SPORT MEDICINE MANUAL

5

EMERGENCY TREATMENT OF LIFE THREATENING CONDITIONS

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5

Effective care of the severely ill or injured athlete demands advance planning and preparation, education of personnel and team rehearsal. Knowledge of injury mechanism, heightened index of suspicion, aggressive resuscitation, vigilance and continual reassessment and early access to appropriate surgical care will allow for the best possible outcome.

This unit summarizes the sequence of priorities that should be followed when assessing and treating a severely injured athlete. The emphasis is on trauma resuscitation. Other threats to life and limb, including anaphylaxis, acute compartment syndrome and burns are also briefly reviewed. Heat stroke is discussed in Unit 9 - Environmental Factors.

A. Assessment of the Injured Athlete

Resuscitation is ideally and most appropriately accomplished by a coordinated team, which requires advance preparation, appointment of team leader, effective communication and orchestration of roles and tasks. The leader may at times need to participate in resuscitation procedures, but his/her primary responsibility is overall coordination of the assessment and treatment process.

Knowledge of injury mechanism, index of suspicion and a rapid but complete physical examination will help ensure occult injuries are not overlooked. In particular, the speed of a projectile or victim, impact with stationary objects, the use of protective equipment, and information provided by witnesses should be sought. Blunt injuries of the abdomen or chest may only be suggested by history. Likewise, deceleration injuries should heighten suspicion for tears of the thoracic aorta or capsular tears of solid intra-abdominal organs. Some medications, such as steroids or ß-blockers may alter the physiologic response to injury and obscure typical physical findings. Thus, while the initial approach to trauma is based on a linear protocol (“the recipe”), successful trauma care most often demands lateral thinking (“the thinking cook”). In brief, the approach consists of:

1. establishing scene safety
2. a rapid primary survey with resuscitation and treatment of immediate life and limb threatening problems
3. a detailed secondary survey
4. initiation of definitive care based on the secondary survey

1. Primary Survey

In the primary survey, threatening problems are identified and resuscitation initiated. Care of the airway, primary cardiopulmonary resuscitation, and other direct life or limb saving manoeuvres should begin as soon as the problem is identified. Resuscitation is a dynamic process and should be undertaken in conjunction with the primary survey. The time-honoured and widely followed ABCDE protocol includes:

A - Airway maintenance with cervical spine (C-spine) control
B - Breathing
C - Circulation and haemorrhage control
D - Disability/drugs – assessment of neurologic status
E - Exposure - completely undress the patient being conscious of the potential for hypothermia

The primary survey can be completed rapidly in the conscious athlete by asking their name, what happened, if they can take a deep breath without pain and where they are injured. Should they respond appropriately with a normal voice, and if C-spine control is established (if required), and if no gross haemorrhage is evident and peripheral pulses are present and adequate, then the ABCDEs are sufficiently intact to proceed with the secondary survey. Failure to respond, or a response suggesting major injury, demands a more detailed primary assessment.

Airway and Cervical Spine Protection

The airway is the first priority. A patent and protected conduit for gas exchange must be maintained. A quick assessment is made by looking for chest wall motion and listening and feeling for air movement at the mouth. Clearing foreign debris or teeth with a finger sweep and/ or lifting the mandible with a jaw thrust may be required to establish patency.

The jaw thrust manoeuvre is performed by grasping the angles of the lower jaw (one hand on each side) and displacing the mandible forward. Since the jaw thrust manoeuvre does not hyperextend the neck, it is the method of choice for a trauma victim with a potential C-spine injury. It is better to assume the possible co-existence of a C-spine injury in any trauma patient and take care to provide immobilization, than to inadvertently convert an injury without a neurologic deficit into one with permanent paralysis.

Figure 5.1 Jaw thrust manoeuvre to open the airway of an individual with a suspected cervical spine injury.

In the alert, conscious patient, who has no distracting injuries, intoxication or neurologic impairment, with absence of neck pain and tenderness, a full, painless cervical spine range of motion and a normal neurological examination, a C-spine injury can be confidently excluded by the clinical assessment. No radiographic investigations are required. However, in any other circumstance, adequate assessment of the cervical spine cannot be provided by physical examination alone. The patient's head, neck and spine should be immobilized with an appropriately fitting stiff collar, secure lateral support and a long spine board until adequate radiographic and clinical assessment can be made.

Inability to open or maintain airway patency requires immediate attempts with an oropharyngeal or nasopharyngeal airway. The oral airway should only be used if the gag reflex is absent. It is inserted into the mouth behind the tongue and can be facilitated by using a tongue blade. An alternative technique is to insert the oral airway in the upside down position with its concavity upwards. When the soft palate is encountered, the airway is rotated 180° and slipped over the tongue. Care must be taken not to push the tongue backwards and thereby block rather than clear the airway.

A nasal airway is an effective and easily placed pliable tube that can be used in a sonorous patient with an intact gag reflex. Lubricating the tip, preferably with lidocaine jelly and inserting it into the most patent nostril, parallel to the palate and with gentle rotation, eases its passage. This simple device allows air to bypass upper airway obstruction caused by the tongue and/or decreased pharyngeal muscle tone.

Should efforts to obtain a patent airway continue without success, direct laryngoscopy and tracheal intubation, with rapid sequence induction (RSI) and accompanying in-line C-spine immobilization, should be attempted by the most experienced person available. However, time should not be wasted with repeated trials of intubation. If successful airway capture is not immediately evident, then proceed to a surgical airway (see airway management below).

Conditions that demand airway management prior to the onset of frank compromise of the airway or gas exchange include severe head injury, status epilepticus, penetrating neck trauma, upper airway burns and anaphylaxis with upper airway edema. Early, definitive airway management with endotracheal intubation is required. Failure to act early with neck trauma, burns or edema may lead to complete upper airway obstruction. In addition, any patient that may deteriorate during transport requires intubation prior to departure.

Oxygen via mask should be administered to all severely ill or injured patients. Pulse oximetry and cardiac monitoring should be utilized to assess oxygenation and heart rhythm.

