obtained, and the patient's level of consciousness and blood pressure should be frequently reassessed. If the patient becomes hemodynamically unstable or symptomatic due to hypotension or bradycardia, they may require intravenous fluid boluses, calcium chloride and vasopressors. Treatment of bradycardia may include atropine and pacing as appropriate, however these interventions may be ineffective in this setting. See Adult Cardiac Arrhythmia Clinical Practice Guideline for more details.

Though glucagon can be used as an antidote for beta-blocker overdose (**PEP white**), however high-

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doses are required which are most often unavailable in the pre-hospital setting.

Organophosphates & Carbamates

Organophosphates and carbamates are potent cholinesterase inhibitors commonly used as insecticides. Exposure can be through absorption, inhalation, or ingestion.

Acute toxicity presents with the cholinergic toxidrome (see Figure 2). This toxidrome represents one of the exceptions where decontamination occurs prior to ABC management. Because the toxin can be absorbed through the skin, there is significant risk to care providers who are in contact with the patient. Thorough decontamination and personal protective measures are extremely important.

Patients with markedly depressed mental status require airway management as per the Adult Airway Management Clinical Practice Guideline. Excessive bronchorrhea or respiratory secretions are the major cause of respiratory failure for these patients. Atropine should be administered to symptomatic patients (**PEP white**), as it competitively binds to cholinergic receptors. Atropine administration should be titrated to clearing respiratory secretions, allowing adequate oxygenation and ventilation. Very high and repeat doses of atropine are frequently required to achieve clinical improvement.

SPECIAL POPULATIONS

Pediatric Patients

Toxicologic emergencies in the pediatric population are most often due to unintentional exposure to a small amount of some particular substance. Because the quantity of substance ingested is usually small, pediatric mortality due to toxicologic emergencies is low. However, there are some substances that are associated with a higher mortality including carbon monoxide, iron and hydrocarbons. Clinicians should also be aware of medications where “one pill can kill” if ingested by a child. These are classes of medications which can kill a toddler with one or two standard doses, and includes:

- Tricyclic antidepressants (e.g. amitriptyline)
- Antipsychotics (e.g. loxapine)
- Antimalarials (e.g. chloroquine)

- Class I antiarrhythmics (e.g. procainamide)
- Calcium channel blockers (e.g. diltiazem)
- Camphor (used in analgesic/cooling gels and cough suppressants)
- Methyl salicylate (oil of wintergreen)
- Theophylline
- Narcotics
- Oral hypoglycemics – Sulphonylureas
- Podophyllin 25%

Considering what is accessible to the child is very important if the ingestant is not known. Consider plants, animals, other people’s medications or vitamins, etc. For instance, life threatening iron toxicity can easily occur in a child who has ingested their mother’s maternal vitamins.

Any overdose or toxicologic exposure in a very young child (under 6 months) is due to either passive exposure to a substance, inadvertent administration of an incorrect substance or dose by another individual or intentional administration of a substance. The clinician should consider the possibility of abuse or neglect when presented with a young child presenting with an overdose or exposure. Self-harm or suicidal intent should also be considered in older children (i.e. children over the age of 5).

In the adolescent population, toxic effects commonly occur with drug experimentation. Some of the more common causes of death due to toxicologic emergencies in this group are from hydrocarbons, antidepressants and analgesics.

The IWK Regional Poison Centre should be contacted for any pediatric patient with a toxicologic exposure. Based on type and amount of substance ingested, their presentation, as well as patient’s body weight, the patient may require transport to the hospital, on scene treatment only, or may remain at home. Review all non-transport decisions for toxicology patients with OLMC as well.

Elderly Patients

Toxicologic emergencies are associated with a high mortality rate in the elderly population. This increase in mortality is the result of several factors including:

- Underlying disease

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- Polypharmacy
- Unrecognized toxicity with delay in diagnosis
- Unexpected side effects due to alterations in pharmacodynamics
- Drug interactions
- Atypical symptoms and signs of toxicity
- Delayed manifestations of toxicity
- Availability of highly toxic agents
- Increased risk of chronic toxicity
- An increased rate of suicide

Impaired vision, hearing, and dementia also increase the risk of toxicity and/or overdose in the elderly. Because drug dependence may not be considered in older patients, withdrawal may occur, particularly during hospitalization.

