
CHAPTER 14 – GENERAL EMERGENCIES AND MAJOR TRAUMA

First Nations and Inuit Health Branch (FNHIB) Clinical Practice Guidelines for Nurses in Primary Care.
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RESPONDING TO GENERAL EMERGENCIES AND MAJOR TRAUMA

For any emergency, always remember your ABCs (airway, breathing, circulation) as the priority. Primary survey and resuscitation are followed by secondary survey, definitive care and, finally, transport.

The primary survey and resuscitation are done simultaneously. During this period, a patent airway is established while control of the cervical spine is maintained. Maintenance of airway patency is obviously the most critical factor, and cervical spine injury should be assumed in every seriously injured individual, until proven otherwise.

The next priorities are as follows:

- Adequate ventilation
- Treatment of shock
- Identification of life-threatening injuries

See “Primary Survey” and “Resuscitation” sections under “Responding to General Emergencies and Major Trauma” in the pediatric Chapter 20, “General Emergencies and Major Trauma” for a general approach to use with all clients in an emergency.

GENERAL EMERGENCY SITUATIONS

ANAPHYLAXIS

Anaphylaxis is an acute hypersensitivity reaction with multi-organ-system involvement that has a rapid onset and may cause death.^{1,2} The symptoms develop over several minutes to several hours,³ may involve multiple body systems (for example, skin [90% of episodes], respiratory [70% of episodes], gastrointestinal [40% of episodes], circulatory [35% of episodes]³) and may progress to unconsciousness as a late event in severe cases. Rarely is unconsciousness the sole manifestation of anaphylaxis. The severity and differentiation of an anaphylaxis reaction can be implied by the presence of cutaneous or multi-system findings, in addition to the involvement of cardiovascular and/or respiratory findings.⁴

Anaphylaxis is a medical emergency and must be distinguished from fainting (vasovagal syncope), which is more common and benign. Rapidity of onset is a key difference. When a person faints, the change from a normal to an unconscious state occurs within seconds. Fainting is managed simply by placing the patient in a recumbent position and elevating the feet. Fainting is sometimes accompanied by brief clonic seizure activity, but this generally requires no specific treatment or investigation.

CAUSES

The most common causes of fatal anaphylactic reactions are:

- Drugs (for example, penicillin and cephalosporin antibiotics, NSAIDs [nonsteroidal anti-inflammatory drugs] including ASA [acetylsalicylic acid], anesthetics)¹
- Foods (most common in children,⁵ for example, peanuts, shellfish, nuts, sesame seeds, fish products, eggs)⁵
- Insect venom (for example, bees, wasps)

In contrast, fatal reactions to vaccines and latex rubber are rare.⁶

HISTORY

Most anaphylactic episodes involve an immediate hypersensitivity reaction following exposure to an allergen.¹ Symptoms often occur within 5–30 minutes of exposure to trigger factor. Anaphylaxis can be biphasic with recurrence of symptoms occurring, usually within eight to ten hours, but occasionally up to 72 hours after the resolution of the initial anaphylactic event.⁷ Anaphylaxis may be fatal within minutes, usually through cardiovascular or respiratory compromise.¹

The signs and symptoms may include:⁸

Skin

- Flushing
- Feeling of warmth
- Itching (may begin on palms and soles, may include the external auditory canal)
- Urticaria (hives)
- Angioedema (facial edema)
- Morbilliform rash
- Piloerection (hair standing on end)

Oral

- Itching or tingling or edema of lips, tongue, palate or uvula
- Metallic taste

Gastrointestinal

- Nausea, vomiting, abdominal pain, diarrhea, difficulty swallowing

Respiratory

- Pruritus of the larynx and tightness in the throat
- Dysphagia, dysphonia or hoarseness
- Respiratory difficulties: shortness of breath, wheezing, cough dyspnea, tightness of the chest
- Nasal symptoms including, itching, congestion, sneezing, rhinorrhea

Ocular

- Periorbital itching, erythema, tearing or edema
- Red, itchy eyes⁵
- Conjunctival erythema

Neurologic

- Anxiety
- Apprehension, sense of impending doom
- Confusion
- Seizures
- Headache

Cardiovascular

- Feeling faint, dizziness, syncope
- Palpitations, tachycardia

Hypotension

- Lower back pain due to uterine cramping in women
- Cardiovascular collapse can occur without respiratory symptoms

SEVERE REACTION

- Severe respiratory distress (lower respiratory obstruction characterized by high-pitched wheezing, upper airway obstruction characterized by stridor)
- Difficulty speaking, hoarseness
- Difficulty swallowing
- Agitation
- Shock
- Loss of consciousness

PHYSICAL FINDINGS

- Tachycardia
- Tachypnea, laboured respiration
- Blood pressure low-normal (client hypotensive if in shock)
- Pulse oximetry may show hypoxia
- Client in moderate-to-severe distress
- Use of accessory muscles of respiration
- Chest: air entry reduced, mild-to-severe wheezing
- Stridor, rapid or shallow breathing, cyanosis⁹
- Client flushed and diaphoretic
- Generalized urticaria (hives)
- Facial edema, angioedema
- Diminished level of consciousness
- Confusion, anxiety, agitation (caused by hypoxia)⁹
- Skin feels cool and clammy

DIFFERENTIAL DIAGNOSIS

- Asthma
- Acute anxiety (panic attack), breath-holding episode in a child⁹
- Foreign-body aspiration
- Angioedema
- Pulmonary embolism
- Vasovagal syncope (fainting) (pulse and BP are generally normal, and there is usually no evidence of airway symptoms)⁹
- Hypoglycemia⁹
- Seizure disorder⁹
- Septic shock⁹
- Mastocytosis, carcinoid syndrome, scromboid poisoning⁹

CASE DEFINITION

As anaphylaxis may present with a number of symptoms and/or signs, a case definition provides a standard approach to describing the degree of clinical severity and the level of diagnostic certainty. The case definition and guidelines for clinical application, including reporting adverse events, were published by the Brighton Collaboration Anaphylaxis Working Group in “*Anaphylaxis: Case definition and guidelines for data collection, analysis and presentation of immunization safety data*”.¹⁰

COMPLICATIONS

- Hypoxia
- Shock
- Airway obstruction due to edema of upper airway
- Convulsions
- Aspiration
- Death

DIAGNOSTIC TESTS

Diagnosis made on clinical findings.¹

MANAGEMENT

Goals of Treatment

- Improve oxygenation
- Alleviate symptoms
- Prevent complications
- Prevent recurrence
- Treat as a medical emergency and manage airway, breathing and circulation⁹

Early recognition and treatment of anaphylaxis is vital.

Nonpharmacologic Interventions

- Place the client in a recumbent position (if tolerated), elevating the feet
- Establish an oral airway if necessary

Adjuvant Therapy

Should anaphylaxis progressively become severe:

- Give oxygen by mask, 10–12 L/min or more; keep oxygen saturations > 97% to 98%
- Start intravenous (IV) therapy with normal saline or Ringer’s lactate to keep vein open, unless severe anaphylaxis and signs of shock are evident; see section “Shock” later on in this chapter for details of fluid resuscitation in shock

Pharmacologic Interventions

Epinephrine is the drug of choice for the treatment of anaphylaxis, and the IM route is preferred.¹¹

There are no absolute contraindications to the use of epinephrine for the treatment of anaphylaxis.^{7,11}

Speedy intervention is of paramount importance. Failure to use epinephrine promptly is more dangerous than using it quickly but improperly. Failure to administer epinephrine promptly and use of antihistamines and salbutamol rather than epinephrine are important errors in the treatment of anaphylaxis.⁷

Promptly administer:

epinephrine 1 mg/mL solution (may be labelled 1:1000), 0.2–0.5 mg = 0.2–0.5 mL intramuscular (IM)^{7,11} in the midanterolateral thigh to achieve peak plasma and tissue concentrations rapidly¹²

Repeat at 5–15 minute intervals, as necessary, depending on the severity of the reaction, to control symptoms and to sustain or increase blood pressure.¹¹

Published national anaphylaxis guidelines agree that epinephrine is fundamental to acute management, although they do not agree on the initial dose or route of injection.¹² The subcutaneous route and injecting in the opposite limb, when immunization is the cause, can also be used.¹³

Epinephrine Dose in Children

Calculations based on body weight are preferred when weight is known. When body weight is not known, the dose of epinephrine (1:1000) can be approximated from the subject’s age (see Table 1, “Epinephrine Dose on the Basis of Age”).¹⁴

Table 1 – Epinephrine Dose on the Basis of Age

Age	Dose
2–6 months*	0.07 mL (0.07 mg)
12 months*	0.1 mL (0.1 mg)
18 months* to 4 years	0.15 mL (0.15 mg)
5 years	0.2 mL (0.2 mg)
6–9 years	0.3 mL (0.3 mg)
10–13 years	0.4 mL (0.4 mg)
≥ 14 years	0.5 mL (0.5 mg)

*Doses for children between the ages shown are approximated (the volume being intermediate between the values shown or increased to the next larger dose, depending on practicability).

Source: *Canadian Immunization Guide*, 7th ed. (Health Canada, 2006).

Excessive doses of epinephrine can compound a patient's distress by causing palpitations, tachycardia, flushing and headache. Although unpleasant, such side effects pose little danger. Cardiac dysrhythmias may occur in older adults but are rare in otherwise healthy children and young adults.