Breathing

Injuries to the chest may prevent adequate oxygenation and ventilation even in the presence of a patent airway. The patient's chest should be completely exposed to be sure that ventilation is adequate and symmetric. The trachea should be palpated for deviation and the presence of subcutaneous emphysema. Five life threatening conditions - tension pneumothorax, open pneumothorax, massive haemothorax, flail segment and cardiac tamponade - should be systematically identified or excluded (each of these is discussed in further detail in Unit 6 - Trauma). Tension pneumothorax is relieved by inserting a 14-gauge intravenous catheter-over-needle through the second intercostal space at the mid-clavicular line on the affected side. Needle decompression and/or covering a sucking wound will temporarily stabilize the patient until more definitive interventions are possible. Volume resuscitation is required for a large haemothorax, but early surgical intervention may be more appropriate if it is secondary to penetrating trauma (see below). Chest tube thoracostomy can be delayed if personnel are limited, but, if insertion will not delay transport to definitive care, it is preferably completed prior to patient transport.

Circulation and Haemorrhage Control

Adequacy of circulation is best obtained initially by assessing skin colour and capillary refill and palpating the peripheral pulses for presence, strength and rate. In a normovolemic patient, capillary refill should occur within two seconds.

Blood pressure measurement is unnecessarily time consuming and often inappropriately reassuring. As a rough guide, a palpable pulse is associated with the following minimum systolic pressures: radial - 80 mmHg, femoral - 70 mmHg and carotid - 50 mmHg. Obvious blood loss should be identified and controlled with direct pressure. Tourniquets, haemostats and pneumatic splints are generally to be avoided because of their potential for tissue injury, negative effects on cellular metabolism and frequent inability to control bleeding.

Two large-bore intravenous (IV) catheters (18 gauge or larger) should be inserted and, if possible, blood drawn for type and cross-match, haemoglobin and haematocrit, coagulation profile and basic chemistry [ie. electrolytes, blood urea nitrogen (BUN) and creatinine]. Blood glucose levels should be determined at the bedside. If unable to secure peripheral venous access, central venous access (femoral access preferred) or saphenous cutdown at the ankle should be performed. However, IV access must not delay transport to a surgical facility and is often better established en route.

A pulseless extremity should be assessed by Doppler: urgent action may be required. If circulation is compromised, then decompression of circumferential burns with escharotomy, and acute compartment syndrome by fasciotomy, should be achieved early. Vascular compromise secondary to joint dislocation should be relieved by longitudinal traction and attempted reduction. Neurovascular assessment should be documented before and after any manipulation.

Disability (brief neurologic examination)

A rapid neurologic assessment is performed toward the end of the primary survey in order to establish the patient’s level of consciousness and pupillary responses. One simple method utilizes the mnemonic AVPU:

- A - Alert
- V - responds to Vocal stimuli
- P - responds to Painful stimuli
- U - Unresponsive

However, the Glasgow Coma Scale (GCS) is preferred. The GCS (see next page) can be used to determine the urgency of head computerized tomography (CT) and need for intracranial pressure (ICP) monitoring. Recent guidelines from the Brain Trauma Foundation in conjunction with the American Association of Neurological Surgeons recommend ICP monitoring for head injured patients with a GCS of 3 to 8 and traumatic intracranial lesions and avoidance of prophylactic hyperventilation in the absence of a measured increase in ICP (Brain Trauma Foundation, 1997). Hence, urgent head CT should be pursued in any patients who are comatose following head trauma.

Table 5.1 Glasgow Coma Scale.

Eye Opening (E)	Verbal Response (V)	Motor Response (M)
4=spontaneous 3=to voice 2=to pain 1=none	5=normal conversation 4=disoriented conversation 3= incoherent words 2=no words, only sounds 1= none	6=normal 5=localizes to pain 4=withdrawal to pain 3=decoricate posture 2= decerebrate posture 1=none
Total = E + V + M		

Expose (completely undress patient)

All clothing should be removed to allow for a careful examination and assessment of the entire patient, being careful to maintain core temperature by providing warm blankets or warming lights.

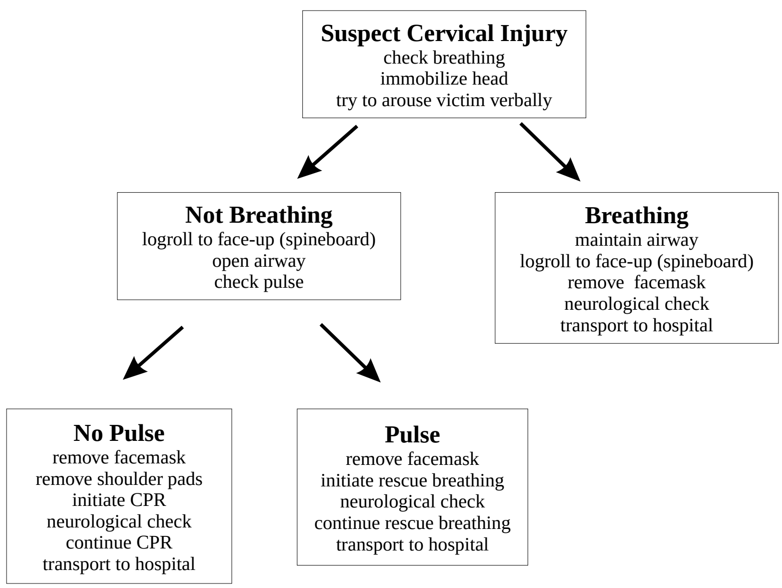

Figure 5.2 Field decision-making - head and neck injuries of an unconscious athlete. (From Vegro J, Bryant M, Torg J, Field Evaluation of Head and Neck Injuries, Athletic Injuries to the Head, Neck and Face, Joseph Torg (Ed). Lea and Febiger, Philadelphia. 1982; p43.)

At completion of the primary survey, a decision should be made regarding the level of care required. Timely transport should be initiated and unnecessary delays avoided. A philosophy of “load and go” is preferable if definitive surgical care is nearby. Prolonged transport, however, will dictate more extensive field resuscitation and stabilization in order to establish a secure airway, redundant large-bore vascular access and chest, gastric and bladder decompression. These procedures are generally easier to perform prior to departure as limited space in either ground or air transport vehicles makes them that much more challenging.