As age increases, several pharmacokinetic characteristics change. Renal function decreases, hepatic blood flow declines, hepatic enzyme activity may fall, body fat content increases, and protein synthesis declines. All these changes combine to alter drug metabolism, distribution, and excretion in often unpredictable ways. These patients have an increased risk of toxic effects even with therapeutic dose ranges, particularly in the setting of polypharmacy.

Substance Addiction

Substance addiction is difficult to define as it involves complicated social, medical, and cultural factors. Individuals who experience substance addiction are at higher risk for a number of related health issues including smoking, cardiovascular risk, diabetes, high blood pressure, and high cholesterol. In addition, drug addiction is associated with an increase in criminal activities, violence, and homelessness.

The presence of contaminants in “street drugs” and their associated toxicity must always be considered. Common adulterants include local anesthetics, quinine, talc, phencyclidine, LSD, sugars, and sodium bicarbonate. Some are added with malicious intent (strychnine, thallium), and clandestine laboratories may accidentally produce additional toxic agents.

Medical complications must always be considered in the differential diagnosis, requiring a detailed history and physical examination in patients with substance addiction. Intravenous drug users represent a group at particular risk for medical problems, including human immunodeficiency virus infection, hepatitis, endocarditis, skin and soft tissue infections, thrombophlebitis, osteomyelitis, and central nervous system infections.

The emergency system of care is often the only contact these patients have with the health care system. This circumstance offers the unique opportunity to intervene both medically and socially. Their social, medical, and cultural influencing factors can be further assessed in the emergency health care system.

Subsequent ED Management

If the substance is unknown, supportive care will ensue while collecting further information. Blood tests may be appropriate to look for some toxins. Common co-ingestants (e.g. acetaminophen, aspirin, ethanol) are also screened for on blood work as they may impact management.

Consideration is given as to whether there are indications to decontaminate the patient’s GI tract. Though uncommonly used, activated charcoal or whole bowel irrigation can be used for this. Indications for this are very rare. Gastric lavage (“stomach pumping”) is also very rarely performed as the potential complications outweigh the benefits. If the patient has ingested a dialyzable drug, arrangements may be made to initiate hemodialysis.

The ED clinician will explore whether there is an antidote or specific recommended therapy for the particular exposure they are treating (Figure 3).

Patients are carefully monitored until their level of consciousness, vital signs, ECG or diagnostic/lab test abnormalities return to normal. If a patient has suicidal thoughts, they are not discharged until these thoughts are addressed. Psychiatry service will often assess and possibly admit these patients.

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TRANSFER OF CARE

Provide the receiving facility advanced notice while en route so that they can take appropriate protective measures and have rooms ready for your patient.

Upon transfer of care make sure to inform receiving hospital of:

- Identity of toxin/drug, route and amount ingested (if known), and the time the incident occurred
- Any treatment that occurred before you arrived on scene
- Events that led up to the incident and patient condition upon arrival to scene
- Patient symptoms
- ECG changes
- Treatment that was rendered (as well as accurate times of treatments)
- Any advice provided by the IWK Regional Poison Center

CHARTING

Not only is it important to clearly document the patient assessment findings and interventions, but details about the scene and substance, time of exposure, and any discussions had with OLMC or the IWK Regional Poison Centre. Ensure an accurate and complete patient medication list is documented.

Key Points to Remember

Focus on stabilizing the patient's airway, breathing, and circulation

Thoughtful consideration of scene, history, and physical exam findings is essential

Supportive care is sufficient for the vast majority of patients with toxic exposure

Call the IWK Regional Poison Centre (1-800-565-8161 or through the Medical Communications Centre)

KNOWLEDGE GAPS

The incidence and prevalence of true toxicologic emergencies in an EMS system is relatively unknown. Delineation between intentional vs unintentional overdose is often unclear and requires a thorough history in order to help determine the cause.