Some drugs can interfere with the efficacy of epinephrine. Beta-blockers (for example, atenolol and metoprolol) block the effects of epinephrine (and salbutamol). Patients taking beta-blockers may have more severe anaphylactic reactions or reactions that are refractory to epinephrine. Glucagon (1–2 mg IV administered over 5 minutes in adults may be administered to counteract the effects of the beta-blocker in such patients).¹⁴

Angiotensin converting enzyme inhibitors and angiotensin II receptor blockers may also interfere with the effects of epinephrine and result in more severe or prolonged symptoms.⁷

In addition to epinephrine, the following medications may be administered depending on the circumstances:

Antihistamines⁷

diphenhydramine hydrochloride (Benadryl) for itching and hives 25–50 mg IV/IM in adults (1 mg/kg IV/IM to a maximum of 50 mg in children)⁷

Diphenhydramine is an adjunct to rather than a substitute for epinephrine and

ranitidine 50 mg IV/IM in adults (1 mg/kg to a maximum of 50 mg in children). Dilute in 5% dextrose to a volume of 20 mL and inject over 5 minutes

Corticosteroids^{7,14}

Corticosteroids are commonly administered during anaphylactic reactions although there is little evidence that they are of benefit. They are unlikely to be helpful in the treatment of acute anaphylaxis, but may help to prevent biphasic or protracted reactions;⁷ thus, preparation and administration of a dose of corticosteroid should not take priority over prompt administration (or re-administration) of epinephrine.

methylprednisolone 1–2 mg/kg/day IV/IM divided q6h
or

oral prednisone 0.5 mg/kg/day

Bronchodilator

salbutamol (Ventolin), via nebulizer 2.5–5 mg, repeat as necessary¹⁴

Recommended Epinephrine Kit Contents

The Canadian immunization guidelines suggest the following content; regional policies may vary.

- Copy of the anaphylaxis procedures and doses recommended of epinephrine and diphenhydramine for weight and age
- Two 1 cc syringes with attached needles (one 25 gauge, 5/8" needle; one 25 gauge, 1" needle)
- 2 ampuls of epinephrine 1:1000 (check expiry date monthly and replace once expired)
- 1 vial of diphenhydramine (pills or oral solutions optional, check expiry date monthly and replace once expired)
- One 25 gauge, 5/8" needle (extra)
- One 25 gauge, 1" needle (extra)
- 2 alcohol swabs (optional)¹⁵

Because anaphylaxis is rare, epinephrine vials and other emergency supplies should be checked regularly and should be replaced before they are outdated.

Monitoring and Follow-up

Severe Anaphylaxis

Monitor airway, breathing and circulation (ABC), vital signs and cardiorespiratory status every 15 minutes until client's condition stabilizes.

Since 20% of anaphylaxis episodes follow a biphasic course of recurrence, the reaction after a 2–9 hour asymptomatic period, hospitalization or a long period of observation is recommended. For all but the mildest cases of anaphylaxis, patients should be hospitalized overnight or monitored for at least 12 hours.¹⁵

Prevention

If anaphylaxis is a potential recurrent risk, consider the use of an epinephrine self-injector (for example, Epipen or Twinject). Assess and educate potential users regarding the proper use and storage of the device.¹³ Consider having a personalized anaphylaxis emergency action plan and an up-to-date medical identification.¹⁶

Appropriate Consultation

Consult a physician as soon as client's condition stabilizes; discuss use of IV steroids.

Referral

Medevac as soon as possible. In all but the mildest cases, clients with anaphylaxis should be hospitalized overnight or monitored for at least 12 hours.

SHOCK

Shock is an acute widespread process of impaired tissue perfusion that results in cellular, metabolic and hemodynamic alterations. Ineffective tissue perfusion occurs when an imbalance develops between cellular oxygen supply and cellular oxygen demand, which can occur for a number of reasons and eventually result in cellular dysfunction and death.¹⁷

Shock is categorized in many ways, for example, according to the state of physiologic progression that has occurred:

- *Compensated shock*: vital organ perfusion is maintained by endogenous compensatory mechanisms
- *Decompensated shock*: compensatory mechanisms have failed; associated with hypotension and impairment of tissue perfusion
- *Irreversible shock*: multiple end-stage organ failure and death occur, despite occasional return of spontaneous cardiorespiratory function

Arterial blood pressure is often preserved by compensatory vasoconstrictive mechanisms until very late in shock. An over-reliance on arterial blood pressure readings can delay recognition and timely treatment of shock.

TYPES OF SHOCK

- *Hypovolemic shock*: inadequate perfusion of vital organs results from a loss of circulating or intravascular volume¹⁷ (hemorrhage, trauma, gastrointestinal [GI] fluid loss)
- *Cardiogenic shock*: due to the inability of the heart to pump blood to tissues (decreased cardiac output), as in congestive heart failure, myocardial ischemia
- *Distributive shock*: result from maldistribution of circulating blood volume causing vasodilation. It can be classified further as septic, neurogenic or anaphylactic. Septic results from microorganisms entering the body. Anaphylactic shock is the result of a severe antibody-antigen reaction (histamine). Neurogenic shock is the result of loss of sympathetic tone (spinal cord injury)¹⁷
- *Obstructive (mechanical) shock*: obstruction of cardiac filling such as that caused by pericardial tamponade or tension pneumothorax
- *Dissociative shock*: oxygen is not released from hemoglobin to the cells (as in carbon monoxide poisoning)

HISTORY

- Nausea
- Lightheadedness, faintness
- Thirst
- Loss of consciousness

Other symptoms depend upon underlying cause.

PHYSICAL FINDINGS

ABCs are the priority.

Physical findings depend on whether the client is in early or late shock.

Early Hypovolemic Shock

Loss of approximately 15% to 25% of blood volume is enough to cause significant signs and symptoms:

- Tachycardia
- Blood pressure normal
- Postural blood pressure drop present (orthostatic hypotension)
- Narrowed pulse pressure
- Faint or weaker pulse
- Thirst
- Diaphoresis
- Delayed capillary refill possible
- Anxiousness, restlessness
- Tachypnea, increased respiratory depth¹⁷
- Decreased urine output¹⁷
- Pale cool skin¹⁷
- Jugular vein appears flat¹⁷

Late Hypovolemic Shock

Caused by loss of 30% to 45% of blood volume and can be life-threatening.

- Hypotension
- Tachycardia more pronounced
- Pulse weak and thready
- Oxygen saturation decreased
- Respiratory distress¹⁷
- Oliguria¹⁷
- Skin becomes ashen, cold and clammy with marked, delayed capillary refill¹⁷
- Confusion and decreased level of consciousness¹⁷

Tachycardia is one of the early indicators of volume depletion. It may not be as apparent in elderly clients as in younger ones. Tachycardia may be mild if the client is taking certain medications (for example, beta-blockers, calcium channel blockers).

DIFFERENTIAL DIAGNOSIS

- Sepsis
- Myocardial infarction
- Pulmonary embolism
- Anaphylaxis
- Status asthmaticus
- Head, spinal injury¹⁸
- Drug-induced symptoms¹⁸

COMPLICATIONS

- Cardiovascular: ventricular failure, microvascular thrombosis, angina, myocardial ischemia or infarction¹⁷
- Neurologic: sympathetic nervous system dysfunction, cardiac and respiratory depression, thermoregulatory failure, coma¹⁷
- Pulmonary: acute respiratory failure, acute lung injury¹⁷
- Renal: acute tubular necrosis, renal failure¹⁷
- Hematologic: disseminated intravascular coagulation¹⁷
- Gastrointestinal: gastrointestinal tract failure, hepatic failure, pancreatic failure¹⁷
- Multi-system organ failure¹⁷
- Death

DIAGNOSTIC TESTS

- Pulse oximetry (oxygen saturation)
- ABG, serum lactate, CBC, electrolytes, BUN, Cr, glucose, PTT, INR, AST, ALT¹⁸

MANAGEMENT

ABCs are the priority.

Goals of Treatment

- Restore circulating blood volume
- Improve oxygenation of vital tissues
- Prevent ongoing volume losses
- Identify underlying cause¹⁷
- Identify and correct cause of lactic acidosis¹⁷
- Maintain surveillance for complications and provide comfort¹⁷

Nonpharmacologic Interventions

- Assess and stabilize ABC
- Ensure that airway is patent and ventilation is adequate
- Insert oral airway and ventilate with Ambu bag (using oxygen) as needed

- Control any external bleeding; use direct pressure to control bleeding from external wounds
- Put in head-down position

Adjuvant Therapy

- Give oxygen at 10–12 L/min or more by non-rebreather mask; keep oxygen saturation > 97% to 98%
- Start 2 large-bore IV lines (14- or 16-gauge or greater) with normal saline or Ringer's lactate
- Give 1–2 L IV fluid rapidly as a bolus over 15 minutes
- Reassess for signs of continuing shock
- If shock persists, continue to administer fluid in 1 L boluses and reassess after each bolus
- Adjust IV rate according to clinical response
- Ongoing IV therapy is based on response to initial fluid resuscitation, continuing losses and underlying cause
- Aim for heart rate < 100 bpm, systolic blood pressure > 90 mm Hg

The amount of fluid required for resuscitation is difficult to predict on initial assessment.