2. Resuscitation/Reassessment

Constant vigilance for change in patient status allows for early and potentially life saving intervention. The ABCDs should be continually reassessed, particularly following any intervention or change in status. Remaining alert, anticipating problems and being prepared for rapid surgical intervention will offer the trauma victim the greatest chance of recovery.

Fluid Resuscitation

Although shock related to trauma might be due to conditions other than blood loss, for example obstructive shock (cardiac tamponade or tension pneumothorax) or distributive shock (spinal cord injury), a shock-like state associated with trauma is most often due to haemorrhage. Standard treatment continues to be immediate fluid replacement in large volumes. However, controversy exists. Evidence from animal studies and a randomized prospective study of hypotensive patients with penetrating torso injuries suggests resuscitation to normotension is harmful and worsens ongoing haemorrhage. Delay of aggressive fluid replacement until the onset of operative intervention may be more appropriate. This advice, however, is predicated on rapid access to a surgical facility and cannot be extrapolated to blunt trauma or situations of prolonged transport distances. Hence, for now, it remains reasonable to establish IV access en route to a surgical facility.

Several types of fluid replacement exist, but a balanced salt solution (crystalloid), such as Ringer's lactate or normal saline, is the initial fluid of choice. In an adult, if haemodynamic instability (ongoing blood loss, delayed capillary refill, tachycardia and narrowed pulse pressure) persists following rapid infusion of 2 litres (l) of crystalloid (ie. 20-30 ml/kg in the average adult), then properly cross-matched blood should be given as soon as possible. Controlling haemorrhage is the goal in haemorrhagic shock and urgent surgical intervention is required when direct vascular control of ongoing haemorrhage is not achievable. If cross-matched blood is unavailable, then consideration should be given to the use of type O Rh-negative blood. Macro-pore intravenous filtering devices are used for whole blood transfusion in order to remove platelets and fibrin aggregates. Cold blood is associated with a high incidence of myocardial arrhythmia and paradoxical hypotension and blood should be warmed prior to transfusion.

Patients requiring massive transfusions (greater than blood volume) may need fresh whole blood, fresh frozen plasma (FFP) and/or platelets to restore clotting function. FFP and platelets should be given based on evidence of coagulopathy or thrombocytopenia. If, in the actively bleeding patient, prothrombin (PT) prolongation is greater than 1.5 times normal, or platelet count is <50,000, replacement is indicated.

The primary treatment of hypovolemia is fluid or blood replacement. However, vasopressors may be helpful in the early treatment of shock of those patients for whom prolonged systemic hypotension may be lethal (eg. patients with coronary or cerebrovascular disease). Restoration of mean arterial pressure to 60 mmHg may forestall the coronary and cerebral complications of diminished blood flow. Even so, vasopressors are most effective when vascular volume is restored, they should not be used in place of ongoing fluid resuscitation and generally used only following volume replacement. Vasopressors will also artificially elevate central venous pressure and pulmonary artery occlusion pressure and care should be used when interpreting these variables.

Fluid management in burns generally follows the Parkland formula and is titrated to maintain adequate urine output. In adults, 4ml of Ringer's lactate x body weight (kg) x % body surface area burns is given over the first 24 hours, with half given in the first 8 hours and half given in the following 16 hours. Urine output should be maintained greater than 50ml/hr. In children, the formula is modified to 3ml of Ringer's lactate x body weight (kg) x % BSA burns, again with half given in the first 8 hours and half given in the subsequent 16 hours. For children, urine output should be between 0.5 to 1.0 ml/kg/hr.

Pneumatic Anti-Shock Garments

When available, pneumatic anti-shock garments (PASG), in conjunction with the above resuscitative measures may be useful in the setting of unstable pelvic fractures or ruptured abdominal aortic aneurysms. If used, they are inflated from the periphery towards the core until an adequate blood pressure response has been obtained. Likewise, the deflation process should be gradual while vital signs are monitored. These garments act to increase afterload and do not autotransfuse pooled peripheral blood. There is no evidence they improve survival in patients with haemorrhagic shock following trauma. Furthermore, they are contraindicated if pulmonary edema is present and should not be used if thoracic or diaphragmatic injuries, evisceration of abdominal contents, penetrating objects or pregnancy are present or suspected. Pressure changes can occur in the anti-shock garment and include rapid expansion in a warm room after being in a cold environment or rapid expansion in an unpressurized cabin of an aircraft at altitude.

3. Secondary Survey

The secondary survey involves examination of the entire body from the top of the patient's head to the feet. The exam may be divided into anatomic regions or physiologic systems, depending on the preference of the examiner, but must include a thorough assessment of the head and neck, including the eyes, ears, nose and mouth, the chest and abdomen, including the rectum and pelvis, the spine and extremities and a neurologic evaluation.

Head

The eyes should be re-evaluated for pupillary size, the fundi for haemorrhages, the lens for dislocation, the conjunctivae for haemorrhages and the globe for any penetrating injuries. A quick visual examination of both eyes can be performed by having the conscious patient read a Snelling chart or the words written on the side of an intravenous bag.

Maxillo-Facial Trauma

Maxillo-facial trauma, if not associated with airway obstruction, can be treated after the patient is stabilized and not suffering from other life threatening injuries. An unconscious patient with maxillo-facial trauma should be presumed to have a cervical spine fracture until proven otherwise. The absence of a neurologic deficit does not rule out injury to the cervical spine and such an injury should be assumed to be present until it is ruled out by adequate radiographic and clinical assessment. Patients with mid-facial fractures may have a fracture of the cribiform plate and gastric tube insertion should be performed orally rather than nasally.

Chest

Visual examination of the chest both anteriorly and posteriorly will identify sucking chest injuries. These should be covered and sealed on three sides; one side is left open to allow air to escape. Each rib and clavicle are individually palpated and gentle anterior/posterior or lateral pressure is applied across the chest to identify the presence of fractured ribs. Circumferential burns may restrict chest expansion and impair ventilation; if so, release by escharotomy is required. In the patient with suspicion for inhalation injury, arterial blood gases, carboxyhaemoglobin and an electrocardiogram should be obtained.

