Chapter 10
ENVIRONMENTAL EMERGENCIES

Learning Objectives:

- Identify different environmental Injuries.
- Initial emergency management of Environmental emergencies
- Able to make decision for referral for advanced management and care.

INTRODUCTION

Environmental injuries and illness can happen at home, work or recreational settings. These injuries require basic knowledge in emergency management. In Bhutan more than 42 truckloads of firewood are burnt each day leading air pollution. Industrial activity is growing in Bhutan leading to chemical exposures. Flash flood and landslides are common during monsoon extending from May to August. It has caused about hundreds of lives and damage worth in millions. Population of Bhutan are migrating and exposed to different environmental injuries.

FROSTBITE AND OTHER LOCALIZED COLD INJURIES

Cold related injuries depend on the degree of cold exposure as well as individual factors.

Nonfreezing cold injuries: Which are due to exposure to wet conditions when temperature is above the freezing point. E.g., Trench foot, Chilblains Panniculitis & cold Urticaria. These conditions require supportive care, keeping the part dry.

Freezing Injuries: Frostbite is the prototype of freezing injury and is seen when the ambient temperature is below the freezing point. Areas most commonly affected are head (39%), hands (27%) and feet (24%). Frostbite may result to permanent tissue damage.

Table 10.1 TREATMENT

Prehospital

- Prevention of further cold injury, Hypothermia & dehydration.
- Remove wet, constrictive cloths and replace with dry clothing.
- Avoid heating of frozen areas & thawing should not be attempted until the risk of freezing is eliminated.
- Adequate analgesia
- Immobilize, Elevate and handle gently the frozen extremities.
- Do not allow patient to ingest alcohol or smoke.

Core Treatment

- Immersion in or application of water at 40-42 deg C (104-107 deg F) until affected area is pliable and erythematous.
- Topical Aloe vera Cream every 6 hourly
- No blister of soft tissue debridement acutely
- Meticulous local care
- Tetanus immunization
- Parenteral opioids for pain management
- Ibuprofen 12mg/kg/day in divided doses

Optional Treatment:

- Topical silver sulfadiazine (SSD) Cream for prophylaxis (do not use on face)

HYPOTHERMIA

Definition: Hypothermia is when the core temperature is <35 deg C (<95 deg F). Hypothermia causes characteristic ECG changes and may induce life threatening dysrhythmias. The Osborn or J wave – a slow, positive deflection at the end of QRS complex is characteristics, though not pathognomonic, of hypothermia. Patients are at risk of

dysthymias at body temperature below 30 deg C (86 deg F). Treatment includes general support and specific rewarming techniques.

Table 10.2 Treatment of hypothermia

General Support

- Careful, gentle handling of patient because manipulation can precipitate ventricular fibrillation.
- In unmonitored patients spent 30-45 seconds to detect respiratory activity and to palpate pulse, if none is detected, CPR should be initiated.
- Oxygen and IV fluids should be warmed and patients core temperature, cardiac rhythm and oxygen saturation should be monitored.
- Intubate if indicated.
- Attempt to bring temperature above 30-degree C (86-degree F)
- Give IV Thiamine 100 mg if patient is alcoholic

Rewarming Techniques

1. Passive Rewarming
    - Removal from cold environment
    - Insulation
2. Active external rewarming
    - Warm water immersion
    - Heating blankets set at 40 deg C (104 deg F)
    - Radiant heat
    - Forced air
3. Active Core rewarming at 40 deg C (104 deg F)
    - Inhalation rewarming
    - Heated IV fluids
    - GI tract lavage
    - Bladder lavage
    - Peritoneal lavage
    - Pleural lavage

HEAT EMERGENCIES

Heat emergencies represents a spectrum of disorders, including heat cramps, heat syncope, heat exhaustion and heat stroke. Human body tends to maintain its core temperature between 36 deg C and 38 deg C (96.8 to 100.4 deg F) Native thermal regulation mechanism begin to fail at core temperature of <35 deg C (<95 deg F) and > 40 deg C (>104 deg F).

Heat Exhaustion: Heat exhaustion can be either by water depletion or sodium depletion, but often is characterized by combination of both. They present with the symptoms of headache, nausea, vomiting, malaise, dizziness and muscle cramps as well as signs of signs of dehydration. On physical examination temperature may be normal or elevated, usually not above 40 deg C (104 deg F). Patient with heat exhaustion do not manifest central nervous system impairment. It is treated with volume and electrolyte replacement and rest. Of note, heat exhaustion can progress to heat stroke, therefore if patient do not respond to 30 minutes of fluid resuscitation they should be aggressively cooled until their core temperature drop to 39 deg C (102 deg F).

