

# ***ETLS***

## **COURSE MANUAL**

# **EUROPEAN TRAUMA COURSE MANUAL**

## **Contents:**

1. Initial assessment and resuscitation of the severely injured patient
2. The roles and responsibilities of the trauma team
3. Airway management
4. Thoracic trauma
5. Shock
6. Abdominal and pelvic trauma
7. Head trauma
8. Spinal trauma
9. Extremity and soft tissue trauma
10. Trauma in children
11. Trauma in women
12. Medical problems in trauma patients
13. Maxillofacial trauma
14. Injuries due to burns
15. Hypothermia and cold injury
16. Analgesia
17. Chemical, biological, radiological and nuclear (CBRN) incidents and trauma
18. Inter- and intra-hospital transfer of the trauma patient
19. Psychological effects of trauma
20. Epidemiology of trauma
21. Scoring systems for trauma patients

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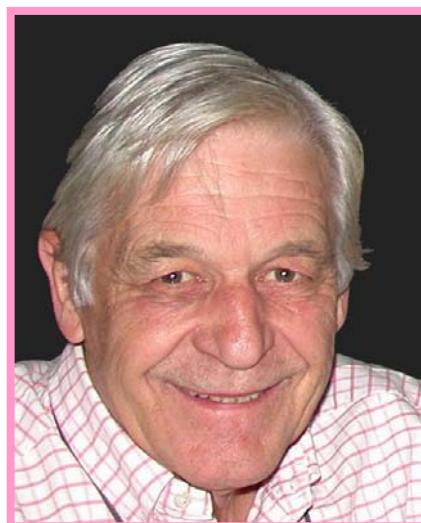
## **Trauma Care – The Team Approach**

The European Trauma Course (ETC) has been developed to teach a system of care for managing trauma patients that reflects the reality we experience on a daily basis. As a person who deals with trauma victims from the time that they arrive in hospital, you may already have some knowledge and skills, and experience of working in a team. However, these might differ from the other people that you are working with, and this can impair the efficiency of the team. This course aims to give you the opportunity to acquire new knowledge and skills to enhance the abilities you already have, but equally as important, learning how to become both an effective trauma team member and a good team leader. This approach of improving knowledge, skills **and** team work is reflected in the material on this CD and throughout the activities undertaken during the course.

The contents of the ETC are based upon the working practices of the relevant specialties that treat trauma patients within Europe and, where it exists, evidence of best practice. Under the direction of the ETC Working Group of the European Resuscitation Council (ERC), the contributors have taken a pragmatic approach to improving trauma care by concentrating on the principles that are common throughout Europe. Consequently, the course is not didactic in all areas of management, but retains an element of flexibility; where effective local variations in management exist, they can be acknowledged and recognised as an alternative approach.

The material on this CD is divided into two sections; the first part is the “must-know” of trauma care, containing the details about how to be a safe and effective team member managing trauma patients. The second part is the “should–know”, those details that will enhance your understanding of trauma and the problems that may be faced infrequently, but that still pose a challenge. Not surprisingly, the first part is essential information without which attendance on an ETC would be time wasted. The second part covers topics that can be reviewed later and will help you understand how to face almost any eventuality. Therefore before attending the ETC it is essential that you have read and assimilated the “must-know” parts of the CD; your fellow team members will be relying on it.

On the course you will learn by working through a series of trauma cases of varying complexity. In the majority of cases the patient's survival is dependent on the trauma team working effectively rather than the abilities of any one individual. As in real life, your role in the team will change depending on the needs of the patient. Our aims are that by the end of the course you will have improved your knowledge, skills, and abilities as a team member and understand how this enhances team performance and contributes ultimately to patient outcome. Furthermore you will also have identified your own weaknesses and learnt how these can be overcome.



### **Dedication**

The European Trauma Course would probably not exist had it not been for the vision and encouragement of Doctor Peter Baskett, the epitome of both a team member and team leader. His contribution as a master teacher of resuscitation in all its guises is the stuff of legends. Peter's drive and enthusiasm have taken resuscitation training to the highest levels in numerous countries in Europe, the Middle East and Africa. Huge numbers of healthcare professionals world-wide will tell you of the influence that Peter had on them and as a result what they were subsequently able to achieve. There is no doubt that, amongst all who have contributed to this project, we feel enormously privileged and fortunate to have such an inspirational figure to motivate and guide us. This dedication is but a small measure of not only the enormous respect in which Peter is held, but also of the deep gratitude of his friends and colleagues throughout Europe.



## Chapter 1

# INITIAL ASSESSMENT AND RESUSCITATION OF THE SEVERELY INJURED PATIENT

## Objectives

At the end of this chapter the reader should understand:

- How to prepare to receive a trauma patient
- How to perform a primary survey on a trauma patient
- The initial resuscitation of the trauma patient
- The initial investigations required in a trauma patient
- How to perform a secondary survey
- Have an overview of the whole of the management of the patient in the resuscitation room.

Many of the points discussed below will be reinforced and described in greater detail in the ensuing chapters. After studying Chapters 3, 4 and 5 this chapter should then be reread so that the clinical issues described can be considered.

## Introduction

The in-hospital management of trauma patients is similar throughout Europe but the organisation of pre-hospital care varies from country to country. In general there are two main systems; a physician based service (e.g. SAMU in France, Notarztsystem in the German speaking countries) or a paramedic/emergency technician based system (e.g. UK). Consequently patients will arrive at the Emergency Department having received different levels of care at scene and en route to the hospital. Whatever the pre-hospital system, the subsequent management of the severely injured patient is dependent on two key factors; a team approach, and the use of a system to guide the team through the initial assessment and performance of life saving procedures. These two elements enable team members to carry out their tasks simultaneously, reduce the time taken to resuscitate the patient and increase the chances of survival.

## Planning and preparation for receiving a trauma patient

Emergency departments usually have some warning either directly from the pre-hospital team or a central (ambulance) control that a severely injured patient is about to arrive. In some areas, the use of telemedicine systems allows direct contact between the pre-hospital personnel and the receiving hospital, allowing the advice to be obtained while at the same time providing hospital staff with important information (Box 1.1). This allows the team to

assemble any specialist equipment or services that may be required to manage the patient(s).

#### **Box 1.1: Essential pre-hospital information**

- Mechanism and timing of the incident
- Number, age and sex of the casualties
- The patient's complaints, priorities and injuries
- The patient's conscious level
- Airway, ventilatory and circulatory status
- The management plan and its effect
- Estimated time of arrival

Detail of the mechanism of the injury is essential as it gives valuable information about the forces the patient was subjected to, particularly the direction of impact. Further help comes from a description of the damage to vehicles, injuries to other individuals or the weapon used.

Any resuscitation room must always be fully stocked with all the equipment in working order and ready for use. Only minimum preparation should be necessary immediately prior to the arrival of the patient. The trauma team should assemble in the resuscitation room with each member taking universal precautions. If these are not available, surgical gloves, plastic aprons and glasses must be worn, as exposure to any blood or body fluids should be assumed to be an infection risk (e.g. HIV and hepatitis viruses). Whilst protective clothing is being put on, the team leader needs to brief the personnel, ensuring that each member knows the task for which they are responsible (see Chapter 2). If details of the patient's injuries are available, the team leader must ensure that the team have the ability to deal with them; if there are any concerns, an early call for specialist assistance must be made before arrival of the patient. A final check of the equipment, by the appropriate team members, can then be made.

#### **Reception and transfer**

On arrival the patient must be triaged using a recognised system and transferred to the resuscitation room. Once there, the recording nurse should note the time accurately, so that the precise timing of all subsequent events can be recorded.

Most trauma patients will arrive immobilised on a long spinal board with head blocks and straps. This device facilitates the transfer of the patient from the stretcher to the trolley, but

this manoeuvre must be coordinated to avoid displacement of a tracheal tube, removal of IV lines or rotation of the spinal column and exacerbation of pre-existing injuries (Fig. 1.1). A vacuum mattress, although providing spinal immobilisation, does not have sufficient rigidity to allow safe transfer and therefore additional support under the mattress is required during transfer. If the victim is not on any lifting device, six people are required for transfer. The team must be well practiced in this technique and coordinated by a team leader experienced in the safe transfer of patients with possible spinal trauma. One of the airway personnel should stabilize the head and neck, at the same time taking care of any tracheal tube, as four lift the patient from the side and a sixth removes the ambulance trolley.



**Fig. 1.1: Transfer using a long spine board**

While this is being performed the team leader's actions will be initially dictated by the extent of the care delivered by the pre-hospital team. The medical team leader always carries out a very quick overview of the patient to try and identify obvious life-threatening conditions. This is achieved by looking and listening to the patient's response as verbal contact is made, while at the same time feeling the patient's periphery. This is sometimes referred to as the "5 second" round. Its aim is observation, not intervention.

### **The “5-second round”**

- Is the patient conscious or unconscious?
- Is their airway clear, acceptable, obstructed needing immediate intervention?
- Is ventilation and oxygenation adequate or unacceptable?
- Are there signs of massive external haemorrhage or severe hypovolaemic shock
- Are there any major deformities of head, neck, trunk or limbs?
- Note their skin colour and temperature while feeling the pulse

Depending on the findings, immediate action may be to direct the team to:

- Securing the airway, support of ventilation

- Control of external haemorrhage, start CPR
- Request further help or support e.g. massive transfusion protocol
- No immediate life-threatening injuries, proceed with full primary survey

In a physician-based pre-hospital care system, the patient may arrive in the Emergency Department with all or some of the following; a definitive airway, mechanical ventilation, monitoring, vascular access established, fluids being given along with analgesic and sedative (anaesthetic) drugs. In these circumstances all interventions need to be checked by the appropriate team members to ensure they are functioning and have not become misplaced.

Following the 5-second round, the team leader's next action will be to take a handover to allow confirmation of:

- Mechanism of injury
- Injuries identified so far
- Signs and symptoms
- Treatment given
- Response to treatment

The subsequent management of the patient will take into account this information.

Irrespective of the level of care provided in the pre-hospital environment the team leader needs to start his involvement with the patient by ensuring that a full primary survey is completed. Although in some pre-hospital systems a primary survey may already have been done at scene, injuries may have been missed or developed on route to hospital. The primary survey is described sequentially below, but with a team, these tasks can be allocated and carried out simultaneously.

## **Primary survey and resuscitation**

Objectives of the primary survey and resuscitation:

- To identify and treat any immediately life-threatening conditions
- To follow an 'ABCDE' approach in the identification of immediately life-threatening conditions

The following is based upon the management of patients who may have received either paramedic or physician based pre-hospital care.

Generally, after major trauma, depending on their injuries, patients die most rapidly from **Airway** obstruction, followed by **Breathing** or ventilation problems and then from **Circulatory** failure or shock. Injuries or **Dysfunction** of the central nervous system tends to cause death

relatively more slowly and may be the cause or result of these problems (unconsciousness may cause airway obstruction, severe shock may cause loss of consciousness). Ultimately some injuries may not be revealed until the entire patient is Exposed and examined. For these reasons this course advocates an **ABCDE** approach for all trauma patients (Box 1.2) to ensure that injuries are dealt with in terms of their immediacy of threat to life. This approach prevents the unconscious patient with an obstructed airway suffering a hypoxic insult while carers concentrate on major limb injuries. Patients with major trauma often have injuries to more than one system, the advantage of a team approach to management is that these can be identified and dealt with simultaneously. Further details of the roles and responsibilities of the team are given in Chapter 2.

#### **Box 1.2: Primary survey & resuscitation**

- **Airway with cervical spine control**
- **Breathing and ventilation**
- **Circulation and control of haemorrhage**
- **Dysfunction of the CNS (neurological assessment)**
- **Exposure and environmental control**

#### **Airway and cervical spine control**

A patent airway is of paramount importance, but the airway personnel must initially assume the presence of a cervical spine injury, particularly if the patient is a victim of blunt trauma or if the mechanism of injury indicates this region may have been injured (see Chapter 8). Consequently, all activities used to clear and secure the airway should result in minimal movement of the head and neck.

As the airway nurse manually immobilizes the cervical spine, the airway doctor talks to the patient to establish supportive contact and assess the airway. If the patient replies with a normal voice, giving a logical answer, then the airway can be assumed to be patent and the brain adequately perfused with oxygenated blood. An impaired or absent reply may indicate an altered level of consciousness with the potential for airway obstruction. The commonest cause of obstruction is the tongue and an attempt must be made rapidly to relieve this by using either the chin lift or jaw thrust techniques (see Chapter 3). In the deeply unconscious patient, the airway can initially be maintained by the insertion of an oropharyngeal (Guedel) airway, or if the mouth cannot be opened, a nasopharyngeal airway. These patients must then receive 100% oxygen via a facemask. Relative contraindications to the use of a nasopharyngeal airway are facial injuries or possible fracture of the base of skull. In the latter

case there is a small risk that the catheter could enter the cranial vault through a fracture of the cribriform plate.

If these manoeuvres do not improve the airway, it must be checked for obstruction. Vomit or other liquid debris is best removed with a rigid sucker. Flexible suction catheters are more likely to kink and obstruct. A patient with a pharyngeal (gag) reflex is usually capable of maintaining his own airway and no attempt must be made to insert an oral airway as this may precipitate vomiting, cervical movement or a rise in intracranial pressure. A nasopharyngeal airway may be tried in this situation, as it is less likely to stimulate a gag reflex

Trauma victims rarely have empty stomachs. The combination of head, chest or abdominal injuries and alcohol ingestion significantly increases the risk of vomiting or regurgitation and aspiration of gastric contents. If the patient starts to vomit while still immobilised on a spinal board, priority must be given to clearing their airway and they should be turned on their side or the trolley tipped head down and the vomit sucked away as it appears in the mouth. If suction is not sufficient to clear the airway within seconds the whole patients has to be turned on his side rapidly. Every attempt should be made to avoid rotation of the spine, but protection of the cervical spine may be compromised. Therefore, if the gag reflex is absent, tracheal intubation is required to minimise the risk of aspiration. This needs to be carried out by medical members of the team with adequate anaesthetic training and experience, using the procedures described in Chapter 3. As anaesthetic drugs mask any neurological signs it is important that these patients first have a mini-neurological examination carried out by the team leader (See D – Dysfunction of the CNS, below).

Patients who arrive in the Emergency Department having been intubated pre-hospital are at risk of having the tracheal tube displaced during transfer. It is essential that the airway team immediately assess and confirm correct position of a tracheal tube by auscultation and attach a capnograph and observe the tracing, making adjustments as appropriate (see Chapter 3).

**The first priority in patients intubated pre-hospital is to verify that the tracheal tube is in the correct position.**

All severely injured patients must receive as close to 100% oxygen as soon as possible. In the patient breathing spontaneously a mask with a reservoir, attached to a high flow of oxygen (12-15 l/min), should be used. Even in an unconscious patient with partial airway obstruction, this will still improve oxygenation and also help to minimise the periods of hypoxia when procedures are carried out to clear the airway. Intubated patients will need to

be ventilated with an appropriate breathing system and inspired oxygen concentration. At the same time, a pulse oximeter should be attached to check the adequacy of oxygenation. Once the airway has been confirmed as secure, the neck is inspected quickly for:

- Swellings and wounds, which can indicate there is local injury or damaged blood vessels
- Subcutaneous emphysema from a pneumothorax.
- Tracheal deviation resulting from a tension pneumothorax (see below).
- Distended neck veins indicate there is a rise in the central venous pressure. This can result from a tension pneumothorax, cardiac tamponade, damage to the great vessels, pre-existing heart failure or the possibility of fluid overload pre-hospital (see Chapter 4).
- Laryngeal crepitus indicating laryngeal trauma.