Auscultation and percussion of the lung fields may identify a haemothorax if breath sounds are reduced or dullness percussed in dependent regions. Conversely, reduced breath sounds and hyperresonance in the apices or anterior chest may identify a pneumothorax. Distant heart sounds, distended neck veins and hypotension (Beck's triad) are suspicious of cardiac tamponade. A pulsus paradoxus of greater than 10 mmHg would also suggest the diagnosis. An urgent echocardiogram, if available, will confirm the presence of pericardial fluid and help guide pericardiocentesis. If air or blood exists within the thoracic cavity, it requires decompression with a needle (if under tension) and should be drained by chest tube thoracostomy. This is essential if the patient is to be transported over any extended distance and particularly if transported by air.

Abdomen

Any abdominal injury is potentially dangerous, but the specific diagnosis is not as important as the fact that an abdominal injury exists and that surgical intervention may be required. In addition, the initial negative examination may belie the seriousness of the patient's condition and the need for continued re-evaluation must be emphasized.

Suspected intra-abdominal haemorrhage in a stable patient can be assessed by diagnostic peritoneal lavage or CT of the abdomen. If intra-abdominal haemorrhage is suspected as the focus for hypovolemic shock, then urgent surgical exploration is required. A focused abdominal ultrasound for trauma may be performed but should not delay surgical intervention. A more detailed discussion of abdominal trauma follows in Unit 6.

Rectal Examination

Rectal examination should be undertaken to identify blood within the bowel, a high-riding prostate, pelvic fractures and integrity of the rectum or lax sphincter tone.

Fractures

Obvious fractures of the extremities are detected visually or with palpation. Tenderness, crepitation or abnormal movements with rotation are helpful in identifying fractures in which the alignment has been maintained. Gentle pressure over the anterior-superior iliac spines and symphysis pubis with the heels of the hands may identify pelvic fractures.

The amount of haemorrhage following blunt trauma should not be underestimated. A closed femoral fracture can produce loss of up to 2 litres of blood, whereas a pelvic fracture can result in loss of 3-4 litres. Transfusion of 6-2 units of packed red blood cells in the presence of a pelvic fracture is not uncommon. Longitudinal traction of femoral fractures will help reduce blood loss and a pneumatic antishock garment may help temporize bleeding from a pelvic fracture by providing some stability. However, the application of the PASG is secondary to the requirement for external pelvic fixation and is only potentially useful during transport. Laparotomy and angiography with embolization may be needed if the patient is exsanguinating.

Angulated fractures are splinted as they lie unless there is neurovascular compromise. Fracture-dislocations around the elbow, knee and ankle can be associated with vascular occlusion or disruption. Popliteal artery injury occurs in approximately a third of knee dislocations and vascular integrity should be assessed by arteriography if suspicion exists. Any patient with an open fracture should be kept NPO, requires tetanus immunization, IV access for pain control, broad-spectrum antibiotic coverage and urgent orthopedic consultation for operative irrigation and debridement.

Compartment syndrome should be considered and sought for in any patient with an extremity injury marked by haematoma or edema. A conscious patient will complain of pain, but severely injured or unconscious patients may not be able to communicate their distress. Hence, a heightened index of suspicion is required and, if in doubt, compartment pressures should be measured.

Neurologic Evaluation

The neurologic examination includes evaluation of level of consciousness, pupillary reactions and extraocular movements, motor and sensory function in the extremities, cerebellar function, rectal tone, visualization of the tympanic membrane for blood and evaluation of nasal discharges. The Glasgow Coma Scale (see Table 5.1) allows sequential monitoring and standardized communication and should be reported as E+V+M = x/15 to ensure consistent evaluation. Any findings of paralysis or paresis suggest a major injury to the spinal cord or peripheral nervous system. Adequate immobilization, using a long spinal board and a stiff cervical collar with lateral support and secure straps to the head and torso, must be provided. Padding behind the knees, between the legs and under the arms and under the heels and head will make the potentially extended length of time immobilized less uncomfortable for the awake patient.

Acute epidural and subdural haematomas, depressed skull fractures or other intracranial injuries require evaluation by a neurosurgeon. Any deterioration of the patient following a head injury requires reassessment, head CT and possible surgical intervention. If a neurosurgeon is unavailable, then patients with a significant head injury should be intubated by a rapid sequence induction with in-line cervical spine immobilization and considered for immediate transfer (see below).

Radiography

The standard trauma series includes lateral cervical spine, chest and AP pelvis radiographs. Additional images are ordered based on findings in the secondary survey. Extremity injuries should be evaluated with perpendicular images that include the proximate and distal joints. Non-diagnostic plain films are common and additional imaging studies should be pursued if suspicion for bony or significant soft tissue injury remains.

Other procedures

Urinary catheters and nasogastric catheters should be used if urethral transection or cribiform plate fractures do not contraindicate their insertion. Orogastric tubes are preferred in any patient who is head injured, has facial trauma, or may require prolonged intubation.

4. Definitive Care and Ongoing Reassessment

The definitive care for each injury is a continuation of the resuscitative measures provided after the initial assessment. This will be detailed in each of the subsequent sections.

If local providers and resources are unable to adequately manage the patient’s injuries, then early transport to a surgical facility and ideally to the care of a surgeon accustomed to trauma, should be undertaken. Communication should be established between the sending and receiving physician and arrangements made for appropriate, timely transport. A secure patent and protected airway, spinal immobilization, secure duplicate large-bore vascular access, stomach, bladder and chest (if required) decompression, oximetry and cardiac monitoring and trained personnel should be provided.

Continued re-assessment and evaluation of response to treatment is important. A life threatening problem that was not apparent initially may become apparent at a later time. Furthermore, the initial event may have been precipitated by an underlying medical problem, such as cardiac dysrhythmia, myocardial infarction, seizure, intracranial haemorrhage, hypothermia, heat stroke, hypoglycemia, electrolyte abnormality, or drug or toxin effect. Asking not only what, where, when and how the injury occurred, but also *why* the injury occurred at this point in time may help decipher causes that were not initially recognized.

5. Documentation

A quick mnemonic used to assess the athlete's past history is AMPLE:

A - Allergies
M - Medications
P - Past illness
L - Last meal
E - Events surrounding the injury

It is easiest to obtain the history and document the information soon after the event. Additional personnel may be required to contact witnesses or relatives to obtain the necessary information.

Meticulous record keeping, including a chronological report with flow sheets allows easy documentation of events and their treatment as they progress. In life threatening emergencies, consent for treatment is implied; treatment should be given and formal consent for treatment obtained later.