Caution in Cases of Internal Hemorrhage

The use of large amounts of IV fluids in a client with uncontrolled internal hemorrhage from blunt or penetrating trauma may increase the internal bleeding and ultimately lead to death. Administration of IV fluids while increasing blood pressure will also dilute clotting factors and cause more hemorrhage. Use fluids judiciously to maintain peripheral perfusion. Early blood transfusion and surgical intervention to achieve homeostasis is very important in this situation.

After Initial Resuscitation

- Insert indwelling urinary catheter
- Insert a nasogastric tube prn

Monitoring and Follow-Up

- Monitor ABC, vital signs (including pulse oximetry) and level of consciousness every 15 minutes until condition is stable
- Frequent reassessment for continuing blood loss is important
- Monitor hourly intake and urine output
- Identify and manage underlying cause of hypovolemia
- Assess stability of pre-existing medical problems (for example, diabetes mellitus)

Referral

Medevac as soon as possible.

COMA (NOT YET DIAGNOSED)

Altered level of consciousness indicating diffuse or bilateral cortical impairment of cerebral function, failure of brainstem-activating mechanisms (or both). Coma is the deepest state of unconsciousness where both arousal and awareness are lacking. Coma is a symptom, not a disease, and occurs as a result of some underlying process.¹⁹

CAUSES

- Bilateral cortical disease
- Compromise of reticular-activating system

Causes of coma can also be divided into structural or surgical and metabolic or medical:

- Structural or surgical causes: ischemic stroke, intracerebral hemorrhage, trauma and brain tumours¹⁹
- Metabolic or medical causes: drug overdose, infection, endocrine disorder, poisoning¹⁹

See “Differential Diagnosis” in this section.

INITIAL APPROACH TO CLIENT WITH COMA OF UNKNOWN ORIGIN

Perform primary survey (see “Primary Survey” under the section “Responding to General Emergencies and Major Trauma” in the pediatric Chapter 20, “General Emergencies and Major Trauma”).

The Glasgow Coma Scale can help identify the level of consciousness (see “Glasgow Coma Scale” under the section “Head Trauma” in this chapter).

MANAGEMENT**Nonpharmacologic Interventions****Acute Coma**

- Assess and stabilize ABC
- Assess changes in neurologic status¹⁹
- Stabilize cervical neck until traumatic injury is ruled out¹⁹
- Maintain surveillance for complications¹⁹
- Insert oral airway
- Place in recovery position, unless there are contraindications
- Check finger-stick glucose

Long-Term Coma Care

- Provide eye care¹⁹
- Protect skin integrity¹⁹
- Initiate range of motion exercises¹⁹

Adjuvant Therapy

- Give oxygen (10–12 L/min) by mask; keep oxygen saturation > 97% to 98%
- Start IV therapy with normal saline to keep vein open, unless there is evidence of shock (see section “Shock” in this chapter)

Pharmacologic Interventions

Rapidly administer:

thiamine, 100 mg IV (to prevent Wernicke-Korsakoff encephalopathy)

and

dextrose 50%, 25–50 mL preloaded IV (to treat hypoglycemia)

Do not withhold dextrose if thiamine is not available. A single dose of dextrose will not induce Wernicke-Korsakoff encephalopathy.

For patients with signs and symptoms of opioid intoxication give:^{20,21}

naloxone (Narcan), 0.4–2 mg IV, SC or IM to treat potential narcotic overdose (start with 2 mg; if no response in 3–5 minutes, give an additional 4 mg)

Anticonvulsant therapy may also be necessary to prevent further ischemic injury to the brain.¹⁹

Restrain the client if you suspect that naloxone may precipitate narcotic withdrawal.

If unsure whether naloxone is necessary discuss with a physician before administering.

Once the immediate life-threatening concerns have been addressed, the secondary survey can be carried out (see “Secondary Survey” under the section “Responding to General Emergencies and Major Trauma” in the pediatric Chapter 20, “General Emergencies and Major Trauma”).

- Monitor vital signs, including pulse oximetry
- Obtain abbreviated, targeted history
- In particular, determine if person has had any recent illness, antecedent fever, rash, vomiting or trauma or has any chronic illnesses; explore recent exposure to infection, medication or intoxicants

Past medical history and family history should be obtained when time permits.

Observations in the secondary survey should attempt to uncover signs of occult infection, trauma or toxic or metabolic derangements. Signs suggestive of specific toxidromes should be sought (*see section “Overdoses, Poisonings and Toxidromes” in this chapter*).

PHYSICAL FINDINGS

Level of Consciousness

Assess level of consciousness using the Glasgow Coma Scale (*see “Glasgow Coma Scale” under the section “Head Trauma” in this chapter*).

Respiratory Status

Respiratory status focuses on the evaluation of two things: 1) respiratory pattern and 2) airway status.¹⁹

Respiratory Pattern

- Control of breathing is centered in the brain, lower pons and medulla and is modulated by the cortical centres in the forebrain
- Respiratory abnormalities signify either metabolic derangement or neurologic insult
- Several patterns exist (for example, Cheyne-Stokes respiration, apneustic breathing, post-ventilation apnea, cluster, ataxic, central neurogenic hyperventilation)¹⁹

Airway Status

Airway maintenance, secretion control, cough, gag and swallow reflexes responsible for airway protection¹⁹

Ocular Findings

Pupillary Function

- Focuses on three areas: 1) estimation of pupil size and shape; 2) evaluation of pupillary reaction to light and; 3) assessment of eye movement¹⁹
- Remember that dilatation of pupils may be secondary to topical or systemic drugs
- Dilatation of pupils in an alert person is not likely attributable to increased intracranial pressure and herniation
- Dilatation of pupils in an unconscious patient may herald imminent uncal herniation
- Small reactive pupils generally indicate metabolic problem or diencephalic lesion
- Unilateral, dilated, fixed pupils indicate lesion of third nerve or uncal lesion
- Bilateral pinpoint pupils indicate pontine lesion
- Pupils fixed in midposition indicate midbrain lesion
- Bilateral large, fixed pupils indicate tectal lesion

With cerebral lesions, the eyes will deviate toward the side of the lesion, whereas with brainstem lesions, the eyes deviate away from the lesion.

About 5% of the normal population has anisocoria (asymmetric pupils).

A brief funduscopic exam may reveal papilledema or retinal hemorrhage.

Motor Examination

- Evaluation of motor function focuses on two areas:
 - 1) evaluation of muscle size and tone and;
 - 2) estimation of muscle strength¹⁹
- Muscle tone is assessed for signs of flaccidity, hypotonia, hypertonia, spasticity or rigidity¹⁹
- Try to elicit motor response to verbal or physical stimuli
- Assess muscle tone, strength and reflexes for normality and symmetry
- Ability of client to localize, as well as absence or presence of abnormal posture, helps in assessment of severity of involvement

Classifications of abnormal posturing include:

- Spontaneous: occurs without regard to external stimuli and possibly not by request¹⁹
- Localization: occurs when extremity opposite to the extremity receiving stimuli crosses the midline to remove the noxious stimuli¹⁹
- Withdrawal: occurs when the extremity receiving the stimuli flexes normally in order to avoid the noxious stimuli¹⁹
- Decorticate posturing (flexion of the upper extremities with extension of the lower extremities) suggests involvement of the cerebral cortex and subcortical white matter
- Decerebrate posturing (rigid extension of the arms and legs) usually represents added brainstem involvement at the level of the pons

DIFFERENTIAL DIAGNOSIS

Coma with no localizing central nervous system signs may be caused by:

- Metabolic insult, including hypoglycemia, uremia, Addison's disease, diabetic ketoacidosis, hypothyroidism, liver disease
- Children and young adults will often experience hypoglycemia and may present with coma after ingesting alcohol, including alcohol-containing mouthwashes
- Respiratory problems, including hypoxia, hypercapnia
- Intoxication, including that caused by barbiturates, alcohol, opiates, carbon monoxide, benzodiazepines
- Infections (severe, systemic), including sepsis, pneumonia, typhoid fever
- Shock, including hypovolemic, cardiogenic, septic, anaphylactic
- Epilepsy
- Hypertensive encephalopathy
- Hyperthermia (heat stroke), hypothermia

Coma with meningeal irritation but without localizing signs may be caused by:

- Meningitis
- Subarachnoid hemorrhage from ruptured aneurysm, arteriovenous malformation

Coma with focal brainstem or lateralizing signs may be caused by:

- Pontine hemorrhage
- Stroke (cerebrovascular accident [CVA])
- Brain abscess
- Subdural or epidural hemorrhage

Coma in which client appears awake but is unresponsive may be caused by:

- Abulic state: frontal lobe function depressed, so client may take several minutes to answer a question
- Locked-in syndrome: destruction of pontine motor tracts; is able to look upward
- Psychogenic state: unresponsive

DIAGNOSTIC TESTS

- Determine blood glucose level

MANAGEMENT**Nonpharmacologic Interventions**

- Nothing by mouth
- Insert nasogastric tube unless there is suspicion of associated basilar skull fracture or facial trauma
- Insert Foley catheter

Pharmacologic Interventions

If you suspect meningitis, do not withhold antibiotics. Antibiotics should be started before the client goes to the hospital. Discuss with physician. If unable to contact physician within a reasonable time frame, initiate the following:

For adults, antibiotics:^{22,23}

ceftriaxone (Rocephin) or cefotaxime 2 g IV stat
plus
vancomycin 1 g IV stat

Vancomycin should be infused by infusion pump no more rapidly than 1 g/hour to avoid a characteristic infusion reaction associated with rapid, uncontrolled administration.