The cervical spine needs to be secured using a semi-rigid collar and head blocks. Selection of an appropriately sized collar and correct application is essential to prevent compression of the jugular veins and subsequent compromise of the cerebral circulation especially in brain injured patients. This applies equally to those patients who arrive in the Emergency Department having had a semi-rigid collar applied pre-hospital. The only exception to this rule is the restless patient who will not keep still. In this case, immobilizing the head and neck whilst allowing the rest of the patient's body to keep moving, can damage the cervical spine. A sub-optimal level of immobilization is therefore accepted, consisting of only a semi-rigid collar.

### Breathing and ventilation

After clearing the airway, adequate ventilation must be confirmed. This includes an examination of the chest: There are five immediately life threatening thoracic conditions which must be searched for, and if found treated (Box 1.3).

#### **Box 1.3: Immediately life threatening thoracic conditions**

- Tension pneumothorax
- Open chest wound
- Massive haemothorax
- Flail chest
- Cardiac tamponade

The earliest clues as to whether any of these conditions exist in the patient breathing spontaneously are the respiratory **rate** and **effort**. These need to be monitored at frequent

intervals by the recording nurse because they are very sensitive indicators of underlying lung pathology. At the same time, the medical team leader should inspect both sides of the chest assessing the patient's respiratory rate, effort, colour and use of accessory muscles of respiration. Symmetry of chest movement, paradoxical movement and signs of injury are important in patients breathing spontaneously and those who are ventilated. Ventilation of the periphery of the lungs should be assessed in all patients by auscultation and percussion in the axillae, both above and below the nipple line. Listening over the anterior chest mainly detects air movement in the large airways, which can give a false impression of the adequacy of ventilation. Consequently, differences between the two sides of the chest can be missed, especially if the patient is being ventilated.

In those patients who arrive in the ED intubated and ventilated, tidal volume and respiratory rate being delivered by the ventilator, peak inflation pressure, end tidal CO<sub>2</sub> and oxygen saturation should then be checked by the airway personnel.

A difference in air entry and percussion note between the right and left sides of the patient's chest usually indicates a local thoracic problem. In an intubated patient this may be due to unintentional bronchial intubation as a result of inserting the tube too far, usually into the right main bronchus. Chest trauma may cause a pneumothorax, haemothorax or pulmonary contusion, either alone or in any combination. If a pneumothorax is suspected, it is vital to determine if it is under tension. This is more common in patients who are being ventilated when a build up of pressure in the pleural cavity can rapidly compromise the lungs and circulation with fatal consequences (see Chapter 4). In this group of patients, in addition to the findings on examination, the peak inflation pressure on the ventilator will be increased and the end-tidal CO<sub>2</sub> and oxygen saturation will be reduced. If the findings support the diagnosis of tension pneumothorax, a wide bore cannula connected to a syringe is inserted into the second intercostal space in the midclavicular line on the affected side. The aim is to release the positive pressure in the chest. If there is sudden release of air, the diagnosis is confirmed and a chest drain can subsequently be inserted on that side. Alternatively, if there is no rapid decompression of the pleural cavity, then an urgent chest x-ray must be taken before a chest drain is placed. An alternative if the diagnosis is in doubt is to use a needle attached to a syringe containing saline and look for bubbling as the pneumothorax is entered (see Chapter 4).

In the primary survey, only a massive haemothorax, (i.e. over 1.5 litres) is likely to be detected clinically. Initial treatment consists of a chest drain, however there is usually enough time to confirm the clinical suspicion with a chest x-ray or sonographically before this

procedure is carried out. Insertion of a chest drain may increase blood loss and therefore prior IV access is essential.

If there is no air entry to either lung in a ventilated patient, the commonest causes are either complete obstruction of the upper airway, or oesophageal intubation. If a facemask is being used, there is usually an incomplete seal between face and mask. The maintenance of an effective seal is not always easy and the airway personnel need to be skilled in this technique. Ideally a two-person technique should be used, with one holding the mask on and pulling the patient's chin forward with both hands, whilst the other person squeezes the bag. Care must always be taken to maintain cervical immobilisation. If this still fails then the airway doctor and nurse should prepare for urgent tracheal intubation. If the patient has arrived intubated, a rapid check must be made of; all connections, the patency of the tracheal tube, position of the tube, the breathing circuit and function of the ventilator. If no explanation can be found, consider bilateral tension pneumothoraces (see Chapter 4). Ultimately, an alternative method of ventilation must be tried without delay.

An Ashermann seal may have been applied to open wounds pre-hospital or may be used initially in the ED. A chest drain will need to be inserted to prevent the development of a tension pneumothorax.

The immediate treatment of a flail segment of chest wall depends on the extent of the underlying pulmonary contusion. The patient who is becoming progressively hypoxaemic and exhausted, despite high flow oxygen, requires intubation and mechanical ventilation (see Chapter 4).

Any penetrating injury that enters the area indicated in Fig. 4.2 may involve the heart. This can lead to a pericardial tamponade as blood collects in the pericardial sack. If suspected an urgent ultrasound is required, and if the diagnosis is confirmed, thoracotomy will be required. If emergency thoracotomy is not possible then, depending on the competency of the medical team leader, a pericardotomy or pericardial aspiration using the sub-xiphoid approach should be performed (see Chapter 4).

### **Circulation and control of haemorrhage**

Any overt bleeding must be controlled by direct pressure using absorbent sterile dressings. Clean towels are appropriate if large oozing areas have to be covered immediately. Tourniquets are only used in the ED when a limb is deemed unsalvageable.

One of the circulation nurses should measure the blood pressure and pulse using an automated blood pressure recorder and establish ECG monitoring, with appropriate recording of the results at regular intervals. At the same time, the medical team leader must specifically look for clinical evidence of shock - i.e. inadequate oxygen delivery to vital organs **or their ability to utilise it** (see Chapter 5). Skin colour, clamminess, capillary refill time, heart rate, blood pressure, pulse pressure, conscious level and respiratory rate must all be assessed. Intravenous access must be established using two wide bore peripheral cannulae. If this is not possible then cannulation of a central vein should be performed. The site of access will depend upon local policy and the injuries sustained by the patient; the femoral vein is useful as it avoids the risk of causing a pneumothorax but is of limited use in the presence of pelvic fractures. Conversely the subclavian vein tends to remain open even in severe haemorrhagic shock, but cannulation is associated with the risk of pneumothorax.

Once the first cannula is in position, blood is drawn for grouping, typing, or full cross matching, full blood count, analysis of urea and electrolytes, toxicology, pregnancy test in women of childbearing age and bedside blood glucose estimation. The cannula should not be jeopardised; if it is difficult to aspirate blood, the intravenous infusion should be started and the required blood sample can then be taken from a femoral vein or artery. Towards the end of the primary survey, an arterial sample blood sample needs to be obtained for blood gas, pH analysis and lactate measurement. If the team have the skills, consideration should be given to the insertion of an arterial cannula. This will allow regular blood gas estimations to guide ventilation and direct blood pressure measurement.

Cannulas inserted pre-hospital should be checked to ensure that they have not been dislodged before infusing large volumes of fluids. If inserted under dirty or unsterile conditions, consideration should be given to early replacement.

The initial fluid for intravenous infusion can be either crystalloid or colloid; the debate over the type used continues without definitive evidence either way. The key point is that the volume given should be guided by the patient's initial physiological signs and the nature and extent of their injuries to achieve an appropriate blood pressure. Subsequent fluids will depend on their response and blood given if the patient remains haemodynamically unstable (see Chapter 5). To reduce the incidence of hypothermia, all fluids should be warmed either during or before use.

Patients presenting in severe hypovolaemic shock require transfusion of O Rh-negative packed red blood cells in the emergency department (O Rh+ve may also be used in males) and the early use of platelets and clotting factors. There should also be a search for the

source of the bleeding. The common internal sites are the chest, abdomen, pelvis and multiple long bone fractures. Ultrasound is increasingly used to identify bleeding in the chest or abdomen. Torso injuries causing haemorrhage will usually require urgent surgery. In contrast pelvic fractures causing massive haemorrhage benefit from early stabilization in the resuscitation room. Similarly suspected musculoskeletal injuries should be splinted to minimise bleeding before they are x-rayed.

When massive transfusion is indicated, large bore cannulae for example haemofiltration lines can be inserted into central veins. These also allow the central venous pressure (CVP) to be monitored, are ideal. Again early consideration should be given to the insertion of an arterial line, to allow accurate and continual monitoring of the blood pressure and to obtain repeated blood samples. The blood bank should be informed as early as possible to avoid shortage of blood products and exsanguination of potentially salvageable patients. Where there is massive haemorrhage requiring surgical intervention, the aim should be to achieve a balance between perfusion of vital organs perfusion while minimising blood loss. This process should be considered a stop-gap before definitive surgery and therefore be kept as short as possible (see Chapter 5).

### **Dysfunction of the CNS (neurological assessment)**

The mini-neurological assessment is used to provide a rapid assessment of the conscious level and detect the presence of a potentially intra-cranial surgical lesion. It comprises of the Glasgow Coma Scale (GCS) score, limb lateralising signs and pupil size and reactivity. This assessment should be repeated so that any deterioration can be detected early. The presence of lateralising signs indicates the patient needs an urgent CT. This will need to be augmented with a more detailed neurological examination during the secondary survey. It is important to remember that there are a number of causes of a reduced level of consciousness apart from a head injury (Box 1.4).

#### **Box 1.4: Causes of an altered level of consciousness**

Traumatic brain injury	Alcohol
Insulin / diabetes	Epilepsy
Poisons / drugs	Infection
Psychiatric disorders	Opiates
Shock	Urea / metabolic

Assessment of a patient who is intubated and anaesthetised will be limited to detecting the pupillary response. In these situations it is vital that an account of the patient's neurological

status prior to anaesthesia is obtained, in particular their GCS and any discrepancy in the motor response between their right and left sides.

### **Exposure and environmental control**

While the patient's dignity must be respected, it is essential that all clothing be removed so that the entire patient can be examined. The presence of injuries, particularly to the spine, prohibits normal removal of clothes and patients' garments must be cut along seams using large bandage scissors, to facilitate their removal with minimal patient movement. If the patient is conscious an explanation must be given and permission sought! The resuscitation room should be kept warm and overhead or warm air heaters used. The rapid removal of tight clothing, for example motorcycle leathers, in patients who are severely hypovolaemia, can precipitate sudden hypotension due to the loss of a tamponading effect. Therefore these garments should be removed at the team leader's discretion and only after the establishment of adequate fluid resuscitation.

Exposed trauma patients lose body heat rapidly no matter what the season or country. This leads to a fall in the core temperature, particularly if they have a spinal injury. Studies have also shown that if hypothermia is allowed to develop, morbidity and mortality are increased.

In order to prevent further discomfort and the risk of developing pressure sores haemodynamically stable patients can be log rolled at this stage and the spinal board removed. Head restraints need to be maintained until the cervical spine has been cleared of potential injury.

The well-practised trauma team should aim to complete the objectives of the primary survey and resuscitation phase in less than 10 minutes (Box 1.5). Prearrangement with the laboratory for rapid processing and reporting will facilitate the team leader's evaluation of the patient's state.

The heart rate, blood pressure, respiratory rate and pattern, pulse oximetry, capnometry and ECG should be monitored and recorded at regular intervals to identify any change in the patient's condition. An important question to ask repeatedly is, "Is the patient getting better or worse"? This helps to determine if the team needs to move rapidly to definitive care. A typical example would be the need for rapid surgical intervention to gain control of a source of bleeding if there is no response to appropriate intravenous resuscitation

At this point provision of analgesia should be considered. Depending on local policy, opioids (morphine, fentanyl, sufentanil) or ketamine can be used, titrated intravenously (see Chapter 16).

<b>Box 1.5: Objectives of the primary survey and resuscitation phase</b>	
<b>Non-physician pre-hospital care</b>	<b>Physician based pre-hospital care</b>
Assess and secure the airway.	Check the function of any airway device
Stabilize the cervical spine.	Confirm adequacy of spine immobilisation
Assess and correct any breathing or ventilatory problems.	Assess ventilation, ventilatory parameters, correct any problems
Control external haemorrhage.	Control external haemorrhage.
Assess the patient's haemodynamic state.	Assess the patient's haemodynamic state.
Insert two, large bore peripheral cannulae.	Check function of any IV cannulae
Take and send blood samples to the laboratory.	If not already, send bloods to laboratory
Assess the patient's conscious level	Confirm patient's neurological status before sedatives given
Establish supportive contact.	Remove the patient's clothes, while keeping the patient warm.
Remove the patient's clothes, while keeping the patient warm.	Attach or replace vital sign monitors
Record initial vital signs connect monitors.	Insert a gastric tube if appropriate
Insert a gastric tube if appropriate.	Reassurance, comfort
Reassurance, comfort	Adequate analgesia
Adequate analgesia	

**Only when all the airway, ventilatory and hypovolaemic problems have been corrected should the team continue with the more detailed secondary survey.**

During the primary survey an appropriately trained individual (e.g. the relative's nurse) should greet any of the patient's relatives or friends who arrive, take them to a private room, which has all necessary facilities, and remain with them to provide support and information. Periodically, she will have to go to the resuscitation room to exchange information with both team leaders. If the family wish to be present in the resuscitation room the relatives' nurse should accompany them.

Once the primary survey has been completed, the leaders can disband the non-essential members of the team so that they can return to their normal activities in the department.

Depending on the co-ordination of the team, imaging of key areas may already have been carried out (see above). If not, these can be taken at the beginning or end of the secondary survey depending on appropriateness as determined by the team leader. Increasingly common is the early use of total body CT scanning using devices located in or close to the resuscitation room. In Europe this approach has been adopted where patients have received comprehensive pre-hospital care, the primary survey has been completed and the patient has been resuscitated and is known to be haemodynamically stable.

## **Secondary survey**

The objectives of this phase are to:

- Examine the patient from head to toe and front to back to determine the full extent of his injuries.
- Take a complete medical history.
- Assimilate all clinical, laboratory and radiological information.
- Construct a management plan for the patient.

The secondary survey may be completed in the resuscitation room, following vital, life saving surgery or in the Intensive Care Unit. In all cases this detailed secondary assessment needs to be carried out in a systematic fashion and the findings documented. This is the responsibility of the Team Leader.

**In the ED, should the patient deteriorate at any stage, the team leader must abandon the secondary survey and reassess the patient's airway, breathing and circulatory state in the manner described in the primary survey.**

As with the primary survey, a well-coordinated team effort is required. Procedures by individual team members follow a protocol and tasks are performed simultaneously rather than sequentially. The most common error is to be distracted before the whole body has been inspected; this must be avoided as potentially serious injuries can be missed, especially in the unconscious patient.

During the secondary survey the airway nurse maintains verbal contact with the patient while the recording nurse continues to measure the vital signs regularly and monitors fluid balance.

All victims of blunt trauma should now have chest and pelvic x-rays performed, along with a lateral cervical spine x-ray. The latter will help identify up to 85% of cervical spine abnormalities, providing all seven cervical vertebrae as well as the C7-T1 junction are shown. To facilitate this, one of the team members should pull the patient's arms towards their feet as the radiograph is taken (Fig.1.2) to remove the shoulders from the x-ray field. Alternative views (e.g. oblique and 'Swimmer's' view) or investigations (e.g. CT) can be used if this fails to give an adequate view. Further cervical spine views will be required before all injuries to the cervical spine can be excluded.



**Fig. 1.2: Pulling the arms down for a lateral cervical spine x-ray**

There are a number of methods of carrying out a secondary survey; the following is a guide to what must be accomplished.