Heat Stroke: Heat stroke is an acute life-threatening emergency with mortality rate as high as 30-80%. The cardinal features of heat stroke are hyperthermia {>40 deg C (>104 deg F)} and altered mental status. The goal of the therapy are immediate cooling and support of organ system function.

BITES AND STINGS

Venomous bites and stings from arthropods are a significant problem.

Clinical features of venomous bites and stings are local reaction, toxic reaction and anaphylactic reaction.

Majority of systemic reaction occur within 15 minutes to 6 hours. Initial symptoms generally consist of itching eyes, facial flushing, generalized Urticaria and dry cough. Symptoms may intensify rapidly, with chest and throat constriction, wheezing, dyspnea, cyanosis, abdominal cramps, diarrhea, nausea, vomiting, vertigo, chills and fever, stridor, shock, syncope, involuntary bowel and bladder actions. And bloody frothy sputum. Signs and symptoms may recur 8-12 hours after the initial reaction.

Treatment includes:

- Removal of bee stinger
- Wash the site with soap and water and application of ice packs
- Treatment begins similarly to that for anaphylaxis
- The most important agent to administer is Injection Adrenalin0.3-0.5 mg 1:1000 concentration in adults and 0.01 mg/kg in children (never more than 0.3 mg). Injected intramuscular and massaged to hasten absorption
- Antihistamines-Injection Promethazine 25-50 mg IM/IV
- Steroid- Injection Hydrocortisone 100mg or Oral Prednisolone 60 mg
- Salbutamol nebulization for bronchospasm
- IV Crystalloid fluid infusion for Hypotension
- IV Adrenalin infusion (0.1-1 mcg/kg/min) if non-responsive to fluid resuscitation

SNAKE BITES

According to WHO, in 2000 2085 bites and stings were reported common species are cobra, king cobra, krait, and pit viper. More than 90% of snake venom (dry weight) is protein and each venom contain more than hundred different proteins. Bites by small snake should not be ignored, they should be taken just as seriously as bites by large snakes of same species. Contributing factors of death from snake bit can be from inadequate use or mono specific antivenom, delay in hospital treatment. Inadequate artificial ventilation, failure to treat hypovolemia in shock patient, airway obstruction and failure to observe closely after they were admitted to the hospital.

Local symptoms and signs in the bitten part

- Fang marks
- Local pain
- Local bleeding
- Bruising
- Lymphangitis
- Lymph node enlargement
- Inflammation
- Blistering

- Local infection
- Necrosis

Systemic symptoms and signs

General: Nausea, vomiting, malaise, abdominal pain, weakness, drowsiness.
Cardiovascular (Viperidae): visual disturbance, dizziness, faintness, collapse, shock, hypotension, cardiac arrhythmia, pulmonary edema, conjunctival edema.
Bleeding or clotting disorder: Bleeding from fang marks, spontaneous systemic bleeding (epistaxis, gum bleeding, ICH. Hemoptysis, hematemesis, hematuria etc.) and cerebral artery thrombosis.
Neurological (Elapidae): Drowsiness, paresthesia, abnormal taste and smell, heavy eyelid (ptosis), external ophthalmoplegia, paralysis of facial muscles, regurgitation through nose, difficulty in swallowing secretion, respiratory and generalized flaccid paralysis.
Skeletal muscle breakdown: Generalized pain, stiffness and tenderness of muscle, trismus, myoglobinuria, hyperkalemia, acute renal failure and cardiac arrest.
Renal (Viperidae): Loin pain, hematuria, hemoglobinuria, myoglobinuria, oliguria/anuria.
Endocrine: Acute pituitary/adrenal insufficiency from infarction of anterior pituitary.

Table 10.3 Management of Snake Bite

First Aid Treatment

- Reassure the victim who may be very anxious
- Immobilize the whole of the patient's body by laying him/her down in a comfortable and safe position and, especially, immobilize the bitten limb with a splint or sling.
- If the necessary equipment and skills are available, consider pressure-immobilization.
- Avoid any interference with the bite wound (incisions, rubbing, vigorous cleaning, and massage, application of herbs or chemicals) as this may introduce infection, increase absorption of the venom and increase local bleeding.
- Release of tight bands, bandages and ligatures: Ideally, these should not be released until the patient is under medical care in hospital, resuscitation facilities are available and anti-venom treatment has been started (Watt et al., 1988).