B. Airway Management

The upper and lower airways, separated by the vocal cords, may compromise oxygenation and ventilation if obstructed.

1. Upper Airway Obstruction

Upper airway obstruction is most commonly caused by the tongue, a foreign body (including blood, vomitus, a mouthguard, or dentures), edema of the glottis or injury to the larynx or vocal cords.

In an unconscious patient whose muscular tone is reduced or lost, the tongue may fall against the posterior pharyngeal wall and obstruct the airway, particularly if the patient is in the supine position. Since the tongue is attached to the mandible, any manoeuvre that brings the mandible anteriorly will also bring the tongue anterior and open the airway The chin lift, in which the mandible and teeth are grasped between the thumb and index finger, allows the mandible to be lifted upward. The jaw thrust was described in Figure 5.1.

Figure 5.3 Chin lift and head tilt manoeuvre used for opening the airway.

More advanced methods for opening and maintaining the airway include oropharyngeal or nasopharyngeal airways and endotracheal intubation. The oral and nasal airway has been discussed above. Endotracheal intubation is definitive airway management. It allows for protection of a patent airway and gives access for positive pressure ventilation and pulmonary flushing. Successful, atraumatic endotracheal intubation requires training, experience and regular practice. It is incumbent on anyone undertaking to intubate a patient to have assessed for and be prepared for a difficult airway and to have a well thought out plan for the potential "can't intubate, can't ventilate" scenario such as the American Society of Anesthesiologist's difficult airway algorithm (Benumof, 1996; available at http://anes-lxin.ucsd.edu/Airway/Algorithms/ASA.html).

The ideal method of endotracheal intubation in an otherwise intact patient with a suspected or confirmed cervical spine injury is an awake, fiberoptic intubation, but this luxury is rarely available in a trauma resuscitation. Hyperextension of the neck can be avoided if the head is maintained in a neutral position with in-line immobilization applied by an assistant. Hence, the method of choice is the method the provider is most comfortable and competent with and can accomplish in a timely fashion. Nasotracheal intubation has some advantages where oral access is limited, but there is no data to support its preference in patients with head trauma. Orotracheal intubation with in-line immobilization and rapid sequence induction is more commonly performed. Alternate methods utilising a Bullard laryngoscope or lighted stylet may also be used depending on the skill and experience of the practitioner.

The intubation attempt is optimized by:

- utilizing oimetric, cardiac and blood pressure monitoring
- providing pre-oxygenation without positive pressure ventilation for five minutes if spontaneously breathing, or with four vital capacity breaths
- having immediate access to working suction
- confirming the laryngoscope has a working bulb and an appropriately sized blade
- pre-medicating with a narcotic (usually fentanyl) or ß-blocker and/or lidocaine
- allowing three minutes for pre-treatment medications to work
- patient positioning (sniffing position)
- performing a RSI with induction agent (eg. thiopental, propofol, midazolam or ketamine), cricoic pressure and a short acting neuromuscular blocking (NMB) agent (succinylcholine preferred)
- intubating under controlled conditions

If a difficult airway is anticipated, then NMB agents and excessive sedation should be avoided unless the glottis has been visualized on awake laryngoscopy using titrated light sedation and topical anesthesia. The potential for converting a subopitmal airway into no airway ("can't intubate, can't ventilate") must be considered whenever paralytic agents are utilized. However, deep sedation without a NMB agent should also be avoided: it offers suboptimal intubating conditions and has neither the success nor low complication rate of either a properly performed RSI or an awake intubation.

Options for the "can't intubate, can't ventilate" scenario include a laryngeal mask airway, an esophageal tracheal combitube, transtracheal jet ventilation, retrograde intubation and a surgical airway (see below).

An appropriately sized endotracheal tube (ETT) should be selected (approximately the size of the little finger: 8.5 in an adult male, 7.5 in an adult female) and another ETT one-half to one full size smaller. The ETT should not be forced, and care should be taken to avoid mainstem bronchial intubation. Correct placement is 2cm above the carina, which, from the corner of the mouth is 23cm in the average adult male and 21cm in the average adult female. Confirmation of tube placement should be obtained by visualizing the vocal cords, auscultating over the apices and stomach and monitoring end-tidal carbon dioxide ($ETCO_2$). Once correct placement is confirmed, the ETT should be secured. Constant vigilance and continual reassessment is essential as the ETT may become displaced, kinked or obstructed with secretions.

If $ETCO_2$ detection or monitoring is not available, then esophageal detection devices, such as syringe aspiration or a self-inflating suction bulb may aid in confirming tube placement. Both methods attach to the end of the ETT and rely on a good seal and the anatomic differences between the trachea and the esophagus. If the ETT is placed into the esophagus, air cannot be easily aspirated because the walls will collapse; if in the trachea, the cartilaginous rings prevent occlusion. However, these devices are not infallible. Any leak in the system may lead to an incorrect assumption that the ETT is in the trachea.

Attempts at endotracheal intubation without adequate training, experience and preparation in a patient that is obtunded, combative and potentially head injured are to be avoided. Traumatic attempts at endotracheal intubation without appropriate pharmacologic preparation increases intracranial pressure, stresses the cardiovascular system, can injure the upper airway, or induce laryngospasm or aspiration of stomach contents and unneccessarily places the patient at risk.

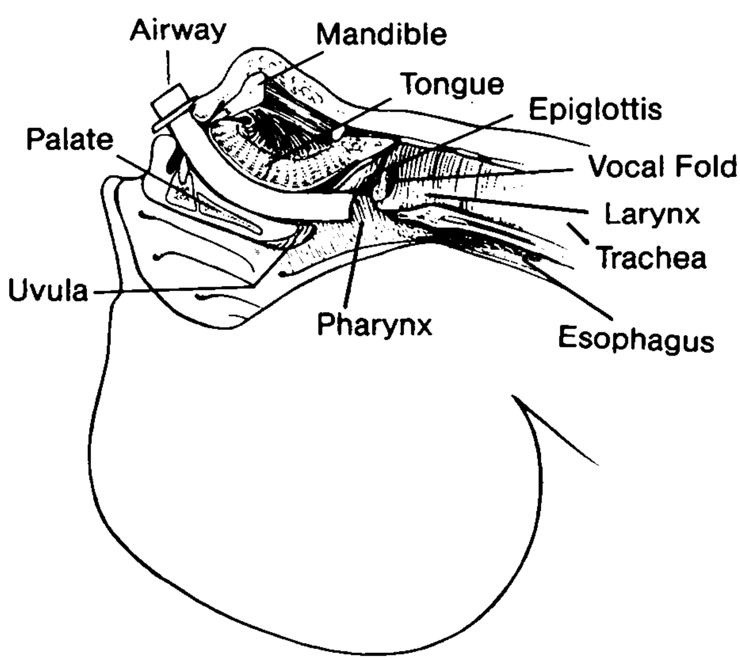

Figure 5.4 Insertion of an oropharyngeal airway.