Monitoring and Follow-Up

Monitor ABC, vital signs, pulse oximetry, level of consciousness, respiratory status and sensory motor deficits every 15 minutes until stable.

Appropriate Consultation

Consult a physician as soon as possible, once the client's condition has stabilized.

Referral

Medevac as soon as possible.

OVERDOSES, POISONINGS AND TOXIDROMES**DEFINITION**

Ingestion of a substance in sufficient quantity to induce symptom complexes associated with toxic effects. Poisoning is an exposure to an amount of substance that is likely to produce untoward effects in an individual.²⁴ If poisoning is suspected contact your poison control centre for management.

SPECIFIC POISONINGS AND CLINICAL TOXIDROMES

Opiates

- Examples: heroin, morphine, codeine, diphenoxylate (Lomotil)
- Toxidrome characterized by sedation, hypotension, bradycardia, respiratory depression, usually pinpoint pupils (may not be present with mixed overdose), somnolence progressing to stupor or coma, flaccidity of skeletal muscle, cold clammy skin, apnea, circulatory collapse, cardiac arrest and convulsions (may occur in children)²⁵

For treatment, see section “Assessment and Management General Approach” in this chapter, including the management specific to opiates.

Petroleum Distillates

- Examples: gasoline, fuel oil, model airplane glue
- Main toxic effect: pulmonary (from inhalation)

For treatment, see section “Management of Specific Overdoses and Toxidromes” in this chapter, including the management specific to petroleum distillates.

Tricyclic Antidepressants

- Main toxic effects: cardiac arrhythmias, anticholinergic effects (*see Toxidrome for opiate poisoning*), vomiting, hypotension, confusion and seizures
- Cardiac complications: prolonged QRS and QT intervals, other arrhythmias, sinus tachycardia, widening QRS complex, prolonged PR intervals, right bundle branch and AV block, ventricular tachyarrhythmias (including torsades de pointes and fibrillation)²⁶
- Neurologic complications: agitation, seizures
- Patients may occasionally become hypothermic²⁶
- Hypotension: Treat initially with IV fluids (*see section “Shock” in this chapter*)

The client may appear fine and then rapidly deteriorate. He or she will need to be admitted to a monitored unit. Be prepared to manage the client’s airway. Even if the client is asymptomatic 6 hours after ingestion, he or she must be admitted to hospital for psychiatric examination. Minimize external stimulation to reduce the risk of seizures.²⁶

For treatment, see section “Management of Specific Overdoses and Toxidromes” in this chapter, including the management specific to tricyclic antidepressants.

Salicylates (for example, Aspirin [ASA])

Main toxic effects: tinnitus, nausea, vomiting, hyperventilation (primary respiratory alkalosis) metabolic acidosis, hallucinations, stupor, cerebral edema, oliguria, renal failure, hemorrhage, cardiovascular failure,²⁷ fever, hypokalemia, hypoglycemia, seizures and coma.

Many patients are misdiagnosed on initial presentation as having sepsis or gastroenteritis (because of fever, acidosis, vomiting and other symptoms). This misdiagnosis is particularly common in the elderly.

For treatment, see section “Management of Specific Overdoses and Toxidromes” in this chapter, including the management specific to salicylates.

Acetaminophen (Tylenol)²⁸

Main toxic effects are hepatic.

- Symptoms may progress in a sequential 3-phase pattern and include from anorexia, nausea and general malaise to confusion, stupor, hepatic necrosis, jaundice, coagulation defects, hypoglycemia and encephalopathy²⁸
- The first begins shortly after ingestion and lasts for 12–24 hours. Client may have nausea and vomiting, anorexia, diaphoresis, pallor and general malaise
- If toxicity continues there is a latent phase of up to 48 hours (during this second phase the patient may feel better; however, hepatotoxicity is ongoing as evidenced by increasing hepatic enzymes (ALT and AST), bilirubin, PTT, INR)
- Symptoms of third phase depend on the severity of hepatic damage and usually occur from 3–5 days after overdose

For treatment, see section “Management of Specific Overdoses and Toxidromes” in this chapter, including the management specific to acetaminophen.

Caustic Agents

- Examples: alkaline (drain cleaner), bleach and battery acid (household bleach is usually not a problem, except for superficial burns), ethylene glycol (major constituent in antifreeze)²⁴
- Main toxic effects: local tissue necrosis of the esophagus with alkali and of the stomach with acids, as well as respiratory distress; obvious facial or oral burns and emesis; hoarseness and stridor reflecting epiglottic edema (especially with acids), headache, hypotension, metabolic acidosis, shock, renal failure, hypocalcemia and central nervous system damage²⁴

For treatment, see section “Management of Specific Overdoses and Toxidromes” in this chapter, including the management specific to caustic agents.

Carbon Monoxide

Main toxic effects:²⁹

- Central nervous system: headache (the most common presenting symptom), dizziness, fatigue, confusion. Syncope, coma, and seizures may occur in severe cases
- Gastrointestinal: nausea
- Other: arrhythmias and cardiac ischemia are possible
- Diagnosis: clinical background (for example, exposure to furnace or car exhaust [especially in children who have been riding in the back of pick-up trucks with enclosed “caps”]); level of carboxyhemoglobin needed to confirm

Arterial oxygen saturation as measured by pulse oximetry is frequently normal in cases of carbon monoxide poisoning.

For treatment, see section “Management of Specific Overdoses and Toxidromes” in this chapter, including the management specific to carbon monoxide.

Cocaine

Main toxic effects: seizures, hypertension, tachycardia, paranoid behaviour or other alterations in mentation, rhabdomyolysis, myocardial infarction and stroke (CVA), hepatic necrosis, liver and renal failure.²⁴

For treatment, see section “Management of Specific Overdoses and Toxidromes” in this chapter, including the management specific to cocaine.

ASSESSMENT AND MANAGEMENT: GENERAL APPROACH

First priority is ABC.

- Insert IV and administer 500 mL of 0.9% saline solution if patient hypotensive. Up to 1000 mL can be infused if hypotension persists²⁴
- If advised to do so by poison control, induce vomiting by pharyngeal stimulation²⁴
- Remember to decontaminate the gastrointestinal tract, any clothing, skin and environment (*see “Gastrointestinal Decontamination” in this section*)
- If client is unconscious, *see section “Coma (Not Yet Diagnosed)”*, in this chapter
- Determine to the best of your ability what was ingested
- For any client with overdose, draw blood sample for determination of serum acetaminophen level (*see section “Management of Specific Overdoses and Toxidromes” in this chapter, including management specific to acetaminophen*)
- Contact the nearest poison control centre for further information about the toxin in question

Appropriate Consultation

Consult a physician as soon as you are able after the initial assessment and stabilization of ABC. Discuss management with regional poison control centre.

Gastrointestinal Decontamination

Activated Charcoal

- Treatment of choice in most overdoses involving ingestion. It is most effective if administered within one hour of ingestion
- May be indicated for overdose with acetaminophen, theophylline, tricyclic antidepressants, phenobarbital, phenytoin, digoxin
- Does not work for metals such as iron or lithium
- Administer 10–25 g for children, 50–100 g for adults (1 g/kg) suspended in 300 mL of water
- If client will drink the mixture, this mode of administration is acceptable; otherwise, administer by nasogastric tube
- 30% of clients will vomit after administration of charcoal; in this case, charcoal can be administered again
- May cause constipation and charcoal impaction²⁴

Gastric Lavage

Discuss with physician first. A poison control centre should also be consulted.

- Lavage alone is not adequate for gastric emptying and delays administration of charcoal
- Not usually indicated especially if benzodiazepines, phenytoin or antibiotics have been ingested because minimum lethal doses of these agents is so high²⁴
- Not effective beyond 1.5 hours after ingestion, but can be tried in severely ill clients in selected situations
- Use largest nasogastric tube or orogastric tube
- Instil 300 mL aliquots of saline, then remove until saline is clear on removal or until 5 L of fluid has been used for irrigation
- Airway protection is recommended (client should be fully conscious and cooperative)
- Aspiration is a common, serious complication of up to 10% of patients

MANAGEMENT OF SPECIFIC OVERDOSES AND TOXIDROMES**Opioids**

Naloxone is a specific antidote for opiate poisoning. Toxic dose varies with the specific drug and an individual's prior history of opioid use.

Use naloxone with caution in those who are narcotic addicts, as it may precipitate acute opiate withdrawal. If this is a concern, the client's airway must be supported until the narcotic wears off.

Always observe the client until there is no chance of further respiratory depression. This is especially important with naloxone, which has a relatively short elimination half-life (1.1 hours) and an even shorter clinical effect (10–30 minutes).²⁴ This means that patients must be monitored closely should naloxone successfully reverse the effects of an opiate overdose.

Naloxone (Narcan), IV (usually start with 0.4–2 mg in adults); dose may be repeated if needed, at 2 to 3 min intervals.

If no response after 10 mg IV, re-evaluate diagnosis of narcotic overdose.

Client may have recurrent narcotization when naloxone wears off.