Clinicians should be aware of substances that can lead to significant cardiovascular and/or neurological complications.

The optimal use of antidotes (e.g. naloxone) in the pre-hospital setting (including bystander administration) is under review.

EDUCATION IMPLICATIONS

As new drugs and toxins become readily available, clinicians must continually adapt in order to devise appropriate assessment and management strategies. Clinicians should remain current by participating in self-directed and/or organized continuing medical education.

QUALITY IMPROVEMENT

Areas of interest as part of a CQI program may include calls where antidotes were used, where advanced airway management was required, or where the clinicians contacted the IWK Regional Poison Centre.

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Figure 1: Possible substances causing vital sign abnormalities.

Vital Sign Abnormality	Causative Substance
Bradycardia	Organophosphates, opioids, sedatives, ethanol, digoxin, calcium-channel or beta-blockers, clonidine, antiarrhythmics
Tachycardia	Sympathomimetics, drug withdrawal, theophylline, antihistamines, tricyclic antidepressants, derivatives of belladonna, antipsychotics
Hypotension	Calcium-channel or beta-blockers, tricyclic antidepressants, opioids, heavy metals, lithium, organophosphates, diuretics, sedative hypnotics, severe ethanol toxicity
Hypertension	Sympathomimetics, drug withdrawal, anticholinergics, theophylline
Hypoventilation	Opioids, sedatives, barbiturates, alcohol
Hyperventilation	Sympathomimetics, salicylates, theophylline, drug withdrawal, carbon monoxide, cyanide, opioids (if pulmonary edema develops)
Hypothermia	Opioids, insulin, ethanol, carbon monoxide, barbiturates, oral hypoglycemics
Hyperthermia	Sympathomimetics, SSRIs, antipsychotics, drug withdrawal, salicylates, tricyclic antidepressants, anticholinergics, antihistamines, derivatives of belladonna

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Figure 2: Common toxidromes

Toxic Syndrome	Common S/S	Causative Agent	Specific Tx
Cholinergic “wet patient” Note: this can affect the muscarinic and/or nicotinic receptors; presentation may differ depending on receptors affected	Muscarinic symptoms include: Salivation Lacrimation Urination Defecation GI Upset Emesis as well as: Miosis Bradycardia Bronchoconstriction Altered mental status Diaphoresis Seizures If the nicotinic receptors are involved, the presentation may be: tachycardia, hypertension, facial fasciculations and weakness or paralysis	Muscarinic agents include: organophosphate and carbamate insecticides and pilocarpine Nicotinic agents include: organophosphate and carbamate insecticides, nicotine and black widow spider venom	Atropine Pralidoxime Diazepam Activated charcoal
Anticholinergics “dry patient”	Delirium Tachycardia Dry flushed skin Dilated pupils Seizures Dysrhythmias Hyperthermia Hypertension Urinary retention Absent bowel sounds	Antihistamines Antiparkinsons Meds Atropine Antipsychotic agents Tricyclic antidepressants Scopolamine Skeletal muscle relaxants Many Plants (jimson weed, Amanita Muscaria, derivatives of belladonna)	Diazepam Activated charcoal Physostigmine (rare)
Opioids	Euphoria Hypotension Bradycardia Respiratory depression/arrest Nausea Pinpoint pupils Seizures Coma Hypothermia	Heroin Morphine Codeine Meperidine Propoxyphene Fentanyl Hydrocodone	Narcan

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6289.03 Toxicologic Emergencies

Sympathomimetic	Delusions Paranoia Tachycardia Diaphoresis Seizures Hypertension Dysrhythmias Anxiety Delirium Dilated pupils Hot skin	Cocaine Amphetamine Methamphetamine Over the counter decongestants Drug withdrawal	Minimal sensory stimulation Calming measures Diazepam PRN
Sedative-hypnotic	Confusion Respiratory depression Hypotension Hypothermia Variable pupillary changes Seizures	Barbiturates Benzodiazepines	

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Figure 3: Common antidotes of toxicologic emergencies