Transport to Hospital

- The patient must be transported to a place where they can receive medical care (dispensary or hospital) as quickly, but as safely and comfortably, as possible.
- If possible, patients should be placed in the recovery position, in case they vomit.

Rapid Clinical Assessment and Resuscitation

- Ensure airway patency, breathing (respiratory movements) is optimal, Circulation (arterial pulse) is adequate, Disability of the nervous system (level of consciousness) is normal and exposure and environmental control (protect from cold, risk of drowning etc.)
- Early clues that a patient has severe envenoming: Snake identified as a very dangerous one. Rapid early extension of local swelling from the site of the bite. Early tender enlargement of local lymph nodes, indicating spread of venom in the lymphatic system. Early systemic symptoms: collapse (hypotension, shock), nausea, vomiting, diarrhea, severe headache, "heaviness" of the eyelids, inappropriate (pathological) drowsiness or early ptosis/ophthalmoplegia. Early spontaneous systemic bleeding. Passage of dark brown/black urine.

Investigations/laboratory tests

- 20-minute whole blood clotting test (20WBCT): If the blood is still liquid (unclotted) and runs out, the patient has hypofibrinogenemia ("incoagulable blood") as a result of venom-induced consumption coagulopathy
- Hemoglobin concentration/hematocrit, Platelet count, White blood cell count, Biochemical abnormalities, Arterial blood gases and pH, Urine examination

Anti-venom Treatment

- Antivenom is the only specific antidote to snake venom. A most important decision in the management of a snake-bite victim is whether or not to administer antivenom.
- Antivenom should be given only to patients in whom its benefits are considered likely to exceed its risks. Since antivenom is relatively costly and often in limited supply, it should not be used indiscriminately. The risk of reactions should always be taken into consideration [level of evidence E].
- Epinephrine (adrenaline) should always be drawn up in readiness before antivenom is administered.
- Antivenom should be given by the intravenous route whenever possible.
- Reconstituted freeze-dried or neat liquid antivenom is diluted in approximately 5-10 ml of isotonic fluid per kg body weight (i.e. 250-500 ml of isotonic saline or 5% dextrose in the case of an adult patient) and is infused at a constant rate over a period of about one hour.
- Patients must be closely observed for at least one hour after starting intravenous antivenom administration, so that early anaphylactic antivenom reactions can be detected and treated early with epinephrine (adrenaline).
- Snakes inject the same dose of venom into children and adults. Children must therefore be given exactly the same dose of antivenom as adults.
- Suggested initial doses of Indian manufacturers polyvalent is 100 ml.

Indications for Anti-venom: Antivenom treatment is recommended if and when a patient with proven or suspected snake-bite develops one or more of the following signs:

Systemic envenoming

- Hemostatic abnormalities: Spontaneous systemic bleeding (clinical), coagulopathy (20WBCT or other laboratory tests such as prothrombin time) or thrombocytopenia (<100 x 109/litre or 100 000/cu mm) (laboratory).
- Neurotoxic signs: ptosis, external ophthalmoplegia, paralysis etc. (Clinical).
- Cardiovascular abnormalities: hypotension, shock, cardiac arrhythmia (clinical), abnormal ECG.
- Acute kidney injury (renal failure): oliguria/anuria (clinical), rising blood creatinine/ urea (laboratory).
- (Hemoglobin-/myoglobin-urea :) dark brown urine (clinical), urine dipsticks, other evidence of intravascular hemolysis or generalized rhabdomyolysis (muscle aches and pains, hyperkalemia) (clinical, laboratory).
- Supporting laboratory evidence of systemic envenoming (see above).

Local envenoming

- Local swelling involving more than half of the bitten limb (in the absence of a tourniquet) within 48 hours of the bite. Swelling after bites on the digits (toes and especially fingers).
- Rapid extension of swelling (for example, beyond the wrist or ankle within a few hours of bite on the hands or feet).
- Development of an enlarged tender lymph node draining the bitten limb

Anti-venom Reactions: Early anaphylactic reactions: severe, life-threatening anaphylaxis can evolve so rapidly, epinephrine (adrenaline) should be given at the very first sign of a reaction, even when only a few spots of urticaria have appeared or at the start of itching, tachycardia or restlessness. The dose can be repeated every 5-10 minutes if the patient's condition is deteriorating. After epinephrine (adrenaline), an antihistamine anti-H1 blocker such as chlorphenamine maleate (adults 10 mg, children 0.2 mg/kg by intravenous injection over a few minutes) should be given followed by intravenous hydrocortisone (adults 100 mg, children 2 mg/kg body weight). The corticosteroid is unlikely to act for several hours, but may prevent recurrent anaphylaxis.