Naloxone produces acute withdrawal from opiates and may precipitate shock, seizures, arrhythmias, hypertensive crisis, pulmonary edema and intractable ventricular fibrillation.²⁴

Petroleum Distillates

- **Do not** perform lavage or induce vomiting if swallowed
- If no symptoms within 6 hours, no need for further observation

Tricyclic Antidepressants

- There is no specific antidote for an overdose of tricyclic antidepressants
- Avoid emesis (client may aspirate)
- Supportive therapy; charcoal may be used on the advice of a poison control centre (*see "Gastrointestinal Decontamination" in this section*)
- Client may appear fine and then rapidly deteriorate
- Client should be admitted to a monitored unit
- Be prepared to manage client's airway
- If client is asymptomatic 6 hours after ingestion, he or she should still be admitted to hospital for psychiatric evaluation and care
- Cardiac complications: prolonged QRS, QT interval, other arrhythmias
- Neurologic complications: agitation, seizures
- Seizures usually brief and self-limited; treat as outlined in "Status Epileptic (Acute Grand Mal Seizure)" (*see section "Status Epileptic (Acute Grand Mal Seizure)" in the adult Chapter 8, "Central Nervous System"*)
- If hypotension occurs, treat initially with IV fluids (*see section "Shock" in this chapter*)

Salicylates (for example, Aspirin [ASA])

- There is no specific antidote for an overdose of salicylates
- Toxic dose: > 300 mg/kg is associated with mild symptoms; ingestions of 300–500 mg/kg moderately toxic and ingestions of > 500 mg/kg are potentially lethal³⁰
- IV administration of normal saline to maintain blood pressure (*see section "Shock" in this chapter*)
- IV glucose²⁴
- Sodium bicarbonate and hyperventilation to correct metabolic and respiratory acidosis (moderate toxicity)²⁴
- Sodium bicarbonate also results in urine alkalization which promotes excretion of salicylates

Acetaminophen (Tylenol)

N-acetylcysteine (Mucomyst) is the specific antidote for acetaminophen overdose.

Toxic dose:³¹

- Single ingestions of < 150 mg/kg in a child or < 7.5–10 g in an adult are unlikely to be toxic
- Single ingestions of > 250 mg/kg or > 12 g in an adult over a 24-hour period are likely to be toxic
- Single ingestions of > 350 mg/kg are likely to result in serious hepatotoxicity
- Unintentional overdose (for example, from use of several acetaminophen-containing products), liver disease and alcohol use/abuse increase the risk of hepatotoxicity³²
- An acetaminophen level 4 hours post ingestion is a good predictor of toxicity

If ingestion is in toxic range (or if ingested quantity is unknown or cannot be verified), treat with:

N-acetylcysteine (Mucomyst) 20%, 140 mg/kg PO and then 70 mg/kg every 4 hours for 17 doses (total duration of treatment = 72 hours)

- Repeat any doses vomited within 1 hour of administration
- *N*-acetylcysteine may also be administered by IV (especially in patients who cannot tolerate oral administration)³²
- Do not withhold *N*-acetylcysteine even if 24–26 hours after ingestion; late administration, though not as effective as early administration, still reduces mortality
- Charcoal use is acceptable in acetaminophen overdose and only minimally interferes with *N*-acetylcysteine; charcoal should be given early and *N*-acetylcysteine at least 4 hours later

Caustic Materials

- Do not induce emesis or perform lavage
- Charcoal is not indicated
- If the client has visible oral burns, he or she has a 50% chance of distal burns of significance (that is, esophageal or gastrointestinal; however, absence of visible lesions does not rule out significant injury (10% to 30% will have burns beyond the mucosa)

Carbon Monoxide

- Administration of 100% oxygen (to displace carbon monoxide from hemoglobin)
- Even if client seems well when seen or is recovering from the central nervous system (CNS) insult, hyperbaric chambers have been shown to reduce long-term sequelae; therefore transfer client to hospital

Cocaine

There is no specific antidote for cocaine intoxication.

- Cocaine has a relatively short half-life, so most symptoms are short-lived unless a serious complication such as a CVA or MI occurs
- For coronary vasospasm, hypertension or tachycardia, observation is probably adequate, because of the short half-life
- For other cases, treat as for myocardial infarction
- Myocardial infarction and CVA may occur up to 72 hours after cocaine use
- Concurrent use of alcohol increases the likelihood of coronary vasospasm
- Do not administer beta-blockers to treat cocaine-related cardiovascular complications because the combination can result in unopposed alpha-adrenergic-induced vasoconstriction and end-organ ischemia³³

Not all chest pain represents myocardial infarction (for example, pneumomediastinum in crack use, bronchospasm).

- Seizures are generally self-limited but will respond to normal seizure treatment (*see section “Status Epileptic (Acute Grand Mal Seizure)” in the adult Chapter 8, “Central Nervous System”*)
- CNS symptoms such as agitation and paranoia can be treated with diazepam (Valium) or lorazepam (Ativan)

Monitoring and Follow-Up

Monitor ABC, level of consciousness, vital signs, oxygen saturation, intake and urine output frequently until the client is stable.

Referral

Medevac as soon as possible.

HYPOTHERMIA

Core temperature of $\leq 35^{\circ}\text{C}$ (95°F). Core temperature below 32°C predisposes patients to ventricular fibrillation, which could be preceded by ECG changes such as QT-interval prolongation, T-wave inversion and atrial fibrillation.²³

RISK FACTORS³⁴

- Age (pediatric patients related to inability to shiver and decreased body fat; elderly patients related to high incidence of cardiovascular disease and decreased body fat)
- Endocrine or metabolic derangements (for example, hypoglycemia)
- Infection (for example, meningitis, sepsis)
- Intoxication
- Traumatic injury and shock (head injury, major burns)
- Environmental exposure
- Iatrogenic (cold IV fluids, exposure during treatment)

Medications (such as phenothiazine, neuromuscular blocking agents, which interferes with the patient's ability to shiver;³⁶ clonidine and antipsychotic agents) may increase the risk of accidental hypothermia.³⁵

HISTORY

The evaluation and treatment of hypothermia is essentially the same whether the client is wet or dry, on land or in water.

- One or more of above risk factors
- The hypothermic client should be assessed carefully for coexisting injury or illness
- Signs and symptoms of hypothermia may be mimicked by alcohol, diabetes mellitus, altitude sickness, overdose and other conditions; therefore, thorough assessment is imperative
- Associated significant illness or injury may exacerbate hypothermia

The hypothermic client may appear “beyond help” because of skin colour, pupil dilatation and depression of vital signs. However, people with severe hypothermia have been resuscitated. Therefore, be cautious about assuming that the client cannot be resuscitated. It is also wise to be cautious about what you say during the resuscitation. Seemingly unconscious patients frequently remember what is said and done.

PHYSICAL FINDINGS

In the cold client, rectal temperature is one of the vital signs.

In terms of the ABCs, think A, B, C and D for hypothermic clients:

- A for airway
- B for breathing
- C for circulation
- D for degrees (body-core temperature)

In the cold client, body-core temperature is an important sign. Although obtaining the body-core temperature is useful for assessing and treating hypothermia, there is tremendous variability in individual physiologic responses at specific temperatures.

Assessment of Temperature

Axillary and oral measurements are poor measures of core temperature. Rectal temperature more closely approximates the core temperature and is a practical method for use in the field.

For clients with cold skin, rectal temperature should be determined with a low-reading thermometer (that is, capable of measuring temperatures as low as 21°C).

Core Temperature 35°C to 36°C

- Client feels cold, is shivering

Core Temperature 32°C to 35°C

- Slowing of mental faculties
- Slurred speech
- Mild incoordination
- Muscle stiffness
- Inappropriate judgment
- Irritability
- Shivering apparent

Core Temperature 32°C

- Shivering stops

Core Temperature $\leq 31^{\circ}\text{C}$

- Semi-comatose
- Progressive decrease in level of consciousness
- Coma likely at temperatures $\leq 30^{\circ}\text{C}$
- Cyanosis
- Tissue edema

Core Temperature 29°C

- Respiratory activity slow, may be difficult to detect
- Heart rate slow; pulse may be difficult to palpate

Core Temperature ≤ 28°C

- Vital signs absent
- Pupils dilated and unresponsive
- Respiratory arrest
- Ventricular fibrillation

MANAGEMENT**Goals of Treatment**

- Rewarm core slowly
- Minimize heat loss³⁵
- Prevent or manage complications

General Principles

The client with severe hypothermia must be handled very gently. The cold heart is highly prone to cardiac arrest, and even cautious movement of the client may induce cardiac arrest. Resuscitation and rewarming are the mainstay of treatment. Rewarming takes precedence once initial resuscitation has been initiated. The three progressive modalities of rewarming are passive external rewarming, active external rewarming and active core rewarming.³⁵

- Carefully remove all wet garments
- Ensure that any items, oxygen or fluids (both oral and IV) coming into contact with the client are warmed beforehand
- Oxygen should be heated to 40.5°C to 42.2°C (105°F to 108°F) and humidified, if possible
- Because cold skin is easily injured, avoid direct application of hot objects or excessive pressure (for example, uninsulated hot water bottles)
- The inside of a vehicle and any rooms where hypothermic clients are treated should be warm enough to prevent further heat loss (ideally above 26.7°C [80°F])
- Splinting should be performed, when indicated and with caution, to prevent additional injuries to frostbitten tissues
- Do not give caffeine or alcohol

Cardiopulmonary resuscitation (CPR) has no significant effect on survival of hypothermic clients in the following situations and should *not* be initiated:

- Cold-water submersion for > 1 hour
- Core temperature < 15.5°C (60°F)
- Obvious fatal injuries

- Client frozen (for example, formation of ice in airway)
- Chest wall so stiff that compression is impossible
- Rescuers are exhausted or in danger

Rise in core temperature may lag behind change in skin temperature and may continue to drop, so monitor rectal temperature frequently.