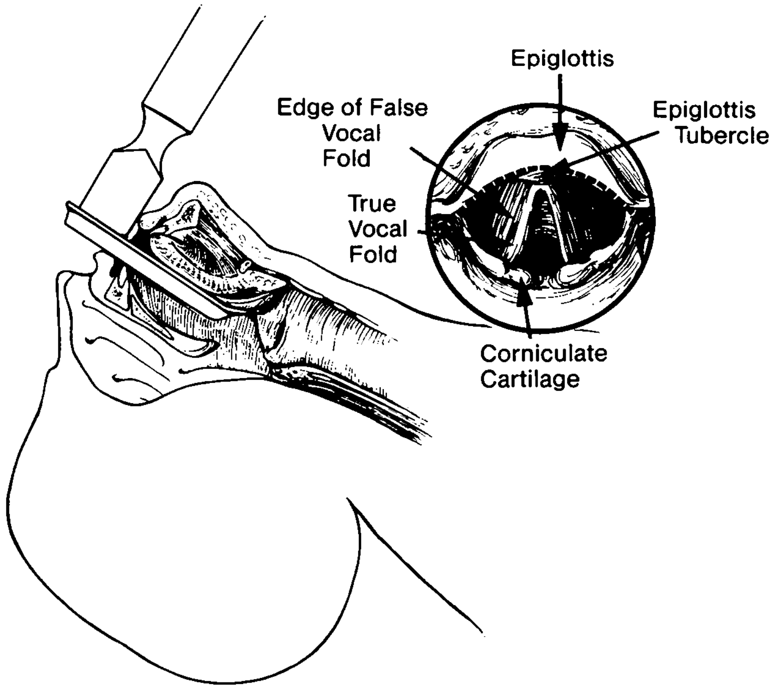

Figure 5.5 Intubation of a child.

Figure 5.6 Mask and bagging.

2. Lower Airway Obstruction

Inability to intubate the trachea is the primary indication for a surgical airway. This may be due to edema of the glottis, a fractured larynx, severe oropharyngeal haemorrhage or laryngeal spasm. A tracheostomy under emergency conditions is difficult to perform. It is often associated with profuse bleeding and the time required is usually excessive. Cricotyrotomy, or needle cricothyrotomy is preferred. In the latter, a #14 gauge catheter-over-needle is inserted into the trachea through the cricothyroid membrane. The cannula is then connected to a high-flow oxygen source via a Y-connector. Intermittent ventilation may be achieved using a rhythm of 1 second on and 4 seconds off by placing the thumb over the open end of the Y-connector. The advantage of this manoeuvre is that the high flow may dislodge an obstruction and move it into the oropharynx and it can provide adequate oxygenation for 30-45 minutes. The disadvantage of this procedure is that although some exhalation will occur, it is insufficient for complete ventilation: carbon dioxide will accumulate.

Surgical cricothyrotomy is easily performed by making a vertical or transverse skin incision with a #11 blade and extending the incision through the cricothyroid membrane. The handle of the scalpel is then inserted and turned vertically to hold the membrane open while a small tracheostomy tube is inserted. This technique is rapid, safe, relatively bloodless and relatively easy. Care must be taken, especially in children, to avoid damage to the cricoid cartilage as this provides the only circumferential support to the upper trachea in patients under the age of twelve. Needle cricothyrotomy is indicated in this age group.

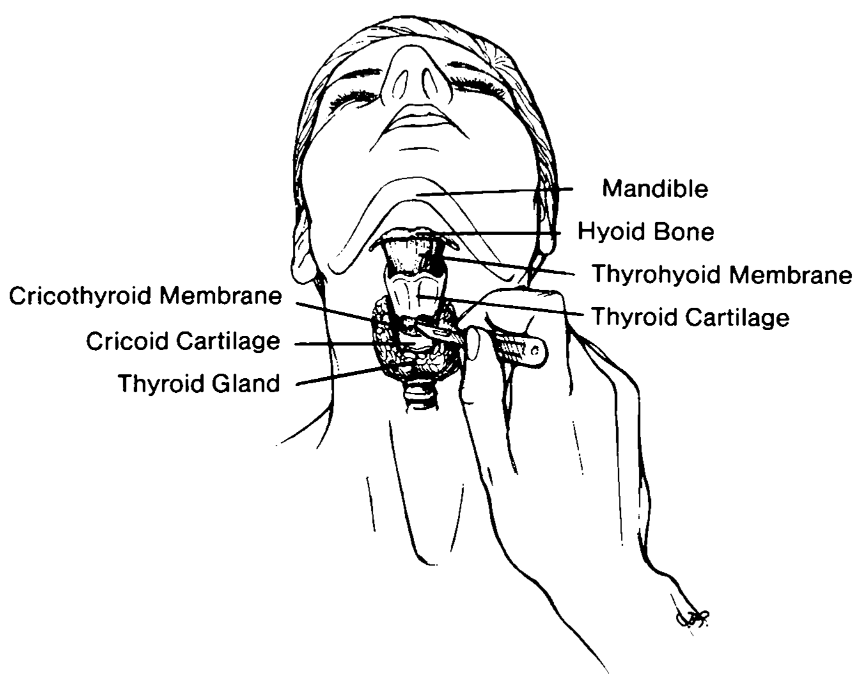

Figure 5.7 Cricothyrotomy: Cricothyroid membrane is opened with a scalpel, knife or other suitable sharp instrument. Twisting the instrument will enlarge the opening and patency is preserved by inserting rubber tubing or equivalent.