Observing the Response to anti-venom

- The patient feels better
- Spontaneous systemic bleeding (e.g. from the gums): This usually stops within 15-30 minutes.
- Blood coagulability (as measured by 20WBCT): This is usually restored in 3-9 hours
- Neurotoxic envenoming of the post-synaptic type (cobra bites) may begin to improve as early as 30 minutes after antivenom, but usually takes several hours.
- Active hemolysis and rhabdomyolysis may cease within a few hours and the urine returns to its normal color.

Treatment of the Bitten part: Infection is common in snake bite, antibiotics line amoxycillin or cephalexin is given. The bitten limb, which may be painful and swollen, should be nursed in the most comfortable position, but not excessively elevated as this may reduce arterial perfusion pressure in a tensely swollen limb and increase the risk of intra-compartmental ischemia. Bullae may be large and tense but they should be aspirated only if they seem likely to rupture. The appearance of an immobile, tensely-swollen, cold and apparently pulseless snake-bitten limb may suggest possibility of increased intercompartmental pressure, needing surgical referral for Fasciotomy.

Rehabilitation: In patients with severe local envenoming, the limb should be maintained in a functional position. Restoration of normal function in the bitten part should be started by simple exercises while the patient is still in hospital. After the patient has been discharged from hospital rehabilitation can be done at home. Relatives can be instructed and given a time table of rehabilitation activities. Conventional physiotherapy may accelerate functional recovery of the bitten limb.

Deciding whether further dose(s) of anti-venom are needed

Criteria for giving more anti-venom:

Persistence or recurrence of blood in coagulability after 6 hours or of bleeding after 1-2 hours same dose should be repeated. Transfusion of FFP/platelets/whole blood to replenish clotting factors should be considered.

DROWNING

All submersion injuries should be termed "drowning". Types and causes of death due to drowning.

- Dry drowning occurs when there is laryngospasm leading to hypoxia followed by loss of consciousness.
- Wet drowning in which water is aspirated into the lungs which causes dilution and washout of pulmonary surfactant and effect ventilation-perfusion miss-match.
- Freshwater aspiration leads to transient hemodilution, hemolysis and hyponatremia.

- Saltwater aspiration leads to hemoconcentration, hypernatremia and hyperkalemia.