### **Neurological State**

This starts with a reassessment of the conscious level using the Glasgow Coma Scale (GCS), the pupillary response and the presence of any lateralising signs (see Chapter 7). One of the circulation nurses should then continue to monitor these parameters. If there is any deterioration, then the primary survey must be repeated to rule out hypoxia or hypovolaemia as a cause before an intracranial injury is considered. The peripheral nervous system should then be examined as abnormalities of motor and sensory function can help indicate the level and extent of spinal injury as well as help identify any possible peripheral nerve injuries. In male patients the presence of priapism, may be the first indication of spinal injury. Once anaesthetised, it is impossible to perform a neurological assessment. However, lateralising eye signs persisting under general anaesthesia indicate a poor prognosis when they are not the result of cranial nerve injury due to base of skull fractures.

If the spinal cord has been transected at or above the mid-thoracic level, there is loss of sympathetic outflow, a reduction in vasomotor tone and peripheral vasodilatation causing hypotension. The degree of vasodilatation depends on how much vasomotor tone is lost and is maximal when transection of the spinal cord occurs in the cervical region. This removes all vasomotor tone and causes profound hypotension. There is no associated tachycardia because the sympathetic innervation of the heart (T1-T4) has also been lost. This condition is referred to as "neurogenic shock" (see Chapter 5).

### **The Scalp**

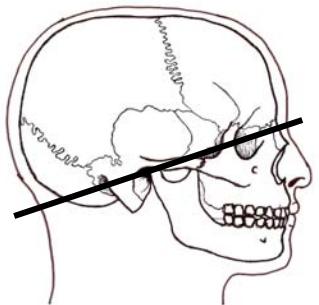
This must be examined for lacerations, swellings or depressions. Its entire surface must be inspected but the occiput will have to wait until the patient is turned or the cervical spine 'cleared' both clinically and radiologically. Visual inspection may discover fractures in the base of the lacerations. Wounds should not be blindly probed as further damage to underlying structures can result. If there is major bleeding from the scalp, digital pressure or a self-retaining retractor should be used. Although not common, scalp lacerations can bleed sufficiently to cause hypovolaemia; consequently haemostasis is crucial.

### **Base of Skull**

Fractures to this structure will produce signs along a diagonal line demonstrated in Fig. 1.3. Bruising over the mastoid process (Battle's sign) usually takes 12-36 hours to appear, it is therefore of limited use in the resuscitation room. A cerebrospinal fluid (CSF) leak via the ears or nose may be missed as it is invariably mixed with blood. Fortunately its presence in this bloody discharge can be detected by noting the delay in clotting of the blood and the double ring pattern when it is dropped onto an absorbent sheet. In this situation nothing, including an auroscope, should be inserted into the external auditory canal because of the risk of introducing infection and hence causing meningitis. As there is a small chance of a nasogastric tube passing into the cranium through an anterior base of skull fracture, these tubes should be passed orally when this type of injury is suspected.

### **Eyes**

The eyes should be inspected before significant orbital swelling makes examination too difficult. Look for haemorrhages, both inside and outside the globe, for foreign bodies under the lids (including contact lenses) and penetrating injuries. Pupil size and response to light, directly and consensually, should be recorded. If the patient is conscious, having them read a name badge or fluid label can be used as a simple test of visual acuity. If the patient is unconscious the corneal reflexes should be tested.



**Fig. 1.3: The line corresponding to the base of skull along which signs of a fracture may be found**

### The Face

This should be palpated symmetrically for deformities and tenderness. Check for loose, lost or damaged teeth and stability of the maxilla (the mid third of the face), by pulling the latter forward (see Chapter 13). Middle third fractures can be associated with both airway obstruction and base of skull fractures. Only injuries causing airway obstruction or severe haemorrhage need to be treated immediately. Mandibular fractures may also cause airway obstruction because of the loss of stability of the tongue.

### The Neck

The immobilisation devices must be removed for the team leader to examine the neck therefore the airway nurse will need to reapply manual inline stabilization. The neck should be inspected for any deformity (rare), bruising and lacerations. The spinous processes of the cervical vertebrae can then be palpated for tenderness or a "step off" deformity. The posterior cervical muscles should also be palpated for tenderness or spasm. The conscious patient can assist by indicating if there is pain or tenderness in the neck and locating the site.

A laceration should only be inspected and **never** be probed with metal instruments or fingers. If a laceration penetrates platysma, definitive radiological or surgical management will be needed. The choice depends on the clinical state of the patient (see Chapter 13).

### The thorax

The priority at this stage is to identify those thoracic conditions which are potentially life threatening, along with any remaining chest injuries (see Chapter 4). The chest wall must be re-inspected for bruising, signs of obstruction, asymmetry of movement and wounds. Acceleration and deceleration forces invariably leave marks on the chest wall, which should lead the team to consider particular types of injury. Good pre-hospital information is vital to determine the mechanism of injury.

The assessor should then palpate the sternum and along each rib, starting in the axillae and proceeding anteriorly. The presence of any crepitus, tenderness or subcutaneous emphysema must be noted. Auscultation and percussion of the whole chest can then be carried out to determine if there is any asymmetry between the right and left sides of the chest.

Potentially life-threatening injuries that need to be excluded include:

- Pulmonary and cardiac contusions – particularly after blunt trauma
- Ruptured diaphragm or perforated oesophagus – after either blunt or penetrating trauma
- Disruption of the thoracic aorta – after a deceleration injury
- A simple pneumothorax or haemothorax

Penetrating chest wounds can injure any of the structures within the thorax. Wounds that appear to cross the mediastinum pose a high risk of damaging the heart, bronchial tree or upper gastrointestinal tract. A high index of suspicion must be maintained and surgical consultation is mandatory.

### **The abdomen**

In the secondary survey, the aim is simply to determine if the patient requires a laparotomy - a precise diagnosis of which particular viscus has been injured is both time consuming and of little relevance. A thorough examination of the whole abdomen is required, including both the perineum and stability of the pelvis. All bruising, abnormal movement and wounds must be noted and lacerations should be inspected but not probed blindly as further damage can result. Any exposed bowel should be covered with warm saline-soaked swabs. If underlying muscle has been penetrated, it is not possible to determine the actual depth of the wound; consequently these cases will require further investigations (see Chapter 6).

The abdomen needs to be palpated in a systematic manner so that areas of tenderness can be detected. An intra-abdominal bleed should be suspected if the ribs overlying the liver and spleen (5 to 11) are fractured, the patient is haemodynamically unstable or if there are seat belt marks, tyre marks or bruises over the abdominal surface. Further investigation of possible abdominal injury can be achieved with ultrasound, CT scanning or diagnostic peritoneal lavage, according to local protocols. This should not delay the treatment of the haemodynamically unstable patient who should proceed directly to theatre. Early liaison with a surgeon is vital.

**The detection of abdominal tenderness is unreliable if there is a sensory defect due to neurological damage or drugs, or if there is fractures of the lower ribs or pelvis.**

Marked gastric distension is frequently found in crying children, adults with head or abdominal injuries and patients who have been ventilated with a bag and mask. The insertion of a gastric tube facilitates the abdominal examination of these patients and reduces the risks of aspiration.

A rectal examination should always be carried out. This provides five pieces of information:

- Sphincter tone - this can be lost after spinal injuries.
- Evidence of direct pelvic trauma.
- Evidence of a pelvic fracture.
- Prostatic position - this can be disrupted after posterior urethral injury.
- Blood in the lower alimentary canal.

The rate of urine output is an important indicator in assessing the shocked patient (see Chapter 5). Therefore it should be measured in all trauma patients and in most cases this will require catheterisation. If there is no evidence of urethral injury, the catheter is passed transurethrally. If urethral trauma is suspected (Box 1.6), and the patient is unable to urinate, a suprapubic catheter may be necessary. The urine that is voided initially should be tested for blood and a sample saved for microscopy and subsequent possible drug analysis.

**Box 1.6: Signs of urethral injury in a male patient**

- Bruising around the scrotum.
- Blood at the end of the urethral meatus.
- High riding prostate.
- Fractured pubic rami
- Inability to pass urine

The limbs are examined using inspection, palpation and movement and all long bones must be rotated. If the patient is conscious, he should be initially asked if any of the limbs are painful and subsequently to actively move each limb.

Any wounds associated with compound fractures must be swabbed and covered with a non-adherent dressing. As different surgeons will need to examine the limb, a digital photograph of the wound before it is covered will reduce the number of times the dressings have to be removed.

All limb fractures should be splinted to reduce; fracture movement, pain, bleeding, formation of fat emboli and secondary soft tissue swelling and damage.

A detailed inspection of the whole of the patient is needed to determine the number and extent of any soft tissue injuries. Each breach in the skin needs to be inspected to determine its site, depth and the presence of any underlying structural damage that will subsequently require surgical repair. Superficial wounds can be cleaned, irrigated and dressed in patients who are clinically stable.

Upon completion of the examination, the presence of any bruising, wounds and deformities must be noted along with any crepitus, instability, neurovascular abnormalities or soft tissue damage. As time delays can result in tissue loss, gross limb deformities need to be corrected and the pulses and sensation rechecked before any radiographs are taken.

### **The back**

If a spinal injury is suspected the patient should only be moved by a well-coordinated log rolling technique (Fig. 1.4). The patient is turned away from the examiner who takes this opportunity to clear away all the debris from under the patient. The whole of the back is assessed, from occiput to heels, looking for bruising and open wounds. The back of the chest must be auscultated, the area between the buttocks inspected and the vertebral column palpated for bogginess, mal-alignment and deformities in contour. The examination finishes with palpation of the longitudinal spinal muscles for spasm and tenderness. The patient is then log rolled back into the supine position. If the patient is still on a long spine board, this should now be removed, however, a patient may remain on a correctly applied vacuum mattress.



**Fig. 1.4: A well-coordinated log rolling technique**

Meticulous attention to prevention of decubitus ulcers must be made from the outset, particularly for patients with spinal injuries and the elderly because they are at high risk. Remember the patient may have already spent a considerable time in one position before being rescued and if surgery is required may have to remain in the same position for several more hours. It is therefore important to note how long the trauma victim remains stationary and to move whatever can be moved, every thirty minutes using hip lifts for example. For patients identified as high risk, consideration should be given to performing a formal assessment using a scoring system, e.g. the Waterlow system.

### **Medical history**

By the end of the secondary survey, the medical team leader must have assembled the patient's medical history. Some information will have already been acquired from the ambulance personnel or relatives. Further sources of information are the patient's Primary Care Physician and hospital records. A comprehensive medical history may help clarify clinical findings that do not appear to relate to the history of the incident, which led to the victim's condition. The important elements of the medical history can be remembered by the mnemonic AMPLE:

- A - Allergies
- M - Medicines
- P - Past medical history
- L - Last meal
- E - Events leading to the incident

### **Assimilation of information**

As the condition of the patient can change quickly, repeated examinations and constant monitoring of the vital signs is essential. The recording nurse, responsible for recording the latter at 5-minute intervals, must be vigilant and bring any deterioration in the respiratory rate, pulse, blood pressure, conscious level and urine output to the immediate attention of the team leaders.

By the end of the secondary survey, the answers to the following questions must be known:

1. *Is the patient's airway and ventilatory function satisfactory?*

If it is not adequate then the cause must be sought and corrected as a priority.

*2. Is the patient's circulatory status satisfactory?*

With less than 15% of the blood volume lost, vital signs usually return to normal after less than two litres of fluid. If they then remain stable then it implies that the patient is not actively bleeding. Patients whose vital signs initially improve but then decline suggests that they are actively bleeding or have started to bleed again during the resuscitation, may require typed blood and invariably require surgery. The total lack of response to a fluid challenge suggests that either the patient has lost 30-40% of their blood volume, depending on the mechanism of injury (see Chapter 5) and is bleeding faster than the rate of the fluid infusion or shock is not due to hypovolaemia. In the case of major haemorrhage, surgery is required urgently, and while this is arranged, the aim should be to resuscitate to ensure perfusion of the vital organs rather than restore normality. This will need invasive techniques to monitor the arterial and central venous pressures to help guide resuscitation (see Chapter 5).

*3. Is the patient's neurological status stable?*

Has there been any deterioration in the patient's GCS, have any localising signs developed?

*4. Are any further radiological investigations required?*

Any hypoxic or haemodynamically problems must be addressed first. Once his condition stabilizes, radiographs of particular sites of injury can be performed along with other specialized investigations. It is an important part of the team leaders' responsibilities to determine the priorities of these investigations.

*5. What is the extent and priorities of the injuries?*

The ABCDE system is used to categorize injuries so that the most dangerous is treated first. For example, problems with the airway (A) must be corrected before those of the circulation (C).

*6. Have any injuries being overlooked?*

The mechanism of injury and the injury pattern must be considered to avoid overlooking sites of damage. Trauma rarely 'skips' areas, for example if an injury has been found in the thorax and femur, but not in the abdomen, then it probably has been missed.

*7. Is tetanus toxoid, human antitetanus immunoglobulin (Humotet) or prophylactic antibiotics required?*

This will depend on both local and national policies that should be known by the team leaders.

*8. Is analgesia required?*

Severely injured patients need analgesia. Entonox (premixed nitrous oxide 50% and oxygen 50%) can be given if available, until the baseline observations are recorded unless there are any contraindications (i.e. pneumothorax and head injuries). Intravenous opioids can then be titrated against the patient's pain level.

### **Timing of interventions**

Treatment of trauma patients is often time-critical, but it is very easy for patients to spend a considerable time in the Emergency Room with little being done. The Trauma Team should therefore aim to complete each phase of the patient's care within specific time limits. This will help ensure that the patient's treatment continues to progress rather than come to a halt. An outline of such a system is shown in Box 1.7.

### **Definitive care**

This can only start once the patient has been adequately assessed and resuscitated. In many cases this will require transfer to either the operating theatre and/or intensive care. It is therefore very important that the transfer from the resuscitation room to these areas is done as smoothly as possible.

While the move is being planned, the medical team leader must complete the medical notes. At the same time the charts, vital signs, fluid balance, drug administration and preliminary nursing care documentation need to be collated. A purpose designed single trauma sheet can facilitate this process that also acts as a reminder so nothing is omitted and allows a record of all events. The relative's nurse can then brief the team leaders about the condition of any relatives or friends who are in the department on the patient's behalf. The medical team leader should accompany her back to the relative's room, to talk to them. If this doctor has had to accompany the patient another clinician, fully versed with the situation, should speak with the relatives.

### **Box 1.7: Timing of interventions**

**Red:** (within 1 minute)

*Immediately life-saving interventions (“ABCDE”)*

- Clearing airway
- Oxygenation and ventilation
- Compression of massive external haemorrhage, pelvic compression

**Orange:** (within 15 minutes)

*Very Urgent Interventions*

- Intubation
- Immobilisation of the cervical spine
- IV access and bloods sent for laboratory examination
- Fluid therapy
- Commence monitoring
- Sonography
- Undressing the patient
- Complete primary survey
- Chest and pelvic x-rays

**Yellow:** (within 30 minutes)

*Urgent Tests and Interventions*

Complete secondary survey

Urinary catheter

CT-Scan

Focused x-rays

**Green** (within 3 hours)

- Completion of diagnostics and therapy
- Special investigations and x-rays
- Operative care
- Intensive care

If the patient is unconscious, his clothing and belongings may provide essential information to help establish his identity. Whether the patient's name is known or not, a system of identification is required, in order that drugs and blood can be administered safely. This is usually in the form of emergency hospital numbers, reserved for such occasions. This becomes more important when there are several patients in the resuscitation room. If identity bracelets are impractical, then indelible markers can be used to write a number on the patient's skin. The rescue personnel must hand over any possessions brought in with the patient to the nursing staff. These must be kept safely, along with the patient's clothing and property. At the end of the secondary survey, or during it if there are hands to spare, all these articles must be searched. A check is needed for any medical alert card or disc, a suicide note and any medicine bottles or tablets.