C. Shock

Shock may be caused by a wide variety of medical problems and traumatic injuries, but with the exception of cardiac tamponade and pneumothorax, the most common cause of shock in the first hour after an injury is haemorrhage.

Shock is defined as cellular hypoperfusion and manifests an inability to maintain the balance between cellular oxygen supply and oxygen demand. Shock progresses through phases of cell injury and death, organ failure and if uncorrected, ultimately death of the organism. Circulatory insufficiency is initially recognized by chemosensitive and pressure-sensitive receptors in the carotid artery. These, in turn, activate compensatory mechanisms, including release of catecholamines (norepinephrine and epinephrine) from sympathetic ganglionic nerve endings; tachycardia, narrowed pulse pressure and peripheral vascular constriction result with redistribution of regional blood flow away from cutaneous, splanchnic and muscular beds. Hence, early signs of shock are subtle and may only include delayed capillary refill, relative tachycardia and anxiety. Systolic hypotension is a late sign and suggests the compensatory mechanisms for increasing peripheral resistance and redistribution of circulation have exceeded their limits.

The “classic” signs of shock - pallor, clammy skin, confusion, tachycardia, hypotension and reduced urinary output - are more likely to present late during acute blood loss in a young, healthy endurance athlete. His/her enhanced vagal tone, increased blood volume, cardiac reserve and peripheral circulation allow for much greater compensation. A 50% increase in pulse rate and decrease in pulse pressure, as might occur with a 25% reduction in blood volume (class II haemorrhage - see below), is the difference between a blood pressure of 130/70 mmHg and a heart rate of 60 beats per minute (bpm) and a blood pressure of 115/90 mmHg and heart rate of 90 bpm. Such haemodynamic “stability” belies compensatory mechanisms under stress. When these fail, vascular collapse can be sudden and dramatic. Failure to recognize ongoing haemorrhage delays aggressive pursuit of the source of bleeding with investigations or surgical exploration. A high index of suspicion, based on suspected or known mechanism of injury, must be maintained.

1. Hypovolemic/haemorrhagic Shock

Total body fluid is 50% of lean body weight (LBW) in women (500ml/kg) and 60% LBW in men (600ml/kg). Blood volume, in turn, is 6 to 6.6% LBW (60 to 66 ml/kg) in women and men, respectively. In children blood volume is approximately 70 to 90 ml/kg of ideal body weight. For the average 60kg female or 80kg male, total body fluid is 30l and 48l, respectively and blood volume is 3.6l and 5.3l, respectively.

The American College of Surgeons proposes the following categorization of haemorrhagic shock:

1. Class I: 15% blood volume loss (750ml in a 70kg male).
   This is minimal, uncomplicated haemorrhage, with no changes in heart rate, blood pressure or respiratory rate. Treatment is by crystalloid replacement in a 3:1 ratio (ie. 3l of crystalloid per litre of blood loss).

2. Class II: 15-30% blood volume loss (up to 1500ml in a 70kg male).
   Tachycardia, tachypnea, anxiety, delayed capillary refill and decreased pulse pressure is usually present. Urinary output is only minimally affected. A 2 bolus of crystalloid replaces the equivalent of approximately 600ml of blood and is generally sufficient to restore haemodynamic stability. Blood replacement is not usually required and the remaining fluid deficit can be replaced with further crystalloid infusion.

3. Class III: 30-40% blood volume loss (up to 21 in a 70kg male).
   Classic signs of shock are present with changes in mental status, significant fall in systolic blood pressure and reduced urinary output. Blood transfusion/volume expansion is indicated.

4. Class IV: >40% blood volume loss (>21 in a 70kg male).
   Hypotension is profound, urinary output is absent and the patient is lethargic and near comatose. Immediate blood replacement and surgical intervention is necessary.

After blood loss, fluid is drawn from the interstitium into the vascular compartment in order to replace circulating volume. Hence, interstitial fluid deficits appear early. This forms part of the argument for utilizing crystalloid replacement. Because haemodilution from interstitial transcapillary refill takes 6 to 24 hours, haemoglobin or haematocrit is most often normal in the first few hours despite large blood volume losses. Neither is a reliable or appropriate measure of acute haemorrhage. Furthermore, haemoglobin concentration does not reflect tissue oxygenation and should not be used to determine need for blood transfusion.

2. Neurogenic Shock

Neurogenic shock, marked by bradycardia and hypotension, is a form of distributive impairment. Ventricular performance and fluid volume are preserved, but peripheral perfusion is inadequate secondary to loss of sympathetic input, unopposed vagal stimulation and poor vascular tone. The higher the spinal injury and in particular those above T1, the more likely and more severe the resulting shock state.

The diagnosis and treatment of neurogenic shock comes after the initial ABCDEs and accompanying resuscitation. It should be considered only after other causes of hypotension have been investigated and treated. Initial management remains rapid infusion of crystalloid. If hypotension (MAP<70mmHg) persists after other causes of shock have been excluded, then titrated vasopressor support with dopamine is appropriate. Patients with spinal cord injury who are seen within the initial 8hrs should also receive methylprednisolone 30 mg/kg over 15 minutes within the first hour, then 5.4 mg/kg/hr for the next 23hrs. This intervention has been demonstrated to improve neurologic outcome at 6 weeks and 1 year.

3. Anaphylactic Shock

Reduced peripheral perfusion in anaphylaxis results from widespread increase in vascular permeability and cardiovascular collapse secondary to an acute, abnormal systemic inflammatory response. Early, definitive airway management, fluid resuscitation and pharmacologic therapy comprise the initial approach. As previously noted, pre-emptive airway management can be life saving. Epinephrine is the primary drug treatment of anaphylactic reactions. If upper airway obstruction, respiratory distress or shock is present, oxygen is administered and 100 mg of epinephrine in 1:100,000 dilution (0.1 mg of 1:1,000 epinephrine in 10ml of normal saline) is infused at 1-2 ml/min in conjunction with a fluid bolus of 1-21. Failure to respond requires administration of colloid fluids and an epinephrine infusion (1 mg of 1:1,000 epinephrine in 500 ml NS) of 1-4 mg/min (0.5-2 ml/min) titrated to effect. Additional adjuncts include antihistamines (H1+H2 blockers), corticosteroids, inhaled ß-agonists and aminophylline. For patients on ß-blockers who remain hypotensive despite fluids and epinephrine, glucagon (1 mg IV every 5min until effect) should be used.