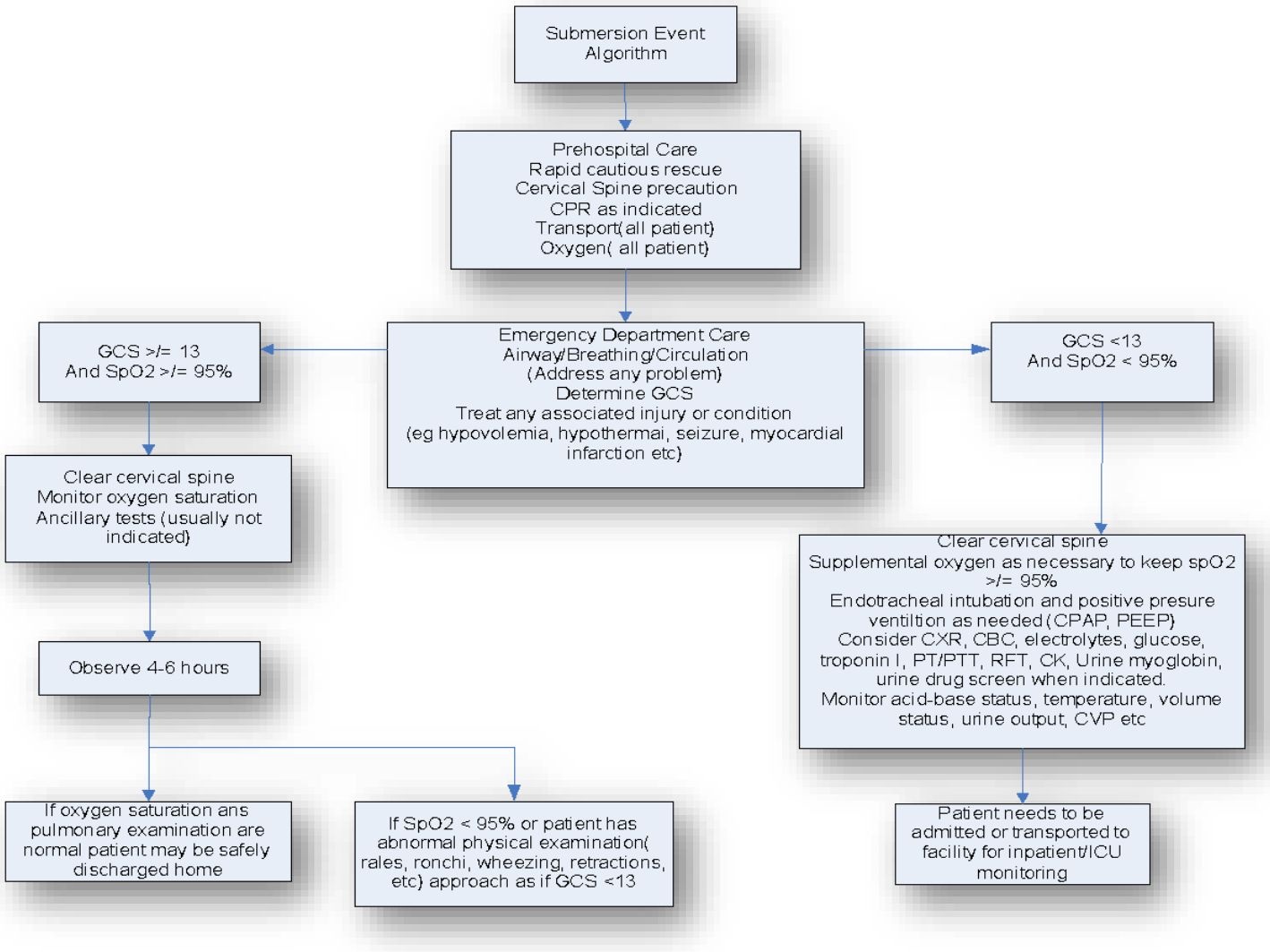

Figure 10.1 Submersion event algorithm.

MUSHROOM POISONING

Fortunately, majority of the reported mushroom exposure have benign outcome. The most important factor in preventing mushroom poisoning is to avoid eating wild mushroom. Mushroom toxins are heat labile and so are not destroyed and deactivated by cooking, canning, freezing, drying, and other means of food preparation. Toxicity may vary on the amount ingested, age of the mushroom, the season, geographic location and the way the mushrooms are prepared prior to ingestion, so one person may show significant effect whereas others may be asymptomatic after ingestion of same mushroom. In general, if toxicity begins within 2 hours of ingestion of a mushroom, the clinical course will be benign. If symptoms begin >/= 6 hours after ingestion the clinical course will be more serious and potentially fatal.

Table 10.4 Symptoms, toxicity and treatment of mushroom poisoning

Symptoms	Toxicity	Treatment

GI Symptoms Onset < 2 hrs. Onset 6-24 hrs.	Nausea, vomiting, diarrhea (occasionally bloody) Initial: Nausea, vomiting, diarrhea Day 2: rise in AST, ALT levels Day 3: hepatic failure	IV Hydration Antiemetic IV hydration, glucose, monitor AST, ALT, bilirubin, blood urea, creatinine, PT/INR Activated Charcoal 0.5-1 gm/kg Consider Penicillin G: 300.000-1,000,000 units/kg/d (blocks the uptake of amatoxin into the liver cells Silymarin, 20-40 mg/kg/d (acts as free radicle scavenger and interrupt the enterohepatic circulation of amatoxin when given orally.
Muscarinic Syndrome	SLUDGE (salivation/ lacrimation/urination/defecation/GI hypermotility/emesis)	Supportive: Atropine 0.01 mg/kg repeat as needed for excessive secretion.
CNS excitation/hallucinations Onset<30 mins	Intoxication, dizziness, ataxia, visual disturbance, seizure, tachycardia, hypertension, warm dry skin, dry mouth, mydriasis (anticholinergic effects)	Supportive: sedation with Diazepam 0.1 mg/kg for children and 5mg for adult IV, or Phenobarbitone 0.5 mg/kg or 30 mg IV for adult
Disulfiram reaction 2-72 hrs. after mushroom and < 30 mins after alcohol	Headache, flushing, tachycardia, hyperventilation, shortness of breath, palpitation	Supportive: IV Hydration Beta Blocker/CCB for SVT Norepinephrine for refractory hypotension