Basic Treatment for All Cases of Hypothermia

Prevent further heat loss: insulate from the ground, protect from the wind, eliminate evaporative heat loss by removing wet clothing or by covering client with a vapor barrier (such as a plastic garbage bag), cover the head and neck, and move the client to a warm environment; consider covering client's mouth and nose with light fabric to reduce heat loss through respiration. Maintain supine position and avoid unnecessary manipulation.³⁵

Mild Hypothermia

Rewarm passively and gradually:

Step 1: Place client in as warm an environment as possible.

Step 2: Increase heat production through exercise (without sweating) and fluid replacement with high-calorie, warm, sweet fluid; this method of adding heat is particularly important when emergency care is not readily available, as in remote or prolonged-transport environment.

Step 3: Rewarm passively through application of insulated heat packs to high heat transfer-loss areas such as the head, neck, underarms, sides of the chest wall and groin; apply heavy insulation to the same areas to prevent further heat loss (goal is to increase temperature by 1°C to 2°C per hour).

Step 4: Consider warm shower or bath if the client is alert.

Do not leave client alone.

Severe Hypothermia with Signs of Life (for example, Pulse and Respiration)

Treat the client as outlined in steps 2 and 3 above, with the following exceptions:

- Do not put a severely hypothermic client in a shower or bath
- Do not give a client fluids by mouth unless he or she is capable of swallowing and protecting the airway
- Treat hypothermic clients very gently (do not rub or manipulate or apply direct heat to extremities)

In addition, the following measures should be taken:

- Reassess ABC and vital signs frequently
- Give warm, humidified oxygen at 10–12 L/min or more by non-rebreather mask
- Administer warmed (to 37–40°C) normal saline by IV
- Clients with moderate-to-severe hypothermia may have a large amount of fluid sequestration and may need aggressive fluid resuscitation; an initial bolus of 1–2 litres is indicated; repeat as necessary, but do not overload with IV fluids

Severe Hypothermia with No Signs of Life

- If no pulse (after checking for up to 45 seconds), no respiration and no contraindications, start CPR unless contraindicated
- Ventilate with Ambubag with 50% warm, humidified oxygen; aim for 12–15 ventilations and 80–100 compressions; continue as long as you can
- Administer warmed (to 37–40°C) normal saline by IV

- Clients with moderate-to-severe hypothermia may have large amount of fluid sequestration and may need aggressive fluid resuscitation; an initial bolus of 1–2 litres is indicated; repeat as necessary
- Rewarm passively as outlined above

No drugs are used in resuscitation unless core temperature > 30°C and drugs are ordered by a physician.

Consultation

If resuscitation has been provided in conjunction with rewarming techniques for more than 60 minutes without the return of spontaneous pulse or respiration, continue efforts but contact the physician for recommendations.

Referral

Medevac as soon as possible.

MAJOR TRAUMA SITUATIONS

HEAD TRAUMA

Head injury is often associated with motor vehicle accidents, falls, violence and sports injuries. Severe head injury can lead to secondary brain damage from cerebral ischemia resulting from hypotension, hypercapnea and raised intracranial pressure.³⁵ It can also be defined as blunt, forceful injury to the soft tissues or bony structures of the scalp, skull or brain.

The initial response of the bruised brain is swelling. Bruising causes vasodilation through increased blood flow to the injured area; because there is no extra space within the skull, an accumulation of blood takes up space and exerts pressure on the surrounding brain tissue. This pressure results in decreased blood flow to uninjured areas of the brain. Cerebral edema does not occur immediately but develops over 24–48 hours. Early efforts to decrease the initial vasodilation in the injured area can save the person's life.

TYPES OF HEAD INJURIES

- Scalp wounds (lacerations)
- Skull injury (fracture)

- Brain injuries:
 - *Concussion*: no significant injury to brain, brief period of unconsciousness then return to normal; short-term retrograde amnesia, dizziness, headache, nausea, ringing in ears
 - *Cerebral contusion*: prolonged unconsciousness or serious alteration in level of consciousness; may have focal neurologic signs. Usually related to acceleration-deceleration injuries resulting in hemorrhage into the superficial parenchyma, often the frontal and temporal lobes³⁶
 - *Intracranial hemorrhage*: bleeding into brain tissue
 - *Acute epidural hematoma*: bleeding between the dura and the skull (collection of blood between the inner table of the skull and the outermost layer of the dura). Most often associated with skull fractures, meningeal artery lacerations or skull fractures and venous bleeding³⁸
 - *Acute subdural hemorrhage*: bleeding between the dura and arachnoid associated with underlying brain injury. Most often related to a rupture in the bridging veins between the cerebral cortex and the dura. Also caused by acceleration-deceleration and rotational forces³⁸

HISTORY AND PHYSICAL FINDINGS**Low-Risk Injuries**

- Criteria: Minor trauma, scalp wounds, no signs of intracranial injury, no loss of consciousness. Glasgow Coma Scale (GCS) score of 13 to 15 with a loss of consciousness that lasts up to 15 minutes³⁸
- Treatment: Observation for any sign or symptom of brain injury; must discharge to a reliable observer who will continue observation at home

Moderate-Risk Injuries

- Criteria: Symptoms consistent with intracranial injury, including vomiting, transient loss of consciousness, severe headache, post-traumatic seizures, amnesia, evidence of basilar skull fracture (cerebrospinal fluid [CSF] rhinorrhea, Battle's sign, raccoon eyes, hemotympanum, non-focal neurologic signs), GCS score of 9 to 12 with a loss of consciousness for up to 6 hours³⁸
- Patients are at a high risk for deterioration from increased cerebral edema and intracranial pressure and as such serial clinical assessments are necessary³⁸

High-Risk Injuries

- Criteria: Depressed level of consciousness, focal neurologic signs and penetrating injury of skull or palpable, depressed skull fractures. GCS score of 8 or less after resuscitation or those who deteriorate to that level within 48 hours

Other Aspects

The initial neurologic assessment is critical as a baseline.

- Head injury is frequently associated with other severe trauma
- Hypotension in adults is never caused by an isolated head injury, except if the client is near death; look for other injuries, including spinal cord injuries
- Physical examination should include a complete neurologic exam, as well as inspection for evidence of basilar skull fracture (for example, CSF rhinorrhea, Battle's sign, raccoon eyes, hemotympanum)
- Assume injury to the cervical spine in all cases of head trauma
- Remember that multiple trauma may be present

In cases of head injury, the clinical picture will evolve. The client is either improving or deteriorating over time; frequent reassessment is therefore critical.

Glasgow Coma Scale

The Glasgow Coma Scale is used to assess the severity of coma (see Table 2, "Scoring for the Glasgow Coma Scale").

- Assess client frequently
- Monitor for a drop in the score
- Any drop in the score is a danger sign

Table 2 – Scoring for the Glasgow Coma Scale*

Eye-Opening		Best Motor Response		Best Verbal Response	
Response	Score	Response	Score	Response	Score
		Obeys commands	6		
		Localizing response to pain	5	Oriented	5
Spontaneous	4	Withdrawal response to pain	4	Confused	4
To voice	3	Abnormal flexion (decorticate rigidity)	3	Inappropriate words	3
To pain	2	Abnormal extension (decerebrate rigidity)	2	Incomprehensible sounds	2
None	1	None	1	None	1

*Score is obtained by determining the score for each of the three criteria (eye-opening, best motor response, best verbal response) and summing them. Record scores individually, for example E2M4V3 for a total score of 9.

Sources: Young B. (2009). *Stupor and coma in adults*. Available by subscription: www.uptodate.com
Jarvis C. *Physical examination and health assessment*. 3rd ed. Philadelphia, PA: W.B. Saunders; 2000. p. 732.

Interpretation of Score

- Score < 9: severe head injury
- Score 9–12: moderate head injury
- Score 13–15: minor head injury

The Glasgow Coma Scale is not useful for the diagnosis of coma and may be unreliable in children. However, it has good interobserver reliability and is easy to use. The GCS on admission to a tertiary care centre has been linked to prognosis prediction for a number of conditions including traumatic brain injury, subarachnoid hemorrhage and bacterial meningitis. Intubation and use of sedating drugs interfere with its utility; for this reason, it is useful to obtain a GCS prior to these interventions.²¹

Coma by definition has no eye-opening, no ability to follow commands and no word verbalization.

COMPLICATIONS

- Seizures
- Vomiting
- Shock

DIAGNOSTIC TESTS

None.

MANAGEMENT**Minor Head Trauma**

- Characteristics: No signs of intracranial injury, no loss of consciousness
- Treatment: Observe for 12–24 hours for any sign or symptom of brain injury; discharge to a reliable observer who will continue observations at home

Major Head Trauma

In the pre-hospital setting, a major head trauma will require the critical tasks of an examination to recognize severe injuries with potential to cause rapid decompensation, stabilization for transport to a tertiary care centre and triage if multiple victims are involved.³⁷ The principles of assessment and management for trauma apply (*see “Primary Survey” and “Secondary Survey” under the section “Responding to General Emergencies and Major Trauma” in the pediatric Chapter 20, “General Emergencies and Major Trauma”*).