## **No patient must is allowed to leave the department without a form of identification.**

Jewellery, and when appropriate dentures, need to be removed with permission if conscious and stored in a labelled valuables bag or envelope. As soft tissue can swell, constrictive jewellery must be removed and if this is not possible, it should be cut off. At an appropriate moment, the patient's property is collected preferably by nurses outside the trauma team, checked, recorded, signed for and locked away. Whatever the outcome of the resuscitation, relatives take a dim view of items of property being misplaced. Nurses are legally responsible for this property and prolonged problems can result from disregarding the patient's seemingly unimportant effects in the heat of the moment. If a criminal case is suspected, all clothing, possessions, loose debris, bullets and shrapnel are required for forensic examination. These too must be collected, labelled, placed in individual bags and signed for prior to releasing them to the appropriate authorities according to established procedures.

If a delay in transfer is anticipated, the patient can be given a gentle, preliminary wash to remove any blood, mud or other contaminating material.

### **Preparation for transfer of the patient**

The safe transfer of patients is vital, whether intra or inter-hospital. This topic is covered in greater detail in Chapter 18. First, it is necessary to decide where the patient needs to go to, then, how soon do they need to be there?

### **Communication**

To facilitate a smooth transfer, it is important to ensure the receiving facility and personnel have been contacted directly by the medical team leader. If an inter-hospital transfer is envisaged, the clinicians must also decide on the most suitable method of transportation and how the patient should be prepared for the journey. For example, the journey may be quicker by air, but take much longer to organise, and involve additional journeys at both ends.

### **Assessment**

Any intervention during the primary survey must be reassessed before the patient leaves and appropriate adjustments made. For example, the patient who tolerates an oropharyngeal airway should be intubated and ventilated to secure the airway and prevent hypoxia and hypercarbia. All cannulas, catheters, tubes and drains must be firmly secured.

## **Monitoring**

Monitoring must be continued during the transfer period to ensure that ventilation and tissue perfusion are adequate. An ECG monitor, automatic BP recorder, capnograph and pulse oximeter are the minimum acceptable and all devices must be fully charged.

**If a parameter needs to be monitored before transfer,  
it also needs to be monitored during transfer.**

## **Equipment and drugs**

The trolley carrying the patient during inter hospital transfer must also transport the suction system, oxygen supply, ventilator, portable blood pressure monitor and defibrillator-monitor. Airway adjuncts, needles and drugs, appropriately labelled and packed, are usually carried separately by one of the medical team. Fully charged spare batteries for the monitors should be available. Portable ventilators use their gas supply as their power source, therefore an adequate amount of oxygen must be taken. The same principles apply for intra-hospital transfers.

## **Transfer personnel**

The patient needs to be accompanied by appropriately trained staff to enable them to monitor and intervene with any “ABCDE” problems. If he is intubated, the most suitable personnel are the airway nurse and an anaesthetist.

## **Records**

All the medical and nursing notes, radiographs, blood tests, Identifying labels and, if necessary, consent forms or good quality copies must be taken with the patient.

## **Relatives and friends**

The nurse dealing with these people must inform them about the transfer. When the trauma victim is to be moved to another hospital, this nurse should also help the friends and relatives make their own transportation arrangements.

## **Final check**

Before moving off, the team must ensure that the patient is appropriately secured to the transfer trolley and covered to prevent cooling.

### **Upon arrival**

The transfer team must hand over to the doctor and nurse who will be in charge of the patient's definitive care. In this way a summary of the initial resuscitation and important events during transfer can be given. All documentation can be handed over at this stage and the transfer equipment retrieved.

### **Preparation of the resuscitation room**

As the transfer is under way, the remaining staff can begin preparing the area for arrival of the next trauma victim. Throughout the resuscitation the team should have kept the area as tidy and organized as possible with sharps, open packs and instruments being disposed of as they are used. This is essential for both safety and efficiency. Wet, greasy floors need to be wiped or covered immediately after spillage to avoid accidents to staff.

Once the patient has been transferred and the resuscitation area restocked, checked and made ready, the team can get together for a preliminary or definitive debriefing session.

### **Summary**

To enable the patient to receive the most efficient resuscitation, a group of appropriately trained personnel must be ready to meet him when he arrives at the emergency department. These people must be coordinated by nursing and medical team leaders so that they are all aware of the tasks they have to perform, and that they are carried out simultaneously.

The first priority is to detect and treat the immediately life threatening conditions. Following this, a detailed head to toe assessment can be completed. The team leaders can then list the patient's injuries and their priorities for both further investigations and definitive treatment.



## Chapter 2

# THE ROLES AND RESPONSIBILITIES OF THE TRAUMA TEAM

## Objectives

At the end of this chapter you should understand:

- The factors affecting a team's performance
- The structure and function of a trauma team.
- The role and responsibilities of the trauma team members

## Team performance

A number of factors are known to affect a team's performance. Four of these inter link and are particularly important when considering the effectiveness of a trauma team:

- Team function
- Team leadership
- Team organisation & task allocation
- Team size & personnel

## Trauma team function

When dealing with a trauma victim, the team has clearly defined goals which need to be known and understood by each member:

- Identify and treat life threatening injuries
- Identify any other problems
- Arrange appropriate treatment and investigations
- Arrange and transfer to definitive care

The type and extent of the injuries will determine how difficult the case is. From the team's perspective there is a range of problem complexity which will allow them to work effectively. Members can become careless or bored when dealing with something they feel is too easy or familiar. Conversely, too hard a situation can lead to defeatism and team dysfunction. These thresholds are therefore also dependent upon individual team members' experience and competencies.

## Trauma team leadership

To enable the team to achieve the resuscitation goals, the team leader needs to take up the following key roles:

1. Command & control
2. Coordination
3. Communication

## **1. Command & Control**

At all times the team leader must remain in overall control of the team even as the make up of the team changes as the resuscitation progresses. This means the person needs to have the experience of dealing with similar situations in the past and be aware of the tasks which are required. Furthermore, the team leader ideally should be aware of the hospital resources at his disposal, for example; critical care beds, surgical capacity, radiology etc. Without this the leader is at risk of loosing credibility and authority if the resuscitation becomes difficult. This does not mean the leader has to have a greater level of competence in each of the skills compared to the actual team member (e.g. greater airway skills than the doctor allocated to deal with the airway). However the leader needs to recognise when a skill is needed, what is required and how to recognise and deal with the problem of not being able to complete a particular intervention (e.g. failed intubation). In most of Europe this means he/she will be a medical specialist in Emergency Medicine, Surgery, Anaesthesia or Critical Care.

### **Team leaders need experience not simply seniority**

In an ideal system, command and control starts with the team leader ensuring that local facilities are capable of managing the expected problems. If not, the question may need to be asked “is redirecting the patient a safer option?” Once the decision has been made, or when there are no options, the particular skills of each of the team members and ensuring that they are tasked appropriately, preferably before the arrival of the patient. As soon as the patient arrives the leader must take control of the situation, firstly by carrying out the “5 second round”. This ensures the team responds to the most obvious life-threatening conditions as a priority and changes to the team organisation can be carried out. Once this has occurred, the team leader should “take a step back” and allow the team to perform. Control should not mean that every action of the team has to be initiated by the leader – by observing the actions of the team and only intervening when there are concerns or a need for information will allow the team to perform most effectively.

## **2. Coordination**

The team leader is responsible for ensuring the team members carry out their allocated tasks effectively and efficiently (see below).

As the team carry out their tasks they will start to relay information to the leader. This will need to be assimilated and acted upon accordingly to ensure that problems are anticipated and interventions are carried out in a timely manner. Identification of a major airway problem requiring drug-assisted tracheal intubation will require the team leader to coordinate personnel to assist with this task. In addition there are likely to be several investigations or interventions being performed simultaneously and the team leader will need to coordinate these in order that they are performed safely and efficiently. This is particularly true when obtaining x-ray investigations to ensure that team members are not inadvertently exposed.

### **3. Communication**

#### *Communication with the team*

The team leader needs to communicate with all the team members. This requires knowing the names and competences of each member of the team. The team leader must create an environment that promotes open communication between all the team to ensure that any concerns about the clinical condition of the patients and adequacy of procedures are freely and openly expressed to prevent errors of omission and commission. This facilitates the flow of information in both directions between the team members and the team leader (Box 2.1).

Once the patient arrives the leader needs to question the ambulance personnel or the pre-hospital medical team to ascertain if there are any immediate problems and clarify the mechanism of injury, the pre-hospital findings, the treatment given so far and response to treatment. As the primary survey and resuscitation progress, the leader acts as a point of focus and if necessary be able to gain support to carry out various tasks, either themselves or with the assistance of the team leader. It must also be remembered that in many trauma patients, important communication will occur between the team leader and the patient. Depending on the team's skills the team leader may have to carry out particular procedures, e.g. chest drain insertion, ultrasound, and thoracotomy.

**Box 2.1: Trauma team leader communication**

Ensures that everyone in the team is introduced to each other

Sets the priorities after the five second round, ensuring that any life-threatening injury is immediately addressed

Listens to the information from all the team members:

Pre-hospital team and/or ambulance control

Each of the ABC personnel

Gives advices and/or instruction when necessary.

Liaises with specialists who have been called and communicates this information to the team.

Conveys information from investigation to the team members to facilitate their management of the patient

Discuss with the nursing team leader any areas of concern, e.g. relatives

*Communication with the trauma patient*

When caring for the severely injured, there is a tendency for the team focus on the acute medical problems rather than the patient as an individual, thereby depersonalising the encounter. This can be frightening for the patient and may result in missed injuries, erroneous or unnecessary treatment. Establishing contact with the patient as early as possible and maintaining communication throughout the resuscitation is therefore essential for all team members and especially the team leader. Communication allows:

- Assessment of the patient's level of consciousness (LOC). This is a valuable indicator of alteration of many vital functions, for which the LOC is the final common pathway. Maintaining communication with the patient throughout the resuscitation provides an excellent, non-invasive way of monitoring of LOC. Any deterioration of the LOC indicates deterioration of the patient's condition and the cause rapidly identified and treated.
- The patient to provide useful information. This may be details of the mechanism of injury that may reveal occult injuries, or direct requests to carry out various tasks; asking the patient to "take a deep breath" may reveal asymmetric chest expansion, not seen on normal breathing or cause pain on inspiration indicating rib fractures and the possibility of other underlying injuries.
- An explanation of procedures, particularly those that may cause pain for example inserting a chest drain. Giving comfort and reassurance are important to obtain the patient's cooperation, whilst reducing anxiety and unwanted exaggerated sympathetic activity.
- Pain and other symptoms as shortness of breath, anxiety, nausea etc can be monitored continuously and treatment adjusted accordingly.
- Preservation of dignity in the awkward settings of an emergency department.

It is the team leader's responsibility to ensure that adequate communication with the patient is maintained throughout the resuscitation.

### **Trauma team organisation & task allocation**

In the initial stages of the resuscitation a number of activities need to be carried out. An effective way of doing this is to complete each of these tasks simultaneously. This is known as 'horizontal organisation' as opposed to 'vertical organisation' where the tasks are carried out sequentially, often when personnel and resources are limited. It follows that all team members need to be aware of their duties and be capable in carrying them out. Therefore, before the patient arrives, team members need to be briefed so that they can:

- See where their role fits into the overall organization.
- Appreciate the importance of their role.
- Anticipate what may be required as the resuscitation progresses.
- Extend their role in an emergency.

The tasks which are allocated will vary from unit to unit, and from country to country. A simple and robust way of doing this is for the team leader to allocate appropriate personnel, according to local protocol, to deal specifically with:

- Airway and cervical spine control
- Breathing and ventilation
- Circulation and control of haemorrhage
- Relatives
- Imaging

Depending on the department there may one or two people (e.g. Doctor and/or Nurse) allocated to each of the first three tasks. Pairing-up doctors and nurses, where resources allow, increase the efficiency of the team. Occasionally, when demand exceeds resources, for example multiple patients after a car accident, the numbers of team members may be reduced leading to the need to merge functions. Typically the team leader may have to adopt the role of "breathing". The support for the relatives may be carried out by any of the medical or nursing staff; what is important is that the individual assigned to this task is appropriately trained and skilled in what can be a very demanding role. In most departments, imaging will be carried out by a radiographer. Once tasks have been allocated the following responsibilities arise;

## **Airway personnel**

Establish a rapport with the patient giving psychological support throughout his/ her ordeal in the resuscitation room. Because of their location, information is often given to the patient by the airway team.

- Clear and secure the airway whilst taking appropriate cervical spine precautions
- Check any airway devices inserted pre-hospital
- Ensure adequate ventilation with a high concentration of oxygen and appropriate monitoring
- Place arterial and central lines
- Provide analgesia and anaesthesia

The airway team need to have the skills necessary to deal with the trauma airway (see Chapter 3). In practice this usually means they are either anaesthetists or doctors and nurses with significant anaesthetic training.

## **Breathing personnel**

- Remove chest clothing
- Assess the chest, neck and effectiveness of ventilation
- Establish SpO<sub>2</sub> monitoring
- Take ABG sample if necessary
- Keep contemporaneous records of the patient's vital signs, fluid infusions and drug administration.
- Note times of interventions and arrival of other specialities.
- Help with filling in request cards and liaising with laboratories
- Insert needle thoracentesis or chest drain when required
- Assist in special procedures, for example a thoracotomy

## **Circulation personnel**

- Assist in stemming external haemorrhage by direct pressure
- Establish peripheral intravenous (IV) infusions and take bloods for investigations.
- Confirm IV lines inserted pre-hospital are functioning
- Connect the patient to the monitors and measure the vital signs.
- Assess the abdomen and pelvis
- Carry out certain procedures, for example urinary catheterisation.
- Carry out other procedures depending on their skill level (e.g. placing of arterial, IO and central lines).
- Monitor the fluid balance

- Assist in the removal of the patient's clothes.
- Commence chest compressions if cardiopulmonary resuscitation is required.

### **Support for the relatives**

Whoever is assigned this role will need to be fully informed of the patient's personal details and able to explain to the relatives, in terms that they can understand, the events leading up to admission, care given and response so far.

- Cares for the patient's relatives when they arrive.
- Liaises with the trauma team to provide the relatives with appropriate information and support.
- Accompanies the relatives if they wish to be present in the resuscitation room.

### **Radiographer**

- Takes two standard x-rays of the chest and pelvis on all patients subjected to blunt trauma.
- Dependent upon the mechanism of injury and the departmental protocols, takes a lateral view of the C-spine
- Depending upon the unit, may perform abdominal, pleural and cardiac sonography

**The team leader needs to be aware of the competence of each member of the team so that additional help can be provided should it be necessary.**

### **The trauma team size and personnel**

As described above the typical trauma team is made up of several members of staff. Which staff are used will vary between hospitals. The key point is they know their role and are competent to carry it out.

**Each member has to be thoroughly familiar with his/her respective duties so that tasks can be performed simultaneously, in the shortest possible time, for the maximum benefit of the patient.**

On first sight it appears that a lot of personnel will be taken away from the department for a long time. However, the aim is to achieve an efficient and rapid correction of all the immediately life threatening conditions. Once this has been completed, only the core personnel need to remain.

The group of doctors and nurses who make up the trauma team must be immediately available and organised to ensure that delays are avoided. They need to assemble once the

pre-hospital information is received, so that they are in place when the patient arrives. During this preparatory time they need to take universal precautions and check the equipment relevant to their allocated task.

The optimal team size is dependent on the complexity of the problem faced, the type of organisation used, quality of leadership and the familiarity of the personnel working together. To avoid confusion there should be no more than six people physically touching the patient. The other team members must keep well back.