HIGH ALTITUDE MEDICAL ILLNESSES (HAMI)

High altitude (>2440 m or >8000 ft) is a hypoxic environment and hypoxia not the hypobaric at high altitude is responsible for altitude illness. High altitude medical illnesses typically occur at altitude > 8000 feet and rarely occurs at altitude 6000-8000 feet. There is no predilection based on gender. HAMI are more likely if the person rapidly ascents without acclimatization. It can occur in those with no prior problems with altitude exposure and can recur (no consistent tolerance or "immunity".

Table 10.5 Suggested Treatment Option for High- Altitude illness

	Symptoms	**Treatment**
Mild Acute Mountain Sickness (AMS)	Headache, GI disturbance, dizziness or light-headedness, and sleep disturbance	Termination of ascent Descent to lower altitude or acclimatization at the same altitude Acetazolamide 125-250 mg PO twice a day to speed acclimatization Symptomatic treatment with analgesic and antiemetics.

Moderate Acute Mountain Sickness (AMS)	Headache, GI disturbance, dizziness or light-headedness, and sleep disturbance	Immediate descent for worsening symptoms Low flow oxygen if available Acetazolamide 250 mg PO twice a day and/or dexamethasone 4 mg PO every 6 hrly Hyperbaric bag therapy if available
High Altitude Cerebral Edema (HACE)	Severe headache Nausea, vomiting Symptoms of AMS Altered mental status Ataxia, seizure Stupor and coma	Immediate descent or evacuation Oxygen 2-4 L/min or titrate to SpO2 >90 % Dexamethasone 8 mg PO, IM, IV then 4 mg 6 hrly Hyperbaric bag therapy if patient cannot descent
High Altitude Pulmonary Edema (HAPE)	Dry cough later productive cough Decreased exercise performance Dyspnea on exertion Increased recovery time with exercise	Immediate descent or evacuation to medical facility Oxygen 4 L/min or titrate to SpO2 >90 % Nifedipine extended release 30 mg PO twice a day Hyperbaric therapy if patient cannot descent Continuous positive airway pressure (CPAP) Measures to minimize patient exertion and kept patient warm Dexamethasone 4 mg PO, 6 hrly if cerebral signs present Consider Salbutamol nebulization 4 hrly

GENERAL MANAGEMENT OF POISONED PATIENT

Poisoning occurs when exposure to a substance adversely affects the function of any system within an organism. Poison exposure may be occupational, environmental, recreational, or medicinal. Poisoning may occur from varied portal of entry including inhalation, ingestion, cutaneous and mucous membrane exposure and injection.

Resuscitation

The first priority in treatment of poisoned patient is assessment and stabilization of cardiopulmonary function. Once the airway and the respiratory status, blood pressure and pulse are stabilized abnormalities of core (rectal) temperature, oxygen saturation, and hypoglycemia are addressed. Patient may have altered mental status because of hypoxia, opioid intoxication, hypoglycemia, a Wernicke encephalopathy, empiric administration of antidotes (the “coma cocktail”) including supplemental oxygen, naloxone, glucose and thiamine should be considered after the medical history and vital signs.

ED Diagnosis

History: Obtain as much information as possible about the exposure. Ask about the agent or the drug, estimate amount or dose, the route of exposure as well as whether other individuals were exposed and intention. Ask about the environment in which the patient was found, the presence of empty pill bottles or container, any smells of unusual material at home, occupation or the hobbies of the patient, and the presence of suicide note.

Physical examination: An organized approach is recommended. Undress the patient completely. Assess the general appearance of the patient and note any agitation, confusion, or obtundation. Examine the skin for any cyanosis or flushing, excessive diaphoresis or dryness, signs of injury or injection, ulcers or bullae. Examine the eye for pupil size, reactivity, nystagmus, deconjugate gaze, or excessive lacrimation. Examine oropharynx for hypersalivation or excessive dryness. Auscultate the lung field to assess for bronchorrhea or wheezing and the heart for rate, rhythm and regularity. Examine the abdomen noting for bowel sound, enlarged bladder and abdomen tenderness and rigidity. Evaluate the extremities for muscle tone.

Toxidromes: Toxidromes are collection of physical findings that occur with specific classes of substances. In clinical practice, the identification of a specific toxidrome is helpful in establishing potential toxic agents.