Remember, ABC (airway, breathing and circulation), in addition to D (disability [neurologic status]) and E (exposure), need to be addressed.³⁷ The order is important.

Step 1

- Secure the airway and provide supplemental oxygen at 10–12 L/min by non-rebreather mask
- Hyperventilate the client at 24 breaths/min with a bag-valve mask device⁴¹ to maintain adequate oxygenation and reduce intracranial pressure
- Once airway and breathing are secure, assess and manage any uncontrolled hemorrhage using direct pressure⁴¹
- Assess neurologic status⁴¹

Step 2

- Stabilize client on a spine board
- The neck should be immobilized in a rigid collar⁴¹ and a padded head immobilization device
- Nurse in head-up position unless contraindicated (for example, in cases of shock or back injury)
- Avoid tight cervical collar (any pressure on the external jugular veins will increase the intracranial pressure)

Step 3

- Record baseline observations
- Record blood pressure, respirations, PERRLA (pupils equal, round, reactive to light; accommodation normal), sensation and voluntary motor activity

Step 4

- Do serial Glasgow Coma Scale assessments

Step 5

- Monitor and record the above observations frequently

Step 6

- Start IV therapy to keep vein open, unless client is hypotensive
- Fluids are generally restricted in clients with closed-head trauma
- Maintain normal cardiac output
- If hypotensive, suspect hemorrhage or spinal injury (*see section “Shock” in this chapter*)

Step 7

- Insert Foley catheter if client is unconscious
- Monitor urine output hourly

Step 8

- Consult a physician as soon as able

Step 9

- Medevac as quickly as possible
- Review recommended precautions for flight for a person with head injury (*see the “Emergency Medical Transportation Guidelines for Nurses in Primary Care” in chapter 4 “Primary Care During Transport”, section “CNS”, “Head Trauma” [Medical Services Branch, 1985]*)

Increased Intracranial Pressure

- Elevate head of bed by 30°
- Hyperventilate, as above
- Prevent hyperthermia in the patient using antipyretics and cooling (to prevent an increase in cerebral metabolic rate related to increased body temperature)³⁸
- Maintain blood pressure control (must keep blood pressure high enough to ensure cerebral perfusion but not too high to increase ICP)³⁸
- Anticonvulsant medication may be given (on physician’s order) to reduce the risk of secondary ischemic insult associated with seizures³⁸
- Control environmental/noxious stimuli that may increase ICP (pain, environmental irritants, lighting)³⁸
- Osmotic diuretics such as mannitol may be given (on physician’s order) to reduce brain edema in cases of severe brain injury Mannitol, 1 g/kg IV over 20 minutes

CERVICAL SPINE AND SPINAL CORD TRAUMA

CERVICAL SPINE INJURY

Cervical spine injury occurs in up to 3% of trauma patients; this proportion increases to 10% among patients with significant head injury.

Initial care of the client who may have spinal injury is based on the suspicion of injury, stabilization of the spine and prevention of further neurologic injury. Close observation is required.

SPINAL CORD INJURY

Types of injury sustained depends on the mechanism of injury, which can include: hyperflexion, hyperextension, rotation, axial loading (vertical compression) and missile or penetrating injury.³⁸ Look for paralysis and other signs of cord injury, including priapism, urinary retention, fecal

incontinence, paralytic ileus, immediate loss of all sensation and reflex activity below the level of the injury.

CAUSES

- Motor vehicle accident
- Falls
- Sports
- Acts of violence

HISTORY**Traumatic Event and Mechanism of Injury³⁸**

In a traumatic spinal cord injury (SCI), the history, including the mechanism of injury, can provide clues to the pathophysiology of the injury. A detailed history eliciting elements suggestive of the force of impact, time of injury or presence or absence of pain at onset can be valuable. The following causes present the epidemiology of spinal injuries, from more to less common.

- Motor vehicle or bicycle accident
- Fall
- Sporting accident (diving and contact sport)
- Blunt trauma above the clavicles
- Stabbing or impalement near the spinal column
- Shooting or blast injury to the torso
- Sudden onset of symptoms of neck or back pain, numbness or tingling in the limbs, weakness or paralysis of the limbs

PHYSICAL FINDINGS

- Tachycardia
- Tachypnea
- Blood pressure may be low if in shock
- Pulse oximetry may be desaturating if in shock
- Tenderness on palpation or movement of the spinal column
- Obvious deformity of the back or spinal column
- Loss of sensation
- Weakness or flaccidity of muscle groups
- Loss of bladder or bowel control
- Priapism (sustained penile erection)
- Spinal neurogenic shock leads to vasomotor instability from loss of autonomic tone and may lead to hypotension or temperature instability
- Client may have hypoxia or hypoventilation if fracture or compression occurs above C5

Spinal Shock and Transient Paralysis³⁹

Immediately after a spinal cord injury, there may be a physiological loss of all spinal cord function below the level of the injury, with flaccid paralysis, anesthesia, absent bowel and bladder control and loss of reflex activity. In males, priapism may be observed. This finding is more frequent in cervical cord injuries. There may also be bradycardia and hypotension not due to other causes than the spinal cord injury. This altered physiologic state may last several hours to several weeks and is referred to as spinal shock.

A transient paralysis with complete recovery is most often described in younger patients with athletic injuries. These patients should undergo evaluation for underlying spinal disease before returning to play.

COMPLICATIONS

- Autonomic dysreflexia³⁸
- Neurogenic shock³⁸
- Permanent paralysis
- Respiratory arrest
- Spinal shock
- Death

DIAGNOSTIC TESTS

None.

MANAGEMENT

Goals of Treatment

- Stabilize spine
- Administer treatment in timely fashion
- Prevent further damage
- Prevent complications

Initial Treatment

- Assess and stabilize ABCD (airway, breathing, circulation, disability [neurologic status])
- Life-threatening injuries associated with spinal injuries must be addressed first, but the spine must not be put at risk during these maneuvers
- If there is penetrating neck trauma, do not remove foreign body

- Immobilize neck in neutral position and restrain chest to properly immobilize the cervical spine (sand bags are not a good tool for this purpose because if you later want to move the client onto a spine board, the bags may fall against the neck and cause further injury; instead, use soft rolled supports at the sides of the head, for example, rolled blankets)

The primary assessment of a patient with trauma in the field follows the ABCD prioritization scheme: airway, breathing, circulation, disability (neurologic status). If the patient has a head injury, is unconscious or confused, or complains of spinal pain, weakness and/or loss of sensation, then a traumatic spinal injury should be assumed.

Stabilization of Cervical Spine

- All multitrauma clients should be placed on a spine board with cervical spine immobilization. Extreme care should be taken to allow as little movement of the spine as possible to prevent more cord injury. Techniques to minimize spine movement include the use of log-roll movements and a backboard for transfer and placement of a rigid cervical collar.⁴⁰ A light cervical collar is ideal, allowing rapid access to the anterior neck if surgical access to the airway becomes necessary
- The collar is useless if it does not fit the patient, so any collar must be sized correctly
- To complete immobilization of the cervical spine, the client must be fixed as a “package” to the spine board; tape should be placed from board to forehead and back to the other side of the board
- It is important not to use the head alone as a fixation point, as this allows the cervical spine to act as a fulcrum for movement; restraints should therefore also be placed across the client’s shoulders
- Taping across the chin forces the mandible posteriorly and may obstruct the airway
- Consider the relationship of the axial skeleton to the spine board: in adults, the head is relatively smaller anteroposteriorly than the body, and the cervical spine may be in extension without some form of occipital padding
- Adults and older children may require 1–2 inches (2.5–5 cm) of padding under the head to approximate a neutral position

Prolonged immobilization (even < 30 minutes) on a spine board will cause occipital headache and lumbosacral pain in most people, regardless of underlying trauma, and unfortunately predispose the patient to pressure ulcers.

Adjuvant Therapy

- Give oxygen 10–12 L/min by mask; keep oxygen saturation > 97% to 98%
- Start IV therapy with normal saline to keep vein open, unless there is evidence of shock (see section “Shock” in this chapter)

Nonpharmacologic Interventions

- Nothing by mouth
- Insert nasogastric tube unless there is suspicion of associated basilar skull fracture or facial trauma
- Insert Foley catheter if there are no contraindications, such as pelvic fracture or blood in scrotum or at urethral meatus

Pharmacologic Interventions

As directed by the emergency medical service director or the on-call physician.

Monitoring and Follow-Up

Monitor ABCD, vital signs, oxygen saturation, level of consciousness, respiratory status and sensory motor deficits frequently.

Appropriate Consultation

Consult a physician as soon as possible, when client’s condition is stabilized.

Referral

Medevac as soon as possible.

FLAIL CHEST

Unstable segment of the bony chest wall.

CAUSES

Chest wall trauma with fracture of three or more adjacent ribs in at least two places. The result is a segment of the chest wall that is not in continuity with the thorax. Lateral flail chest or anterior flail chest (sternal separation) may occur. The flail segment moves with paradoxical motion relative to the rest of the chest wall.

The force necessary to produce this injury also bruises the underlying lung tissue, and this contusion will contribute to hypoxia. The client is at great risk for pneumothorax or hemothorax (or both) and may be in marked respiratory distress. Also consider the possibility of cardiac contusion and tamponade if there has been trauma to the anterior chest wall.