### **Problems with trauma teams**

Even with an established trauma team there is always the potential for organisational failure. This is particularly likely when there is a hand over between personnel or to other specialists. Such instances of failure are often referred to using the sporting, colloquialism of a team member “dropping the ball”. In all cases it is down to the team leader to ensure that all team members are good “passers and catchers of the ball” to prevent it being dropped (Box 2.2) and the patient’s care being compromised.

#### **Box 2.2: Common examples of “ball-dropping” in the ED**

- Failure to obtain an adequate handover from the prehospital team to the reception team
- Inadequate or inappropriate prioritising of imaging/investigations
- Failure to prioritise the order of definitive care
- Failure of communication with other specialists
- Inadequate arrangements of on-going in-patient care

In the first case the keenness to start their specific task can result in no one debriefing the pre-hospital personnel. Once the secondary survey is finished all the actual and potential injuries should be known. It is then down to the team leader to determine which specialist images or investigations are required, what treatment is needed and, in both cases to prioritise the sequence in which they are performed. Tact, diplomacy and experience are needed to ensure the order is for the patients benefit rather than the convenience of any particular speciality.

In some European countries, a policy of “shared care” is used, especially where the specialty of Emergency Medicine is not established. An example of the system in these circumstances would be for the Anaesthetist to lead the team until the patient reaches the operating theatre. At this point the surgeon takes over and is responsible for leading and co-ordinating subsequent care. It is during the hand over between team leaders that prioritises and information can be lost.

## **The importance of clear, accurate documentation cannot be over emphasised**

Eventually the patient needs to remain under the charge of a particular team once they leave the ED. The speciality responsible for this varies across Europe. In all cases however those in charge must look after the whole patient – not just the part they are interested in. Experience from a number of countries has shown ongoing co-ordinated care of admitted trauma patients can be achieved by using a trauma nurse co-ordinator (TNC). This is replacing the traditional method of having a particular surgical team responsible for all the patients care. Studies have shown that a TNC leads to better integration between specialties, optimal bed usage, better quality control and early discharging.

### **Team training**

As with any activity, practice is important in ensuring the team works in a coordinated fashion and overcomes the problems encountered when a trauma team concept is introduced (Box 2.3).

#### **Box 2.3: Problems encountered on introducing a “Trauma Team” concept**

- Medical & nursing hierarchies
- Speciality variations
- Ignorance of role
- Ignorance of other team members
- Ignorance of effective team attributes
- Frequency of team activation
- Case variation

Infrequency of activation will exacerbate all the problems listed in Box 2.2. Units which are busy will have to opportunity to practice and perfect their teams in real life situations. Conversely, those not seeing regular trauma cases need to maximise the educational input from debriefing after each resuscitation, video and reviewing performances, simulators and audit. Group training, e.g. all members doing the same trauma course, is another method of building and establishing the team. It does this by breaking down inter-professional barriers, increases knowledge and roles and improves communication.

## **Summary**

- Various ways of achieving an effective trauma team dependent on local needs and resources
- Organisation is an important factor effecting trauma team dynamics
- The principles used in achieving a successful trauma team should continue throughout the patients stay in hospital

## Chapter 3

# AIRWAY MANAGEMENT IN THE TRAUMA PATIENT

### Objectives

The objectives of this chapter are to teach the team members allocated to manage the trauma patient's airway:

- How to assess and secure the airway
- Basic airway management
- The use of simple airway adjuncts
- The use of supraglottic airway devices
- Techniques for ventilation of the patient's lungs
- The use of surgical airways in the resuscitation room

Advanced airway management and the role of drugs to aid intubation are covered in Appendix 3.1.

### Introduction

Establishing and maintaining a clear airway are essential prerequisites for successful resuscitation and are therefore the first medical interventions in the trauma patient. If the cervical spine is injured, the spinal cord may be jeopardised if airway interventions are not controlled carefully. This chapter will describe how to assess, clear, and secure the airway while minimising the risk of injuring the spinal cord.

### Causes of airway obstruction

Obstruction of the airway may be partial or complete. It may occur at any level from the nose and mouth down to the bronchi. In the unconscious patient, the commonest site of airway obstruction is at the level of the pharynx. It had previously been attributed to decreased muscle tone allowing posterior displacement of the tongue onto the posterior pharyngeal wall. Studies of patients under general anaesthesia have shown that the site of airway obstruction is more often at the soft palate and epiglottis and not the tongue. Obstruction may also be caused by vomit or blood, unstable fractures of the maxilla or mandible and direct obstruction of the larynx by foreign bodies or swelling. Upper airway stimulation or inhalation of foreign material may cause laryngeal spasm.

## **Standby preparation and transfer**

Although all equipment must be checked regularly, the doctor and nurse responsible for managing the airway and cervical spine must complete a final check of equipment while waiting for the patient to arrive.

On arrival at hospital, the patient's airway must be assessed immediately, even before transfer from the ambulance to the resuscitation room. If there is evidence of compromise, clear and secure the airway using simple techniques (see below) while simultaneously securing the neck. This may be with a rigid cervical collar alone or combined with lateral blocks and tape. Alternatively, manual inline stabilisation (MILS) of the cervical spine (Fig. 3.1) can be used. Ensure that there is no uncontrolled movement of the spine or loss of airway patency during transfer to, or within, the resuscitation room.



**Fig. 3.1: Manual stabilisation of the cervical spine**

### **Assessing and securing the airway**

The quickest way of evaluating the airway is to ask the patient "Are you alright?" A lucid reply implies that the airway is clear, the patient has a reasonable vital capacity breath, and cerebral perfusion is sufficient to maintain consciousness. An impaired response can be caused by partial obstruction. This may be the result of:

- A reduced level of consciousness
- The presence of blood, vomit or a foreign body
- Trauma to the face or neck
- Swelling of the pharyngeal soft tissues
- Laryngeal spasm (stridor)

- Bronchospasm (wheeze)

Assessment of the airway is best achieved using the look, listen and feel approach:

- **LOOK** at the patient and specifically for chest and abdominal movements
  - Patients are usually tachypnoeic (respiratory rate >29/min), agitated, sweaty and tachycardic due to hypoxia and hypercarbia.
  - During airway obstruction, accessory muscles of respiration are used - the neck and the shoulder muscles contract to assist movement of the thoracic cage. There may also be intercostal and subcostal recession and a tracheal tug.
- **LISTEN** for any noise associated with breathing. Normal breathing should be quiet, noisy breathing indicates partial airway obstruction.
  - Inspiratory stridor - is caused by obstruction at the laryngeal level or above.
  - Expiratory wheeze - suggests obstruction of the lower airways, which tend to collapse and obstruct during expiration.
  - Gurgling - suggests the presence of liquid or semisolid foreign material in the upper airways.
  - Snoring - arises when the pharynx is partially occluded by the tongue or palate.
  - Crowing or stridor - is the sound of laryngeal spasm or obstruction.
- **FEEL** for airflow at the mouth and nose.
  - In partial airway obstruction, air entry is diminished.

Complete airway obstruction in a patient who is making respiratory efforts causes paradoxical chest and abdominal movement, described as 'see-saw breathing'. As the patient attempts to breathe in, the chest is drawn in and the abdomen expands; the opposite occurs in expiration. This is in contrast to the normal breathing pattern of synchronous movement of the abdomen upwards and outwards (pushed down by the diaphragm) with lifting of the chest wall. Full examination of the neck, chest and abdomen should enable differentiation of the movements associated with complete airway obstruction from those of normal breathing. Listen for airflow:

completely obstructed breathing will be silent and no air movement will be felt at the mouth.

Unless airway obstruction can be relieved to enable adequate lung ventilation within a few minutes it will cause injury to the brain and other vital organs, and cardiac arrest. Give high concentration oxygen while attempting to relieve airway obstruction. As airway patency is restored, blood oxygen saturation will be restored more rapidly if the inspired oxygen concentration is high.

## **Basic techniques for opening the airway**

Initial manoeuvres to relieve obstruction of the airway include: chin lift (Fig.3.2), jaw thrust (Fig. 3.3), and removal of foreign material using a rigid sucker for liquid, Magill's forceps or similar device for loose or solid debris. Assess the effectiveness of all of these manoeuvres using the look, listen and feel method.



**Fig. 3.2: Chin lift**

Place the fingertips of the one hand under the point of the patient's chin, and gently lift to stretch the anterior neck structures.



**Fig. 3.3: Jaw thrust**

Identify the angle of the mandible. With the index and other fingers placed behind the angle of the mandible, apply steady upwards and forward pressure to lift the mandible. Using the thumbs, slightly open the mouth by downward displacement of the chin. Note use of facemask with reservoir to deliver oxygen.

### Simple airway devices

In the unconscious patient, the chin lift or jaw thrust be all that is required to obtain a clear airway, but once the jaw is released the obstruction will recur. Oropharyngeal and nasopharyngeal airways are designed to overcome soft palate obstruction and backward tongue displacement in an unconscious patient, but jaw thrust may also be required.

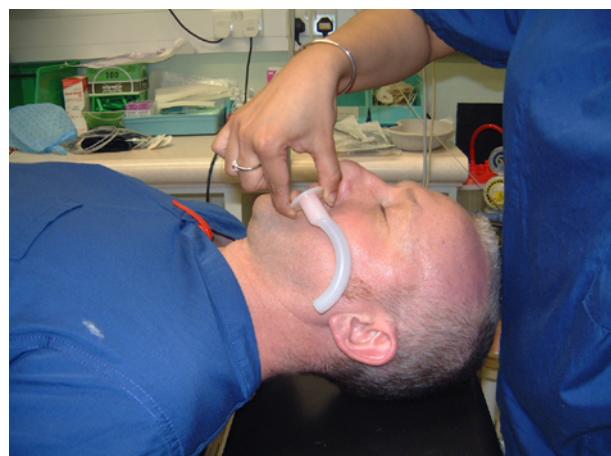
### Oropharyngeal (Guedel) airway

The oropharyngeal or Guedel airway is a curved plastic tube, flanged and reinforced at the oral end with a flattened shape to ensure that it fits neatly between the tongue and hard palate (Fig. 3.4). An estimate of the size required may be obtained by selecting an airway with a length corresponding to the vertical distance between the patient's incisors and the angle of the jaw (Fig. 3.5). The most common sizes are 2, 3 and 4 for small, medium and large adults respectively.

During insertion of an oropharyngeal airway, the tongue can occasionally be pushed backwards, exacerbating obstruction instead of relieving it. Ensuring a correct insertion technique should avoid this problem. Attempt insertion only in unconscious patients: vomiting or laryngospasm may occur if glossopharyngeal or laryngeal reflexes are present.



**Fig. 3.4: Oropharyngeal airways**

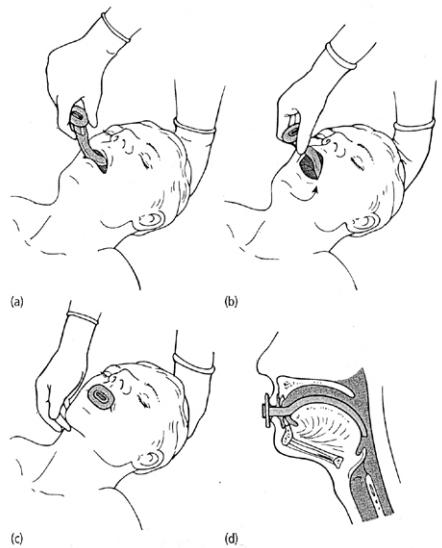


**Fig. 3.5: Sizing an oropharyngeal airway**

### Technique for insertion of oropharyngeal airway

- Open the patient's mouth and ensure that there is no foreign material that may be pushed into the larynx (if there is any, then use suction to remove it).
- Insert the airway into the oral cavity in the "upside-down" position as far as the junction between the hard and soft palate and then rotate it through 180° (Fig. 3.6a-d). Advance the airway until it lies in the pharynx. This rotation technique minimises the chance of pushing the tongue backwards and downwards. Remove the airway if the patient gags or strains. Correct placement is indicated by an improvement in airway patency and by the

seating of the flattened reinforced section between the patient's teeth or gums (if edentulous).



**Fig. 3.6: (a-d): Insertion of an oropharyngeal airway**

### Nasopharyngeal airway

This is made from soft malleable plastic, bevelled at one end and with a flange at the other (Fig. 3.7). In patients who are not deeply unconscious, it is better tolerated than an oropharyngeal airway. It may be life-saving in patients with clenched jaws, trismus or maxillofacial injuries. Inadvertent insertion of a nasopharyngeal airway through a fracture of the skull base and into the cranial vault is possible, but extremely rare. In the presence of a known or suspected basal skull fracture an oral airway is preferred, but if this is not possible, and the airway is obstructed, gentle insertion of a nasopharyngeal airway may be life-saving (i.e., the benefits may far outweigh the risks).



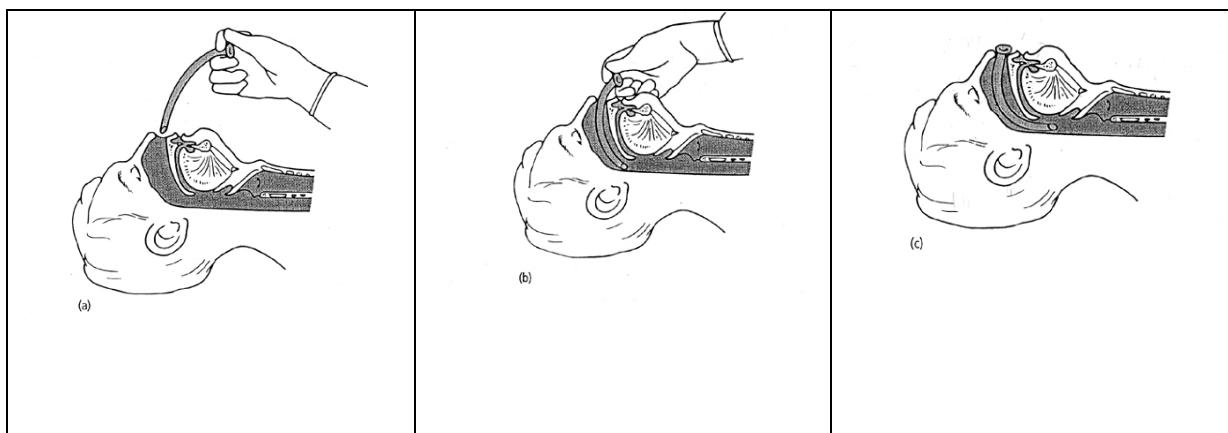
**Fig. 3.7: Nasopharyngeal airways**

The tubes are sized in millimetres according to their internal diameter, and the length increases with diameter. The traditional methods of sizing a nasopharyngeal airway (measurement against the patient's little finger or anterior nares) do not correlate with the airway anatomy and are unreliable. Sizes 6-7 mm are suitable for adults. Insertion can cause

damage to the mucosal lining of the nasal airway, resulting in bleeding in up to 30% of cases. If the tube is too long it may stimulate the laryngeal or glossopharyngeal reflexes to produce laryngospasm or vomiting.

#### Technique for insertion of nasopharyngeal airway

- Check for patency of the right nostril.
- Some designs require a safety pin to be inserted through the flange to provide an extra precaution against the airway disappearing beyond the nares.
- Lubricate the airway thoroughly using water-soluble jelly.
- Insert the airway bevel end first, vertically along the floor of the nose with a slight twisting action (Fig. 3.8a-c). The curve of the airway should direct it towards the patient's feet. If any obstruction is met, remove the tube and try the left nostril.
- Once in place, use the look, listen and feel technique to check the patency of the airway and adequacy of ventilation. Chin lift or jaw thrust may still be required to maintain airway patency.