4. Patient Assessment and Monitoring of Shock

Vital Signs

Important vital signs to monitor include pulse oximetry, pulse rate, respiratory rate, pulse pressure, urinary output, level of consciousness, central venous pressure and blood pressure. Blood pressure may not be significantly affected until class III or IV haemorrhage so is less useful than pulse pressure. The mean arterial pressure is calculated by dividing the sum of diastolic pressure and pulse pressure by three, describes the relationship between cardiac output and vascular resistance. More valuable as a measure than systolic pressure alone, it reflects the ability of the systemic pressure to provide sufficient tissue perfusion. The shock index (heart rate/systolic pressure) is related to left ventricular stroke work. An index of 0.5-0.7 is normal, whereas values persistently >1.0 indicate impaired left ventricular function and carry a high mortality rate.

Urinary Output

Urinary output is not normally affected until class II haemorrhage occurs. Monitoring of urinary output helps determine the adequacy of peripheral blood volume. Adequate volume replacement should produce an expected urinary output greater than 50 ml/hr in the average adult and 0.5-1.0 ml/kg/hr in the child. Low urinary output with high specific gravity indicates the need for additional fluid while oliguria or anuria in the presence of adequate blood pressure suggest renal injury or obstruction.

Central Venous Pressure

Central venous pressure monitoring serves as a guide to right heart filling pressure. It is an elective procedure and should not delay primary fluid resuscitation. Furthermore, the supine CVP is often insensitive to the degree of volume depletion and may not change until volume loss exceeds 30% of blood volume. Measurement of cardiac filling pressures in the presence of hypovolemia may also be inaccurate because of impaired ventricular compliance secondary to sympathetic activation. In addition, cardiac tamponade may elevate CVP despite reduced blood volume. In short, cardiac-filling pressures can be insensitive to volume status and must be interpreted in light of the clinical picture.

Acid-base Balance

Respiratory alkalosis from tachypnea is the initial acid-base imbalance of hypovolemic shock. However, as tissue perfusion falls and compensation for the imbalance between tissue oxygen supply and demand fails, glycolytic metabolism becomes increasingly predominant, and leads to accumulation of lactate and hydrogen ions [H+] and produces a metabolic acidosis.

Base deficit (excess) - millimoles of base required to correct the [H+] of 1l of whole blood to 40 mmol/l or a pH of 7.40 - has been shown to correlate with volume depletion and mortality in victims of trauma. It is reflective of lactate concentration and can be classified as mild (2 to 5 mmol/l), moderate (6 to 14 mmol/l), or severe (>15 mmol/l). Thus a base deficit >5 mmol/l, or lactate concentration >4 mmol/l, suggest ongoing tissue ischemia and the need for more aggressive resuscitation.

Oxygen Extraction

Oxygen extraction in the peripheral circulation increases, at least initially, as oxygen delivery falls. Hence, measurement of the difference between pulse oximetry and oxygen saturation of blood obtained from the CVP catheter (a close approximation and more readily available measure of mixed venous oxygen saturation) allows for a relatively sensitive estimate of the adequacy of peripheral perfusion. Normal values are between 20-30%, whereas values exceeding 30% imply hypovolemia or increased metabolic demand, and values greater than 50% imply a shock state.

5. Complications of Shock Therapy

Haemorrhage may continue despite fluid or blood replacement and immediate surgical intervention should be considered. Failure to respond to resuscitation may be secondary to haemothorax and pneumothorax, unrecognized sources of fluid loss, acute gastric distension, cardiac tamponade, myocardial injury, diabetic ketoacidosis, hypoadrenalism and neurogenic shock. On the other hand, fluid overload may incite heart failure, pulmonary edema and contribute to the adult respiratory distress syndrome.

D. References

1. Anonymous. (1996). Guidelines for the management of severe head injury. Brain Trauma Foundation, American Association of Neurological Surgeons, Joint Section on Neurotrauma and Critical Care. J Neurotrauma, 3(11): 641-734.

2. Benumof, JL. (1996). Laryngeal mask airway and the ASA difficult airway algorithm. Anesthesiology, 84(3):686-699.

3. Bickell, WH, Wall, MJ Jr, Pepe, PE, Martin, RR, Ginger, VF, Allen, MK, & Mattox, KL. (1994). Immediate versus delayed fluid resuscitation for hypotensive patients with penetrating torso injuries. NEJM, 331(17):1105-9.

4. Bracken, MB, Shepard, MJ, Collins, WF, Holford, TR, Young, W, Baskin, DS, Eisenberg, HM, Flamm, E, Leo-Summers, L, Maroon, J, et al. (1990). A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal-cord injury. Results of the Second National Acute Spinal Cord Injury Study. NEJM, 322(20):1405-11.

5. Marino, PL. (1998). The ICU Book. 2nd Edition. Williams and Williams, Baltimore.

6. Rutherford, EJ, Morris, JA Jr, Reed, GW, & Hall, KS. (1992). Base deficit stratifies mortality and determines therapy. J Trauma, 33(3):417-23.

7. Tintinelli, JE, Kelen, GD, & Stapczynski, JS (Eds). (2000). American College of Emergency Physicians. Emergency Medicine. A Comprehensive Study Guide. 5th Edition. McGraw-Hill, New York.

8. Torg, JS (Ed). (1982). Athletic Injuries to the Head, Neck and Face, Lea and Febiger, Philadelphia.

9. Velmahos, GC, Theodoru, D, Tatevossian, R, Belzberg, H, Cornwell, EE 3rd, Berne,TV, Asenio, JA, & Demetriades, D. (1996). Radiographic cervical spine evaluation in the alert asymptomatic blunt-trauma victim: Much ado about nothing? J Trauma, 40(5):768-k.

Further information can also be found at the following web sites:

http://anes-lxin.ucsd.edu/Airway/Algorithms/ASA.html
http://www.emedicine.com/emerg
http://www.mdconsult.com
http://www.techrescue.org/firstaid/firstaid.html
http://www.trauma.org