Table 10.6 Toxidromes

Toxidrome	Representative Agent	Most common findings	Potential Intervention
Opioid	Heroin Morphine	CNS depression, mitosis, respiratory depression	Ventilation or Naloxone
Sympathomimetic	Cocaine Amphetamine	Psychomotor agitation, mydriasis, diaphoresis, tachycardia, hypertension, hyperthermia	Cooling, sedation with benzodiazepines, hydration
Cholinergic	Organophosphate insecticide Carbamate insecticides	Muscarinic effects (SLUDGE) Nicotinic effects (muscle fasciculation and weakness)	Airway protection and ventilation, atropine, Pralidoxime
Anticholinergic	Atropine Scopolamine	Altered mental status, mydriasis, dry flushed skin, urine retention, decreased bowel sounds, hyperthermia	Physiostigmine (if appropriate), sedation with benzodiazepine, cooling, supportive management
Salicylate	Aspirin Oil of wintergreen	Low grade fever, ketonuria, Death may result from acute lung injury or cerebral edema	Multidose activated charcoal, alkalinisation of urine with Sodium-bicarbonate or Potassium repletion, haemodialysis

Sedative-hypnotic	Barbiturates Benzodiazepines	Stupor to coma, respiratory depression, apnea, bradycardia	Ventilatory support
Hypoglycaemic	Sulfonylureas Insulin	Altered mental status, diaphoresis, tachycardia, hypertension	Glucose containing solution IV and oral feedings if possible, frequent glucose measurement, octreotide
Hallucinogenic	Phencyclidine Lysergic acid diethylamide Mescaline	Hallucination, dysphoria, anxiety	Generally supportive
Serotonin	SSRIs Meperidine TCA	Altered mental status, increased muscle tone, hyperreflexia, hyperthermia	Cooling, sedation with benzodiazepine, supportive management
Extrapyramidal	Haloperidol Risperidone Olanzapine	Dystonia, torticollis, tremor, muscle rigidity	Dyphenhydramine, Benztropine, Benzodiazepine

General Decontamination: The general approach to toxins involves removal of patient from the substance and the substance from the patient. Toxins from outside of the body are washed away. For toxins within the body, either the toxins can be bound within the gut lumen to make it unavailable for absorption or its elimination from the gut, blood, or tissues.

Gross decontamination: Surface decontamination by completely undressing patient and thoroughly washing them with copious amounts of water. If possible, surface decontamination should occur prior to the patient`s entry into the health facility.

Eye decontamination: Ocular exposure are treated with copious irrigation using isotonic crystalloids either NS or RL typically 1 to 2 litres per eye depending on the agent. Application of ocular anesthetics may be necessary.

GI decontamination: GI decontamination should never be initiated as a punitive action. The three-general method of decontamination involve removing the toxin from the stomach via mouth, binding it inside the gut lumen or enhancing transit through the intestine.

Gastric Emptying

Emesis: Ipecac Syrup has not been shown to alter outcome compared to treatment with activated charcoal, its use also delays the time to administration of oral antidote. Dose of 15 ml for children and 30 ml for adult given with sips of water.
Orogastric Lavage: Clinical efficacy of orogastric lavage has been found only in small number of patients in whom it is initiated within 1 hour of ingestion.

Toxins adsorption in the Gut

Activated charcoal: Clinical benefit is more likely if activated charcoal is administered within 1 hour of toxin ingestion, but that potential benefit of administration after more than 1 hour cannot be excluded. Recommended dose is 10:1 ratio or 1gm/kg, whichever is larger.
Multidose Activated charcoal: This entails repeated use of activated charcoal to enhance elimination of ingested toxins. Toxins with a long half-life and small volume of distribution are most likely to have their elimination accelerated by repetitive dose of activated charcoal. First dose of 1gm/kg followed by subsequent dose of 0.25-0.5 gm/kg (12.5 gms) given 4 hrly.

Enhancement of Bowel transit

Cathartics: Activated charcoal is often administered with an osmotic cathartic such as 70% Sorbitol (1gm/kg). Evidence do not support giving cathartics.
Whole bowel irrigation: Whole bowel irrigation is performed by instillation of large volumes of polyethylene glycol in an osmotically balanced electrolyte solution that produces rapid catharsis by mechanically forcing ingested substance through the bowel at an accelerated rate.