**Fig. 3.8a-c: Insertion of a nasopharyngeal airway**

After insertion of either an oro- or nasopharyngeal airway, maintain chin lift or jaw thrust, and check the patency of the airway and ventilation using the look, listen and feel technique. Auscultate the chest to confirm bilateral ventilation and give oxygen. If there is any possibility of cervical spine injury, maintain alignment and immobilisation of the head and neck. Suction is usually possible through the airway device using a fine-bore flexible suction catheter. Complications from airway insertion are listed in Box 3.1.

### **Box 3.1: Complications from airway insertion**

- Trauma to all structures encountered, provoking bleeding
- Partial or complete airway obstruction
- Laryngeal spasm because the device is too long
- Vomiting because the patient has a gag reflex

## **Breathing**

Try to deliver an inspired oxygen concentration of 100% to all trauma patients. The concentration of oxygen delivered is often referred to as the  $\text{FiO}_2$  (fractional inspired oxygen concentration) and expressed as a decimal rather than a percentage, e.g. 21% =  $\text{FiO}_2 0.21$

### ***Spontaneous ventilation***

If simple airway manoeuvres, with or without an adjunct achieve a patent airway and resumption of breathing, deliver oxygen using a facemask with a reservoir (non-rebreathing bag). Connect to an oxygen flow of  $15 \text{ l min}^{-1}$ , ensure that the reservoir is full before placing over the patient's nose and mouth and mould to get a good, close fit. During inspiration, the continuous flow of oxygen ( $12\text{--}15 \text{ l min}^{-1}$ ) combined with the oxygen in the reservoir ensures minimal entrainment of air, raising the inspired concentration to approximately 80%, providing that the reservoir bag inflates and deflates with each breath (Fig. 3.3).

### ***Artificial ventilation***

If spontaneous ventilation is inadequate to maintain oxygenation despite an increase in the inspired oxygen concentration using a facemask with reservoir, or if the patient is apnoeic, assisted ventilation is required. The simplest and most widely used device is the bag-mask device. The self-inflating bag can be connected to a facemask, tracheal tube, or alternative airway devices such as the LMA or Combitube™. As the bag is squeezed, the contents are delivered to the patient's lungs. On release, the expired gas is diverted to the atmosphere via a one-way valve; the bag then refills via an inlet at the opposite end. When used without supplemental oxygen, the self-inflating bag ventilates the patient's lungs with only ambient air (oxygen concentration 21%). This is increased to approximately 45% by attaching an oxygen flow of  $5\text{--}6 \text{ l min}^{-1}$  directly to the bag adjacent to the air intake. An inspired oxygen concentration of approximately 85% is achieved if a reservoir system is attached and the oxygen flow is increased to approximately  $10 \text{ l min}^{-1}$ . As the bag re-expands it fills with oxygen from both the reservoir and the continuous flow from the attached oxygen tubing.

Although the bag-mask apparatus enables ventilation with high concentrations of oxygen, its use by a single person requires considerable skill. When used with a facemask, it is often

difficult to achieve a gas-tight seal between the mask and the patient's face, and maintain a patent airway with one hand whilst squeezing the bag with the other. Any significant leak will cause hypoventilation and if the airway is not patent, gas may also be forced into the stomach. This will reduce ventilation further and greatly increase the risk of regurgitation and aspiration. There is a natural tendency to try to compensate for a leak by excessive compression of the bag, which causes high peak airway pressures and forces more gas into the stomach. Some self-inflating bags have flow restrictors that limit peak airway pressure with the aim of reducing gastric inflation. Cricoid pressure can further reduce the risk of gastric inflation but requires the presence of a trained assistant, but if poorly applied, it may make ventilation more difficult. Consequently, on most occasions, the two-person technique for bag-mask ventilation should be used (Fig. 3.9); one person holds the facemask in place using a jaw thrust with both hands and an assistant squeezes the bag. In this way, a better seal can be achieved and the patient's lungs can be ventilated more effectively and safely.

#### *Cricoid Pressure*

This procedure is usually reserved for use during intubation (see Appendix 3.1). However it can be minimise gastric distension during ventilation with a facemask and thereby reduce the chance of aspiration.



**Fig. 3.9: Two person technique for using a bag-mask device. Note third person immobilising the head and neck.**

#### ***Monitoring oxygenation with a pulse oximeter***

A probe, containing a light emitting diode (LED) and a photodetector is attached to the tip of a digit or earlobe. The LED emits red light alternately at two different wavelengths, in the visible and infrared regions of the electromagnetic spectrum. These are transmitted through the tissues and absorbed to different extents by oxyhaemoglobin and deoxyhaemoglobin. The intensity of light reaching the photodetector is converted to an electrical signal. The absorption by the tissues and venous blood is constant and is subtracted from the beat-to-beat variation of the arterial blood to display the arterial oxygen saturation ( $\text{SpO}_2$ ), both as a

waveform and a digital reading. Pulse oximeters are accurate to  $\pm$  2%. The waveform can also indicate the heart rate. Alarms are provided for saturation and heart rate. The pulse oximeter therefore provides information about both the circulatory and respiratory systems, and has the advantages of:

- providing continuous monitoring of oxygenation at tissue level;
- being unaffected by skin pigmentation;
- portability (mains or battery powered);
- being non-invasive.

There are several important limitations to pulse oximetry:

- A saturation of 90% equates to a  $\text{PaO}_2$  of only 8 kPa (60 mmHg) – oxygen content of blood decreases rapidly below this.
- It is unreliable when there is severe vasoconstriction because of the reduced pulsatile component of the signal.
- It provides no indication of adequacy of ventilation: profound hypercapnia (increased  $\text{PaCO}_2$ ) is possible with normal oxygen saturations, particularly when using a high inspired oxygen concentration.
- It is unreliable with certain haemoglobins:
  - a. When carboxyhaemoglobin is present, it overestimates  $\text{SpO}_2$
  - b. When methaemoglobin is present,  $\text{SpO}_2$  is underestimated at values greater than 85%
- It progressively under-reads the arterial blood saturation as the haemoglobin decreases (but is not affected by polycythaemia).
- It is affected by extraneous light and unreliable when there is excessive movement of the patient.

### Advanced airway management

In many patients more advanced airway techniques will be required. The airway is not considered to be reliably secure unless a cuffed tube has been placed in the trachea. This is best achieved by orotracheal intubation (or in some circumstances by a surgical technique). To maximise the safety and efficacy of tracheal intubation it must be carried out by those who are trained and competent in the use of anaesthetic drugs. Details of the procedure are given in Appendix 3.1. The only exceptions to this are patients who are moribund or in cardiorespiratory arrest. If personnel skilled in the use of anaesthetic drugs are unavailable or it is impossible to intubate the patient, insertion of a supraglottic airway device (SAD) may enable effective oxygenation and will provide some airway protection temporarily.

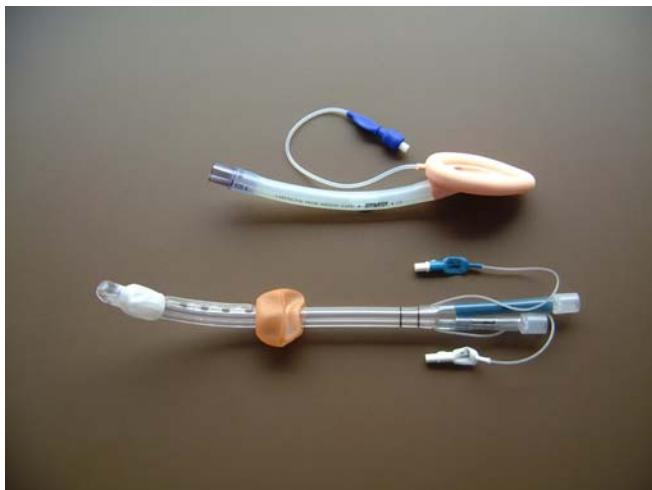
Throughout Europe, the practice of advanced airway management varies considerably both in the equipment used and application of the technique. Countries with physician-based systems will often use drugs-assisted tracheal intubation at an early stage in resuscitation, for example in the pre-hospital setting, whereas in other systems this will not occur until when the patient reaches the emergency department and an anaesthetist is in attendance. The ETC cannot therefore hope to cover all eventualities in this complex area, but supports the concept of competency-based training to allow individuals to follow local protocols in a safe and effective manner.

## **SUPRAGLOTTIC AIRWAY DEVICES**

If the patient's oxygenation ( $\text{SpO}_2$ ) continues to decrease despite optimal attempts at bag-mask ventilation (using a two-person technique and oropharyngeal and/or nasopharyngeal airways) an attempt should be made to insert a supraglottic airway device (SAD) e.g., laryngeal mask airway (LMA), Combitube or other device depending on local availability.

### *Laryngeal mask airway (LMA)*

The laryngeal mask airway consists of a wide-bore tube with an elliptical inflated cuff designed to seal around the laryngeal opening (Fig. 3.10). It was introduced into anaesthetic practice in the middle of the 1980s and has been shown to be a reliable and safe device, which can be introduced easily, with a high success rate after a short period of training. Ventilation using the LMA is more efficient and easier than with a bag-mask apparatus; provided high inflation pressures ( $>20 \text{ cm H}_2\text{O}$ ) are avoided, gastric inflation is minimised. When an LMA can be inserted without delay it is preferable to avoid bag-mask ventilation altogether: the risk of gastric inflation and regurgitation is reduced. Though not guaranteeing protection of the airway from gastric contents, pulmonary aspiration during use of the LMA is uncommon. The LMA does seem to protect against sources of aspiration (e.g., blood) from above the larynx. Furthermore, as the insertion of the LMA does not require extensive movement of the head and neck, it may be used in the presence of a suspected cervical spine injury. Use of the LMA by nursing, paramedical and medical staff during resuscitation has been studied and reported to be effective. The LMA is particularly valuable if attempted intubation by skilled personnel has failed and bag-mask ventilation is impossible (the 'cannot ventilate, cannot intubate' scenario). The conventional LMA (LMA Classic<sup>TM</sup>) can be reused up to 40 times after sterilisation. Single-use versions are now available and may be especially suitable for prehospital use. Some of the single-use LMAs are of a slightly different design and material to the LMA Classic<sup>TM</sup> and their performance may not be quite the same.



**Fig. 3.10: Laryngeal Mask Airway and Combitube**

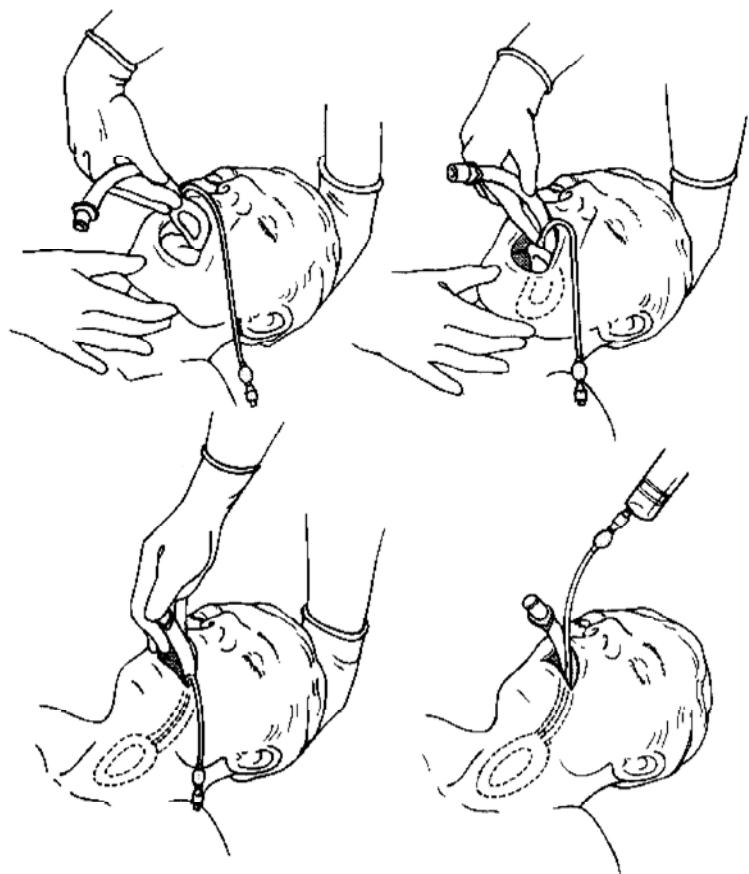
#### Technique for insertion of a laryngeal mask airway

- Select a LMA of an appropriate size for the patient and deflate the cuff fully. A size 5 will be correct for most men and a size 4 for most women. Lubricate the outer face of the cuff area (the part that will not be in contact with the larynx) with water-soluble gel.
- Maintain MILS unless there is no risk of cervical spine injury – in this case, flex the patient's neck slightly and extend the head.
- Release cricoid pressure if it is being applied.
- Holding the LMA like a pen, insert it into the mouth (Fig. 3.11). Advance the tip behind the upper incisors with the upper surface applied to the palate until it reaches the posterior pharyngeal wall. Press the mask backwards and downwards around the corner of the pharynx until a resistance is felt as it locates in the back of the pharynx. If possible, get an assistant to apply a jaw thrust after the LMA has been inserted into the mouth - this increases the space in the posterior pharynx and makes successful placement easier.
- Connect the inflating syringe and inflate the cuff with air (40 ml for a size 5 LMA and 30 ml for a size 4 LMA); alternatively, inflate the cuff to a pressure of 60 cmH<sub>2</sub>O. If insertion is satisfactory, the tube will lift one to two centimetres out of the mouth as the cuff finds its correct position and the larynx is pushed forward.
- If the LMA has not been inserted successfully after 30 seconds, oxygenate the patient using a pocket mask or bag-mask before reattempting LMA insertion.
- Confirm a clear airway by listening over the chest during inflation and observing bilateral chest movement. A large, audible leak suggests malposition of the LMA, but a small leak is acceptable provided chest rise is adequate.
- Insert a bite block alongside the tube if available and secure the LMA with a bandage or tape.

#### *Limitations of the LMA*

- It is not possible to insert an LMA correctly in the presence of cricoid pressure – this prevents the tip of the LMA seating correctly in the upper oesophagus.
- In the presence of high airway resistance or poor lung compliance (pulmonary oedema, bronchospasm, COPD) there is a risk of a significant leak around the cuff causing hypoventilation. Most of the gas leaking around the cuff normally escapes through the patient's mouth but some gastric inflation may occur.
- There is a theoretical risk of aspiration of stomach contents because the LMA does not sit within the larynx like a tracheal tube; however, this complication has not been documented widely in clinical practice.
- If the patient is not deeply unconscious, insertion of the LMA may cause coughing, straining or laryngeal spasm.
- If an adequate airway is not achieved, withdraw the LMA, deflate the cuff and attempt reinsertion.
- Uncommonly, airway obstruction may be caused by the epiglottis folding down to cover the laryngeal inlet. Withdraw the LMA, deflate the cuff and attempt reinsertion.

**Fig. 3.11: Insertion of the LMA**



### The ProSeal LMA

The ProSeal LMA (PLMA) is a modified version of the original LMA. It has an additional posterior cuff and a gastric drain tube. The device has been studied extensively in anaesthetised patients, but there are no studies of its function and performance during resuscitation of trauma patients. It has several attributes that, in theory, make it more suitable than the original LMA for use in trauma patients: improved seal with the larynx enabling ventilation at higher airway pressures (commonly up to 35 – 40 cm H<sub>2</sub>O), the inclusion of a gastric drain tube enabling venting of liquid regurgitated gastric contents from the upper oesophagus and passage of a gastric tube to drain liquid gastric contents, and the inclusion of a bite block. Potential weaknesses of the PLMA as an airway device in the emergency setting are that it is slightly more difficult to insert than the original LMA, it is not available in disposable form at present, it is relatively expensive, and that solid regurgitated gastric contents could block the gastric drainage tube.