Toxin	Antidote
Acetaminophen	N-Acetylcysteine
Anticholinergic agents	Physostigmine
Venom	Antivenin
Benzodiazepines	Flumazenil
Beta-blockers	Glucagon, high dose insulin/dextrose
Calcium channel blockers	Calcium chloride, high dose insulin/dextrose, glucagon
Carbon monoxide	Oxygen and hyperbaric oxygen
Coumadin	Vitamin K, fresh frozen plasma or Octaplex
Cyanide	Sodium nitrite, sodium thiosulfate, hydroxocobalamin
Digitalis	Digibind
Phenothiazines	Diphenhydramine, cogentin
Iron	Deferoxamine
Lead, mercury, arsenic	Dimercaprol, BAL, Dimaval
Methanol, ethylene glycol	Ethanol, fomepizole
Nitrates	Methylene blue
Opiates	Naloxone
Organophosphates	Atropine, pralidoxime
Carbamates	Atropine
Tricyclic antidepressants (and other fast sodium channel blockers)	Sodium bicarbonate, lipid emulsion therapy

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PEP 3x3 TABLES for TOXICOLOGIC EMERGENCIES

Throughout the EHS Guidelines, you will see notations after clinical interventions (e.g.: **PEP 2 neutral**). PEP stands for: the Canadian Prehospital Evidence-based Protocols Project.

The number indicates the Strength of cumulative evidence for the intervention:

- 1 = strong evidence exists**, usually from randomized controlled trials;
- 2 = fair evidence exists**, usually from non-randomized studies with a comparison group; and
- 3 = weak evidence exists**, usually from studies without a comparison group, or from simulation or animal studies.

The coloured word indicates the direction of the evidence for the intervention:

- Green = the evidence is supportive** for the use of the intervention;
- Yellow = the evidence is neutral**;
- Red = the evidence opposes** use of the intervention;
- White** = there is no evidence available for the intervention, or located evidence is currently under review.

PEP Recommendations for Toxicologic Emergencies Interventions, as of 2015/06/11. PEP is continuously updated. See: <https://emspep.cdha.nshealth.ca/TOC.aspx> for latest recommendations, and for individual appraised articles.

Pesticide Poisoning

Recommendation		RECOMMENDATION FOR INTERVENTION			
		SUPPORTIVE (Green)	NEUTRAL (Yellow)	OPPOSING (Red)	NOT YET GRADED (White)
STRENGTH OF RECOMMENDATION FOR INTERVENTION	1 (strong evidence exists)				• Atropine
	2 (fair evidence exists)				
	3 (weak evidence exists)				

Smoke Inhalation

Recommendation		RECOMMENDATION FOR INTERVENTION			
		SUPPORTIVE (Green)	NEUTRAL (Yellow)	OPPOSING (Red)	NOT YET GRADED (White)
STRENGTH OF EVIDENCE FOR INTERVENTION	1 (strong evidence exists)				
	2 (fair evidence exists)	• Oxygen			
	3 (weak evidence exists)		• Direct Transport To Hyperbaric Facility		

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Overdose-Poisoning

Recommendation		RECOMMENDATION FOR INTERVENTION			
		SUPPORTIVE (Green)	NEUTRAL (Yellow)	OPPOSING (Red)	NOT YET GRADED (White)
STRENGTH OF EVIDENCE FOR INTERVENTION	1 (strong evidence exists)	<ul style="list-style-type: none"> Naloxone IM Naloxone IN 			<ul style="list-style-type: none"> Glucagon for Beta-Blocker OD Oxygen for carbon monoxide poisoning
	2 (fair evidence exists)	<ul style="list-style-type: none"> Naloxone IV Naloxone SQ Treat & Release (Opiate OD) 	<ul style="list-style-type: none"> Activated Charcoal 	<ul style="list-style-type: none"> Benzodiazepine antagonist 	
	3 (weak evidence exists)	<ul style="list-style-type: none"> Naloxone-Nebulized Oxygen 	<ul style="list-style-type: none"> Sodium Bicarb for TCA OD 		

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