Enhance elimination

Urinary alkalinisation: Manipulation of urinary pH toward alkaline is done by enhance the clearance of specific toxins. It is achieved by administration of Sodium Bicarbonate 1-2 mEq/kg IV over 1 hr until urine pH is 7.5-8.5.
Forced diuresis: Has never been shown to be effective.

Hemodialysis/Hemoperfusion: HD is generally preserved for specific toxins that are both potentially life threatening and amenable to removal by this method.

Table 10.7 Common Antidotes, initial Dosages and indications:

Antidote	Pediatric dose	Adult dose	Indication

Acetylcysteine	150 mg/kg IV load over 60 minutes, followed by 50 mg/kg IV over 4 hrs. and then 100 mg/kg over 16 hrs.	150 mg/kg IV load over 60 minutes, followed by 50 mg/kg IV over 4 hrs. and then 100 mg/kg over 16 hrs.	Acetaminophen (Paracetamol)
Activated Charcoal	1gm/kg PO	50-100gms	Most ingested poisons
Anti-venom Fab	4-6 vials initially over 1 hr., may be repeated to gain control of progressive symptoms	10 vials initially over 1 hr., may be repeated to gain control of progressive symptoms	envenomation by poisonous snakes
Atropine	0.01-0.02 mg/kg IV, repeat every 5 min until tracheobronchial secretion attenuates	1mg IV, repeat every 5 min until tracheobronchial secretion attenuates	Organophosphate
Calcium gluconate 10%	0.6-0.8 ml/kg IV	10-30 ml IV	Hypermagnesemia Hypocalcemia
Dextrose (glucose)	0.5 gm/kg IV	1gm/kg IV	Insulin Oral Hypoglycemics
Ethanol (10% for IV administration)	10ml/kg IV over 30 mins then 1.2ml/kg/hr.	10ml/kg IV over 30 mins then 1.2ml/kg/hr.	Ethylene Glycol Methanol
Flumazenil	0.01 mg/kg IV	0.2 mg/kg IV	Benzodiazepines
Fomepizole	15 mg/kg IV, then 10 mg/kg every 12 hrly	15 mg/kg IV, then 10 mg/kg every 12 hrly	Methanol Ethylene glycol Disulfiram ethanol interaction
IV lipid emulsion 20%	1.5ml/kg bolus over 1 min (may be repeated two times at 5 min interval) followed by 0.25 ml/kg/min IV	100 ml IV bolus over 1 min, followed by 400 ml IV over 20 mins	IV Bupivacaine Rescue therapy for calcium channel blocker and Beta blocker
Naloxone	As much as is needed Typically starting dose of 0.01 mg IV	As much as is needed Typically starting dose of 0.4-2.0 mg IV	Opioid Clonidine
Octreotide	1 mcg/kg SC every 6 hrs.	50-100 mcg SC every 6 hrs.	Refractor Hypoglycemia after oral hypoglycemic agent ingestion

Physostigmine	0.02 mg/kg IV	0.5- 2.0 mg/kg slow IV over 2-5 min	Anticholinergic agents (not cyclic antidepressant)
Pralidoxime (2 PAM)	20-40 mg/kg IV over 5-10 min, followed by 20 mg/kg/hr. infusion	1-2 gm IV over 5-10 min, followed by 500 mg/h infusion	Cholinergic agent
Protamine	1 mg neutralizes 100 units of unfractionated heparin, administered over 15 min. 0.6 mg/kg IV empiric dose	1 mg neutralizes 100 units of unfractionated heparin, administered over 15 min. 20-50 mg IV empiric dose	Heparin
Pyridoxin	Gram for gram of ingestion if amount of isoniazid is known. 70 mg/kg (max 5 gm IV)	Gram for gram of ingestion if amount of isoniazid is known. 70 mg/kg (max 5gm IV)	Isoniazid Hydrazine
Sodium bicarbonate	1-2 mEq/kg IV bolus followed by 2 mEq/kg/h IV infusion	1-2 mEq/kg IV bolus followed by 2 mEq/kg/h IV infusion	Sodium Channel Blocker (TCA) For Urinary alkalization
Thiamine	5-10 mg IV	100 mg IV	Wernicke syndrome Wet beriberi
Vitamin K	1-5 mg/d PO	20 mg/d PO	Anti-coagulant rodenticides

References

1. Tintinalle`s, Emergency Medicine, A Comprehensive Study Guide.
2. Guideline for the management of snake-bites, WHO, 2010.