### The Combitube

The Combitube (Fig. 3.10) is a double lumen tube introduced blindly over the tongue and into the pharynx. It is designed to provide a route for ventilation whether the tube has passed into the trachea or the oesophagus. The tracheal channel has an open distal end. The oesophageal tube has no terminal opening, but has several small side-holes located between two cuffs. There is a small distal cuff and a large proximal cuff designed to inflate within the pharynx.

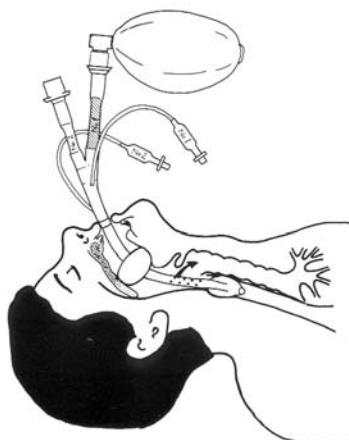
When introduced blindly, the tube usually enters the oesophagus (in 95% of cases), and the patient's lungs are ventilated through the oesophageal channel via the side holes, which are located at or above the larynx (Fig. 3.12a). Gas cannot pass down the oesophagus because of the blind end of the oesophageal channel, and the distal cuff, which is positioned just proximal to the blind end. The pharyngeal cuff prevents air leaking from the mouth. If the tube enters the trachea, ventilation is achieved via the tracheal port through its open distal end (Fig. 3.12b). The Combitube is available in 2 sizes, 37 FG (small adult) and 41 FG – the small version is adequate for most patients.

#### *Advantages of the Combitube*

- It can be inserted with the neck maintained in a neutral position
- Ventilation is as effective as via a tracheal tube
- It prevents aspiration of gastric contents whether in the oesophagus or trachea
- It protects from soiling from above the larynx
- The Combitube functions effectively in the presence of a cervical collar

- It allows generation of relatively higher airway pressures compared to a standard LMA
- It has been successfully used in a variety of difficult trauma scenarios

**Fig. 3.12a; Oesophageal placement of the Combitube**



**Fig. 3.12b; Tracheal placement of the Combitube**



#### Technique for insertion of the Combitube

- Deflate the cuffs fully and lubricate the distal part of the Combitube.
- Bend the device into a 'hockey-stick' shape.
- Maintain the patient's head and neck in a neutral position using MILS; open the mouth and lift the jaw.
- Insert the device taking care to remain in the midline and advance until the teeth or gums are located between the two black lines.
- Inflate the large (No 1) or pharyngeal cuff with air (approximately  $1 \text{ ml kg}^{-1}$ ) and the small (No 2) cuff with 10-15 ml air.
- Attempt and confirm ventilation via the longer or No 1 lumen and appropriate checks made to confirm ventilation (Fig. 3.12a)
- If the lungs are not ventilated, ventilation is attempted via the shorter (No 2) lumen and appropriate checks made (Fig. 3.12b)
- Once correctly inserted and ventilation is confirmed, fixation is not essential

#### *Limitations of the Combitube*

- Difficulty in acquiring training in insertion, as it is not in daily use.
- Difficult insertion where there is restricted mouth opening.

- Risk of oesophageal perforation in the presence of known or suspected oesophageal pathology.
- Pharyngeal or laryngeal trauma on insertion, usually caused by incorrect technique or over-inflation of the pharyngeal balloon.
- Inability to ventilate via either lumen, usually because inserted too far and pharyngeal cuff occludes the larynx.

### Laryngeal Tube

The Laryngeal Tube (LT) is one of many new supraglottic airway devices that have been developed. It is a single-lumen tube with both an oesophageal and pharyngeal cuff. A single pilot balloon inflates both cuffs simultaneously and it is available in a variety of sizes. Successful insertion and airway pressures generated are comparable to the LMA.

### Surgical airway

In some cases, both intubation and ventilation using any of the techniques described above may be impossible. This may be caused by complete obstruction of the airway from oedema (e.g., inhalation burns) or trauma (e.g., laryngeal injury). Most of these patients will arrive in the emergency department unconscious, either as a consequence of their initial injuries or from the resulting hypoxia. In these situations a surgical airway is required urgently. The conscious patient with an obstructed airway will be very agitated. Where there is profuse bleeding the patient may wish to sit forward to stop the blood pooling in the pharynx and to enable unstable facial fractures to fall forward and partially clear the airway. Do not give sedatives or neuromuscular blocking drugs without the presence of someone very experienced in advanced airway management. If intubation is impossible, a surgical airway must be established immediately. Whichever technique is used to create a surgical airway, check the equipment before starting, clear the airway as much as possible and give high-flow oxygen at all times - it is unlikely that there is total airway obstruction. The final choice of surgical airway will depend upon the clinical situation, practitioner skills and experience. The options are; needle cricothyroidotomy, surgical cricothyroidotomy or tracheostomy.

#### **Needle cricothyroidotomy**

Needle cricothyroidotomy is a temporary procedure providing only short-term oxygenation, enabling plans to be made for a more formal airway intervention to be completed. It does not prevent aspiration. Oxygenation is achieved using a high-pressure supply (e.g., Sander's injector, Manujet<sup>TM</sup>, or self-assembled tubing connected to a 400 kPa oxygen source) to deliver oxygen (see Fig. 3.13). A self-inflating bag will not generate sufficient pressure. Whatever system is used it should be readily available rather than constructed in haste in an emergency. Expiration occurs through the larynx, not the cannula. Ensure that gas is escaping via this route otherwise the lungs will be over-inflated causing serious

barotrauma. To prevent disconnection by the high-pressure gas supply, make sure that all connections between the supply and the cannula have Luer locks or are bonded. It is often recommended after failed intubation in a child of less than 12 years, where the risk of cricoid cartilage damage contraindicates surgical cricothyroidotomy. It is unsuitable for use during patient transfer.



**Fig. 3.13: Manujet and cannula for cricothyroidotomy.**

Inset: Commercially available cricothyroidotomy needle.

#### Procedure

- Identify the cricothyroid membrane.
- If time permits, prepare the skin with antiseptic solution.
- Attach a 10 ml syringe to a cannula or, preferably, a purpose-made cricothyroidotomy cannula.
- While stabilising the thyroid cartilage with one hand, insert the cannula through the skin and cricothyroid membrane in a slightly caudal direction while aspirating on the syringe (Fig. 3.14).
- Free flow of air into the syringe indicates that the tip of the needle has entered the trachea.
- Advance the cannula a further 5 mm to ensure the body of the cannula has entered the trachea; continue aspirating on the syringe.
- Advance the cannula over the needle into the trachea; remove the needle and confirm free aspiration of air through the cannula.
- While holding the cannula, connect the ventilation system and commence ventilation.
- One second of oxygen supplied at a pressure of 400 kPa and flow of  $15 \text{ l min}^{-1}$  should be sufficient to inflate adult lungs adequately. Allow a four second pause to enable expiration via the upper airway (expiration does not occur via the cannula). In children, the initial

oxygen flow rate in  $\text{l min}^{-1}$  should equal the child's age in years, and this is increased in 1  $\text{l min}^{-1}$  increments until one second of oxygen flow causes the chest to rise.

- Observe carefully for adequate exhalation through the upper airway. This usually occurs without difficulty, but it is essential to ensure that the chest falls adequately after each ventilation.
- If ventilation fails or complications develop then proceed immediately to surgical cricothyroidotomy.

Complications include:

- barotrauma and pneumothorax;
- haemorrhage, oesophageal damage and surgical emphysema;
- kinking or obstruction of the cannula and failure of oxygenation.
- ineffective in patients with severe chest trauma



**Fig 3.14: Needle cricothyroidotomy**

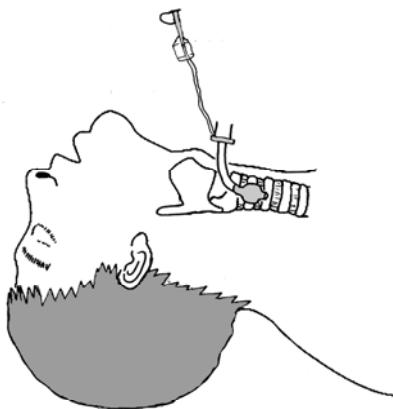
#### Surgical cricothyroidotomy

An incision is made through the skin and cricothyroid membrane into the glottis to enable the insertion of a small tracheal tube (6-7 mm diameter) or cuffed tracheostomy tube into the trachea (Fig. 3.15). A tracheal tube is preferred to a tracheostomy tube because the cuff of a small tracheostomy tube is often too small to occlude an adult trachea. Although a relatively small tube is used, it is sufficient to enable oxygenation and ventilation using standard devices and suction of secretions. The technique is associated with significant haemorrhage from vessels dilated by hypercarbia and hypoxia. If there is laryngeal trauma or an expanding haematoma within the operating field, formal tracheostomy will be required.

#### Procedure

- In the absence of trauma to the cervical spine, extend the patient's head.
- Identify the cricothyroid membrane.
- If time permits, prepare the skin with antiseptic solution and, if appropriate, infiltrate the skin over the cricothyroid membrane with 1% lignocaine with adrenaline (1:100,000).
- Stabilises the thyroid cartilage with one hand and make 2-3 cm transverse incision through the skin.
- Identify the membrane and incise it transversely.
- With the scalpel blade in situ insert a pair of tracheal dilators and open up the incision.
- Remove the scalpel and insert the well-lubricated tracheal tube or tracheostomy between the tracheal dilators.
- Remove the tracheal dilators, inflate the tube cuff and attempt ventilation of the lungs.
- Confirm the correct position by checking for exhaled CO<sub>2</sub> and observing chest movement; listen for breath sounds.
- Secure the tube and suck any secretions from the lungs.

**Fig. 3.15: Surgical cricothyroidotomy**



Take care not to damage the posterior tracheal wall by deep penetration with the scalpel blade. Other complications include haemorrhage and insertion of the tube outside the trachea causing surgical emphysema. If the incision is too small to admit the tube the incision can be enlarged laterally while being held open vertically with the tracheal dilator. Ideally, once a passage is made into the trachea it should be occupied by instruments until a tube is inserted - this minimises bleeding and maintains patency of the passage.

## **TRACHEOSTOMY**

A surgical tracheostomy is rarely indicated as a primary method of securing the airway. It cannot be undertaken safely without training. Percutaneous tracheostomy can be used in emergencies, but only by individuals experienced in the single-stage dilatational approach.

Once the airway is secure and ventilation established, obtain a chest x-ray and measure arterial blood gas values. Small tubes are easily dislodged by the weight of attached ventilator tubes or movement of the patient. If there is any deterioration in the patient's condition, reassess the airway and ventilation rapidly.

### **Summary**

In every trauma patient, the airway must be cleared, secured, and the cervical spine stabilised, as soon as possible. The maintenance of oxygenation and ventilation are the ultimate priorities. Initially, basic techniques are used; more advanced procedures are required only if basic measures are inadequate. When resuscitating the seriously-injured patient, airway management should be undertaken by suitably-trained and experienced individuals.

## **Appendix 3.1: Tracheal intubation**

Indications for intubating the trachea of a trauma patient include:

- apnoea;
- obstructed or partially obstructed airway unrelieved with basic airway techniques;
- the need for invasive respiratory support to treat hypoxaemia and/or ventilatory failure;
- despite effective basic airway care, the predicted clinical course includes high probability of airway obstruction, aspiration or ventilatory failure, e.g., depressed conscious level.

Determine the urgency for intubation:

- immediate - the patient is deteriorating rapidly and definitive airway care is required with a minimum of delay;
- urgent - basic techniques can maintain the physiology of the patient for a short period, pending intubation;
- observant - no indication for intubation currently exists, and the patient can be observed closely for any deterioration.

The success of supplemental oxygen and basic airway manoeuvres is critical in determining the need for and the urgency of the intubation. A comatose patient with airway obstruction but no anatomical abnormalities can usually be oxygenated for a short period using basic techniques with or without a bag-mask device. A comatose patient with facial injuries or vomit in the airway who cannot be oxygenated adequately using basic techniques requires immediate intubation to avoid severe hypoxia. Undertake an early, rapid assessment of the likely technical difficulty of airway management. If management of the patient's airway is likely to be difficult (Box 3.2) use of anaesthetic and neuromuscular blocking drugs is potentially dangerous. A difficult airway can be categorised as:

- Difficult mask ventilation
- Difficult view at laryngoscopy
- Difficult intubation
- Difficult cricothyroidotomy

A high risk of failed intubation must be balanced against the assessed urgency for the intervention.

**Box 3.2: Typical conditions predisposing to difficult intubation**

## Pre-existing conditions

- Pregnancy
- Rheumatoid arthritis
- Ankylosing spondylitis
- Acromegaly

## Anatomical abnormalities

- Obesity
- Bull neck
- Prominent dentition
- Poor dentition

## Obvious trauma

- Maxillofacial
- Neck
- Larynx

## Airway obstruction

Patients may need ‘immediate’ intubation because of anatomical disruption of their airway or because of aspiration of blood or vomit, e.g., facial or laryngeal trauma. These situations are often complicated by reduced consciousness because of hypoxia from the obstructed airway or because of head injury. These patients need prompt intubation to prevent hypoxic brain injury, but can be technically challenging to intubate. Call for senior assistance as soon as these patients are identified or when there is warning of their arrival.

***Facial trauma***

Disruption to normal facial anatomy may mean that intubation is the only way to relieve airway obstruction. This is particularly true of midface fractures, where the upper airway is compromised by the displaced bony segment, and of complex jaw fractures, where the tongue loses its normal support and prolapses into the airway. The accompanying severe haemorrhage may also obstruct the airway and packing to stop the bleeding may further compromise the airway. Patients with complex facial injuries often also have brain injuries, rendering them combative or comatose with impaired protective airway reflexes.

***Laryngeal disruption/swelling***

Laryngeal injury may make the airway impossible to maintain. Blunt trauma to the front of the neck may fracture the larynx and cause stridor, crepitus, and surgical emphysema. An expanding haematoma caused by penetrating neck injury will compress the airway, causing

stridor, hoarseness and respiratory distress. Thermal injury can cause serious swelling of the larynx or epiglottis.

In all these scenarios, the airway is compromised and total obstruction is likely to occur quickly. Distortion of normal anatomy can make intubation extremely difficult, or impossible. Furthermore, a rescue surgical airway via cricothyroidotomy may also be technically difficult. Summon assistance immediately from someone experienced in airway management, along with a surgeon capable of doing an emergency tracheostomy. Despite the risks, if the patient becomes hypoxic, intubation must be performed urgently by the most experienced person present. If enough airway patency can be achieved to enable adequate oxygenation, defer airway intervention until senior anaesthetic assistance arrives.

### ***Impaired consciousness with agitation***

Frequently, patients with a Glasgow Coma Scale (GCS) score of 9-12 need tracheal intubation even if there is no airway obstruction and no ventilatory failure. Obtunded, agitated patients can be exceptionally difficult to manage without anaesthesia. This is because procedures such as CT scanning become impossible, and inserting intravascular cannulas and tubes hazardous; furthermore, many of these patients will deteriorate and are at risk of developing airway obstruction, hypoventilation and aspiration. Intubating an uncooperative patient is challenging: inadequate pre-oxygenation, loss of venous access and sub-optimal positioning will contribute to intubation difficulty.