HISTORY

- Multiple trauma (motor vehicle or other accident)
- Severe chest wall pain
- Pain aggravated by movement and respiration
- Shortness of breath
- Impaired cough³⁸
- Hypoventilation³⁸

PHYSICAL FINDINGS

The physical findings depend on the severity of damage to the underlying lung tissue and the presence of associated injuries.

- Perform primary survey (see “Primary Survey” under the section “Responding to General Emergencies and Major Trauma” in the Pediatric Chapter 20, “General Emergencies and Major Trauma”)
- Carry out emergency interventions as necessary
- Perform secondary survey (see “Secondary Survey” under the section “Responding to General Emergencies and Major Trauma” in the Pediatric Chapter 20, “General Emergencies and Major Trauma”)

Vital Signs

- Heart rate elevated
- Respirations rapid, shallow
- Blood pressure decreased or normal
- Oxygen saturation, if available

Inspection

- Acute respiratory distress
- Sweating
- Cyanosis may be present
- Chest wall bruising
- Abnormal chest wall motion (paradoxical movement of chest wall) easily seen in unconscious client, less apparent in conscious client

Palpation

- Tenderness in injured area
- Crepitus may be felt
- Abnormal movement of chest wall may be palpable

Percussion

- Hyper-resonance and absent vocal and tactile fremitus (if pneumothorax present)
- Dull (if hemothorax, pulmonary contusion present)

Auscultation

- Air entry reduced in injured area
- Crackles may be present due to the presence of fluid

DIFFERENTIAL DIAGNOSIS

- Chest wall contusion
- Simple rib fractures

COMPLICATIONS

- Poor ventilation
- Hypoxia
- Hypovolemia
- Pneumothorax
- Hemothorax
- Pulmonary contusion
- Myocardial contusion
- Cardiac tamponade

MANAGEMENT**Goals of Treatment**

- Ensure patency of airway
- Improve oxygenation
- Replace blood loss
- Identify and treat associated injuries
- Provide pain control³⁸

Appropriate Consultation

Consult a physician as soon as client's condition is safely stabilized.

Nonpharmacologic Interventions

Priority is ABC.

- Control airway
- Ensure adequate ventilation
- Protect cervical spine
- Control pain by gently splinting chest with a pillow
- Do not splint aggressively

In the traumatized client with an injury above the clavicle, assume fracture of the cervical spine.

Adjuvant Therapy

- Give oxygen 10–12 L/min by mask
- Start two large-bore IV lines (16-gauge or larger) with normal saline
- Replace blood losses
- Adjust IV rate according to heart rate, blood pressure and clinical response

See section “Shock” in this chapter for further details.

Monitoring and Follow-Up

- Monitor mental status, vital signs, pulse oximetry and heart and lung sounds frequently
- Confusion, agitation may be signs of hypoxia

Referral

Medevac as soon as possible.

PELVIC FRACTURE

Disruption of the bony structure of the pelvis. Often the result of blunt trauma, pelvic fractures may range from benign to life threatening and include pelvic ring and acetabular fractures, and avulsion injuries.⁴¹

Classification of Pelvic Fractures³⁸

- Lateral Compression (LC): the most common. Produces shortening of the pelvis diameter and typically does not involve ligamentous injury. Generally forgiving to pelvic ring vessels, however, localized bleeding may still occur, particularly to the posterior pelvis
- Anterior Compression (AC): occurs when force is applied in the anterior-posterior direction and the pelvic diameter widens. The injury can be completely ligamentous and manifests as an open sacroiliac joint or open pubic symphysis. Commonly associated with vascular injury

- Vertical Shear: a complete disruption of a hemipelvis associated with hemipelvic displacement. Typically occurs when people fall from great height and land on one extremity
- Open Fracture: involves open wound with direct communication between the site of the fracture and a laceration involving the vagina, rectum or perineum. Mortality is high because of the resulting external exsanguination

CAUSES

Pelvic fractures generally require substantial force, such as a motor vehicle collision or a fall from a significant height, but can also be seen in frail and elderly patients who sustained low energy mechanisms of impact. Risk factors include: low bone mass, smoking, hysterectomy, older age and a propensity to fall.⁴²

- Motor vehicle collisions and motorcycle accidents: 43% to 58% of cases
- Pedestrian struck by a motor vehicle: 20% to 22% of cases
- Falls: 5% to 30%⁴³

The pelvis consists of the ileum (or iliac wings), the ischium and the pubis, which form an anatomic ring with the sacrum. Disruption of this ring requires significant force. Because of the forces involved, pelvic fractures frequently involve injury to the organs contained within the bony pelvis. In addition, the pelvis is supplied with a rich venous plexus, as well as major arteries; therefore, fractures in this area may produce significant bleeding.

The rate of complications related to injury to the underlying organs and bleeding is significant. Patients older than 60 with a significant pelvic fracture predicts a higher likelihood of bleeding that may require angiography.⁴⁴

Because of the tremendous force necessary to cause most pelvic fractures, concomitant severe injuries are common and are associated with high morbidity and mortality rates. The overall mortality rate in adults ranges from 10–16%; open fractures are associated with a mortality rate of 45% but account for 2–4% of all pelvic fractures.⁴⁸ Pelvic hemorrhage is the direct cause of death in less than half of patients with pelvic fractures who die. Retroperitoneal hemorrhage and secondary infection are the main causes of death.

HISTORY⁴⁵

The mechanism of a significant blunt trauma should prompt consideration of a pelvic fracture.

- Ambulatory at the accident scene
- Location of pain
- Bowel and bladder incontinence
- Symptoms of shock
- Bleeding: rectal, vaginal, hematuria³⁸
- Peri-anal ecchymosis³⁸
- Lower limb paresis or hypoesthesia³⁸
- Last menstrual period

Be aware that the amount of force necessary to cause a pelvic fracture is likely to have caused other significant injuries. Investigate for associated intra-abdominal and intra-pelvic injuries.

PHYSICAL FINDINGS

- Tenderness over the pelvis that can be appreciated with pelvic springing, which involves applying alternating gentle compression and distraction over the iliac wings
- Palpable instability of the pelvis on bimanual compression or distraction of the iliac wings (it is important to be very gentle when pelvic tenderness is appreciated; do not rock or apply great force until skeletally unstable pelvic fractures have been excluded by x-ray, since an overly aggressive exam can unnecessarily increase hemorrhage)
- Instability on hip adduction (pain on any hip motion suggests the possibility of an acetabular fracture, in addition to a possible hip fracture)
- Signs of urethral injury in the male, such as scrotal hematoma or blood at the urethral meatus
- Vaginal bleeding in a female
- Hematuria
- Limb length discrepancy or obvious rotational deformity of the pelvis or lower extremity⁴⁶
- Rectal bleeding or Earle's sign which is the appreciation of a large hematoma (swelling) or a palpable coccyx or sacral fracture line along with tenderness on (careful) rectal exam.⁴⁸ Digital rectal exam has a very low sensitivity for pelvic fractures and often misses them⁴⁷
- Destot's sign, a superficial hematoma above the inguinal ligament, on the proximal thigh or over the scrotum/perineum

- Roux’s sign, in which the distance measured from the greater trochanter to the pubic spine is less on one side than the other (indicating an overriding fracture of the anterior pelvic ring – a lateral compression fracture)⁴⁸
- Neurovascular deficits of the lower extremities

DIFFERENTIAL DIAGNOSIS

- Hip dislocation or fracture
- Femur fracture (this is a high-risk situation due to the potential for significant hypovolemia)

COMPLICATIONS

- Continued bleeding from the fracture or injury to the pelvic vasculature
- Shock
- Genitourinary problems from bladder, urethral, prostate or vaginal injuries
- Infections from disruption of the bowel or urinary system
- Deep vein thrombosis
- Death

A woman in the later stages of pregnancy is at increased risk of complications from pelvic fracture, and there is great risk of placental abruption and uterine rupture.

DIAGNOSTIC TESTS

- Obtain sample for urinalysis (look for gross or microscopic hematuria)
- Pelvic ultrasound, CT scan or x-ray may be required. Upon consultation, a routine plain x-ray in patients with a Glasgow Coma Scale score of > 13, presenting with no pelvic, abdominal or back complaints and no tenderness in the lower abdomen, lower back, groin or bony pelvis, may not be required⁴⁸

MANAGEMENT

Goals of Treatment

- Stabilize fracture
- Prevent and treat complications
- Prevent or control life-threatening hemorrhage³⁸

Appropriate Consultation

Consult a physician as soon as possible when a pelvic fracture is suspected or diagnosed. Hemodynamically unstable clients (with unstable pelvic fractures) require emergent orthopedic consultation for consideration of external fixation.

Nonpharmacologic Interventions

- Priority is to assess and stabilize ABC
- Address acute, life-threatening conditions
- Avoid excessive movement of the pelvis

Do not insert a urinary catheter until you have confirmed that there is no urethral injury (by physical exam).

Adjuvant Therapy

- Obtain large-bore IV access and administer normal saline as needed (*see section “Shock” in this chapter*)
- Give oxygen at 10–12 L/min or more by mask; keep oxygen saturation > 97% to 98%

Pharmacologic Interventions

Treat pain with narcotic analgesics:

morphine 5–10 mg IM or SC

Monitoring and Follow-Up

- Closely monitor vital signs and pulse oximetry
- Monitor the client for signs of ongoing blood loss and signs of infection
- Monitor for development of neurovascular problems in the lower extremities

Referral

- Medevac
- Achieve hemodynamic stabilization and transfer on a spine board

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