### **The technique of tracheal intubation**

In the emergency situation, in a patient with a full-stomach, the sequence of events of preoxygenation, application of cricoid pressure and use of a hypnotic and the neuromuscular blocking drug succinylcholine (suxamethonium) by anaesthetists to allow intubation of the trachea is often referred to as “Rapid Sequence Induction”, or RSI. The aim is to keep the time from loss of consciousness to securing the airway to a minimum. To prevent inflation of the stomach and regurgitation of gastric contents, the lungs are not usually ventilated between loss of consciousness and intubation. Emergency physicians use a similar sequence of events. However, in patients in whom preoxygenation is ineffective, they manually ventilate the patient before giving the drugs and while waiting for the succinylcholine to take effect. They refer to this as “Rapid Sequence Intubation” or RSI. The situation is further complicated by the recent introduction of the term “modified-RSI”. This usually implies either giving potent analgesic intravenously along with the other drugs to attenuate the sympathetic response during intubation (for example in a patient with a head injury), or substituting a large dose of a non-depolarising neuromuscular blocking drug (for example rocuronium) for succinylcholine, to avoid potential unwanted side effects

(hyperkalaemia, raised intraocular pressure). In order to avoid confusion, none of these terms will be used, instead the process will be referred to as “Emergency Drug-assisted Intubation”.

### ***Preparation of equipment***

Whenever possible, check the equipment thoroughly before attempting emergency drug-assisted intubation (Box 3.3).

### **Box 3.3: Equipment for emergency drug-assisted intubation**

#### **Basic resuscitation equipment**

- Tilting trolley / stretcher
- Oxygen delivery apparatus including mask with reservoir and oxygen tubing
- Sucker
- Airway adjuncts:
  - Nasopharyngeal airway (sizes 6 and 7)
  - Oropharyngeal airway (sizes 2, 3 and 4)
- IV access equipment
- Monitors

#### **Advanced airway equipment**

- Bag-mask apparatus with reservoir bag and oxygen tubing
- Drugs – in labelled syringes
- Laryngoscope handles & blades (sizes 3 and 4 for adults)
- Magill's forceps
- Intubating bougie and/or stylet
- Water-soluble jelly
- Tracheal tubes in a range of sizes
- 10 ml syringe
- Tie and adhesive tape
- Equipment for exhaled CO<sub>2</sub> detection
- Ventilator

#### **Failed intubation equipment**

- LMA/ProSeal LMA/Combitube/Laryngeal tube
- Surgical cricothyroidotomy set
- Needle cricothyroidotomy kit with high-pressure injector

- Trolley - check to ensure that the height adjustment and mechanism for rapid tilting are working properly. Position it optimally to ensure access to the patient for intubation, effortless view of monitoring and easy reach of the anaesthetic equipment.
- *Suction – place a rigid sucker close to the patient's head.*
- Oxygen delivery apparatus – make sure that the oxygen delivery system is patent and capable of delivering positive pressure for ventilation. Connect a heat and moisture

exchanger (HME) filter to the breathing system: it prevents contamination of ventilation equipment and helps to warm and humidify the oxygen-enriched air in the trachea. Check that all connections are hand tight.

- Laryngoscopes - the curved Macintosh laryngoscope blade is commonly available in sizes 3 (short) and 4 (long); use the size 4 blade in all but the smallest adult patients. Check that the laryngoscope light is bright before starting a emergency drug-assisted intubation. Alternative laryngoscope blades such as the McCoy blade (which enables further elevation of the epiglottis using the lever on the tip of the curved blade) or the Magill straight-bladed laryngoscope are useful alternatives.
- Tracheal tubes – use a 7.0mm tracheal tube for women and 8.0 mm for men; however, a range of tracheal tube sizes should be available. While tubes are often cut to length (22-24 cm for women, 24- 26 cm for men), if facial swelling is likely for example with burns or blunt facial trauma, leave the tube uncut.
- Bougies and stylets - if the view at laryngoscopy is less than perfect, an intubating bougie can be inserted behind the epiglottis and into the trachea, and a tracheal tube then ‘railroaded’ into position over the bougie (see below). Some practitioners prefer to use an intubating stylet: the rigidity of this device enables the tube to be shaped to bring the tip more anterior, forming a ‘J’ shape to facilitate intubation (see below).
- Equipment for failed intubation - check the equipment for failed intubation and place it in an easily accessible location during all intubations.
- Drugs - the choice of drugs for induction of anaesthesia and maintenance of sedation and analgesia are described below. Once the drugs are selected, prepare them in clearly labelled syringes. This preparation must include the drugs that may be required for treatment of any hypotension associated with the drugs used to facilitate intubation.

## **Monitoring**

Attach the following monitors as soon as possible:

- Pulse oximetry
- Non-invasive blood pressure
- Continuous ECG
- Inspired oxygen concentration ( $\text{FIO}_2$ )
- Capnometry

## **Checks**

- Review the ABCs
- Review a brief history using the AMPLE approach (Box 3.4)
- Ensure that there are two large-bore intravenous cannulae in situ before giving induction drugs

- Undertake a brief neurological examination before induction of anaesthesia. This includes an assessment of the GCS, pupil signs and presence of abnormal posturing. Look for the presence of diaphragmatic breathing, vasodilation, or priapism.

#### **Box 3.4: AMPLE History**

- Allergies
- Medications
- PMHx (past medical history)
- Last
  - Anaesthetic (complications)
  - Meal
  - Tetanus
- Events
  - *Leading up to this situation*

### **Positioning of the patient**

Alignment of the oral, pharyngeal and laryngeal axes creates a clear view from the incisors to the laryngeal inlet and maximises the view obtained at laryngoscopy. This view is achieved normally by flexing the neck and extending the head at the atlanto-axial junction. This is not possible in most trauma patients because the cervical spine must be immobilised using MILS.

### ***The procedure***

#### **Pre-oxygenation**

Pre-oxygenation replaces the nitrogen in the alveoli with oxygen and maximises the time before desaturation occurs following the onset of apnoea. This provides more time for intubation to be attempted before having to stop to re-oxygenate the patient's lungs. Whenever possible, give 100% oxygen for three minutes before induction of anaesthesia. A patient who is breathing inadequately may not achieve enough alveolar ventilation to replace nitrogen in the lung with oxygen - these patients may require assisted ventilation to achieve adequate pre-oxygenation before emergency drug-assisted intubation.

The time to desaturation is related not only to the effectiveness of pre-oxygenation, but also the age and weight of the patient and their physiological status. In a healthy adult, after effective pre-oxygenation the time taken for arterial blood to desaturate to 90% may be as long as 8 minutes. Arterial blood will desaturate much faster in critically ill, obese, or elderly patients, or those with respiratory disease. The decline in oxygen saturation is most rapid

below 92%: once the pulse oximeter indicates a SpO<sub>2</sub> of 92% or less, ventilate the patient's lungs immediately with 100% oxygen.

Although it is not conventional to provide assisted ventilation during RSI as used by anaesthetists until tracheal intubation has been achieved, if the trauma patient requires assisted ventilation before induction it should be continued after the induction drugs have been given. Properly applied cricoid pressure should prevent gas passing into the stomach. Patients with limited respiratory reserve, including those with severe chest injuries, will require assisted ventilation while waiting for complete neuromuscular blockade.

### **Manual in-line stabilisation (MILS)**

All patients with significant blunt trauma are assumed to have an unstable cervical spine injury until investigations and reliable clinical examination have ruled out this possibility. For this reason, all airway interventions are undertaken with the cervical spine stabilised. An assistant kneels at the head of the patient and to one side to leave room for the person intubating. The assistant holds the patient's head firmly down on the trolley by grasping the mastoid processes (Fig.3.16); the tape, lateral blocks, and front of the collar are removed. The front of a single-piece collar can be folded under the patient's shoulder leaving the posterior portion of the collar in situ behind the head. Do not attempt laryngoscopy and intubation with the collar in place – it will make it very difficult to get an adequate view of the larynx. Although MILS can be provided from the front of the patient, it may interfere with the application of cricoid pressure and, if required, achievement of a surgical airway.



**Fig. 3.16: Emergency drug-assisted intubation in the ED using MILS.** Note 4 people are required to perform this technique safely.

## **Injection of drugs**

The induction drug produces unconsciousness, and is followed immediately by a neuromuscular blocking drug, using a pre-calculated dose. Both drugs are injected rapidly into a functioning intravenous line with an infusion running to expedite drug delivery.

## **Cricoid pressure**

A trained assistant applies cricoid pressure as the induction drug is injected and consciousness lost. The cricoid ring is identified below the thyroid cartilage and cricothyroid membrane, and direct firm pressure using two or three fingers is applied, pressing backwards to compress the upper oesophagus between the cricoid ring and the cervical vertebra posteriorly, preventing passive reflux of gastric contents into the pharynx (Fig. 3.17). The correct pressure is 30 - 40 N, which is enough to be painful in the conscious patient. Inadequate pressure will not occlude the oesophagus; however, excessive force or incorrect placement will deform the larynx and make laryngoscopy and intubation more difficult. Do not apply cricoid pressure if there is active vomiting because it may cause oesophageal rupture. There is no evidence that a second hand applied behind the neck (two-handed cricoid pressure) in an attempt to restrict cervical spine movement is any safer than the standard technique. The cricoid pressure is removed only on the instruction of the intubating clinician once correct tube placement has been confirmed.



**Fig. 3.17: Cricoid pressure**

### **Standard intubation technique**

- Hold the laryngoscope in the **left** hand.
- Open the mouth using the index and thumb of the right hand in a scissor action.
- Insert the blade of the laryngoscope along the right side of the tongue, displacing it to the left.
- Advance the tip of the laryngoscope into the gap between the base of the tongue and the epiglottis (vallecula).
- Apply force in the direction the handle is pointing (Fig. 3.18), thereby lifting the tongue and epiglottis to expose the larynx.
- There should be **no** wrist movement, all the force comes from the upper arm.
- Use rigid suction to clear any secretions, blood or vomit before attempting to insert the tracheal tube.
- Advance the tube from the right hand corner of the mouth through the cords.
- Withdraw the laryngoscope taking care not to dislodge the tube.
- Inflate the cuff and attempt to ventilate the lungs.
- Confirm the position of the tube and secure it using tape or a tie.



**Fig. 3.18: Laryngoscopy in an elective procedure.** Note tip of epiglottis just visible. Assistants finger retracting patient's cheek to improve the view.

Use of MILS makes it more difficult to get a good view of the larynx and use of an intubating bougie or an intubating stylet is invaluable. For this reason, some practitioners prefer to use an intubating bougie or stylet routinely when intubating trauma patients.

### **Technique for using an intubating bougie (Fig. 3.19)**

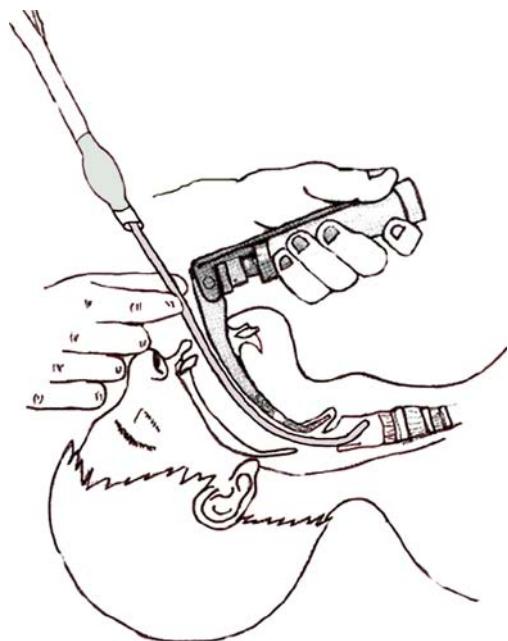
- With the laryngoscope in place, pass the 'hockey-stick' or coudé-tipped bougie behind the epiglottis and into the trachea.
- Detection of 'clicks' as the bougie slides over the tracheal rings helps to confirm correct placement: hold-up of the bougie in the distal airways provides secondary confirmation of placement.
- While maintaining the best possible view of the larynx, 'railroad' the tracheal tube over the bougie into the trachea.
- Rotate the tracheal tube 90° counter-clockwise to ease passage through the cords.
- While grasping the tracheal tube firmly, the assistant removes the bougie.
- Confirm correct placement of the tracheal tube using the methods described below.

### **Technique for using an intubating stylet**

- Insert the introducer into the tracheal tube ensuring that the tip does not protrude beyond the distal end.
- Advance the tracheal tube until the black line just proximal to the tube cuff lies at or just beyond the vocal cords. Sometimes rotation of the tube through 45 degrees as it is inserted into the mouth can improve the view: this enables the tube tip to be followed without the view being obscured by the proximal end of the tube.

The following interventions may also help to improve the view of the larynx:

- Use a long-blade laryngoscope – routine use of a size 4 blade is sensible.
- The McCoy laryngoscope is very useful for difficult intubations – it has a hinged tip that is elevated using a lever on the handle (Fig. 3.19). This enables the epiglottis to be lifted and improves the view in many cases.
- Backward, upward and rightward pressure (the BURP manoeuvre) on the larynx by an assistant may improve the view.
- Incorrect application of cricoid pressure may obscure the view by distorting the larynx – ask the assistant ease the cricoid pressure slightly to see if this improves the view.



**Fig. 3.19: Use of a bougie and McCoy laryngoscope to facilitate tracheal intubation**

#### ***Confirmation of tracheal tube placement***

- Detection of carbon dioxide in exhaled gas (capnometer or colormetric carbon dioxide detector) is considered to be the gold standard for confirming that the tracheal tube is in a major airway; however, it will not discriminate a tube placed in a main bronchus from one placed in the trachea.
- Look for symmetrical movement of the chest wall with ventilation.
- Listen in both axillae for breath sounds and over the stomach for absence of sounds.
- Oesophageal detector device – this creates a suction force at the tracheal end of the tracheal tube, either from pulling back the plunger of a large syringe or releasing a compressed flexible bulb. Air is aspirated easily from the lower airways through a tracheal tube placed in the cartilage-supported rigid trachea. When the tube is in the

oesophagus, air cannot be aspirated because the oesophagus collapses when aspiration is attempted. The oesophageal detector device is generally reliable in patients with both a perfusing and a non-perfusing rhythm but it may be misleading in patients with morbid obesity, late pregnancy, severe asthma or when there are copious tracheal secretions; in these conditions the trachea may collapse when aspiration is attempted.

Once tracheal intubation is confirmed, ask the assistant to release the cricoid pressure and secure the tube with tape or a ribbon tie. In a patient with raised intracranial pressure use of adhesive tape instead of a tie will avoid compression of the jugular veins, which may increase intracranial pressure. Insertion of an oropharyngeal airway next to the tracheal tube reduces the risk of the patient biting on the tube and occluding the airway. Check the monitors for heart rate, arterial oxygen saturation, blood pressure, and end-tidal CO<sub>2</sub>.

### ***Complications***

Emergency drug-assisted intubation may be accompanied by hypotension, hypoxaemia, and airway trauma.

#### *Causes of hypotension:*

- Vasodilation and myocardial depression caused by the induction drug – this will be compounded greatly by hypovolaemia. Give fluid 500-1000 ml rapidly and reassess the blood pressure. A vasoconstrictor (vasopressor), such as ephedrine (3-6mg) or adrenaline (10-20micrograms), if the hypotension does not respond immediately to fluid. When there is any suspicion of hypovolaemia before induction of anaesthesia give 500-1000 ml fluid rapidly before and during the pre-oxygenation phase: have vasopressor drugs ready to give immediately and, whenever possible, monitor blood pressure continuously using an arterial line
- Hyperventilation with air trapping, especially if the patient has chronic obstructive pulmonary disease. If this is suspected, disconnect the breathing system from the tracheal tube and allow adequate time for exhalation of trapped alveolar gas. Continue positive pressure ventilation with a reduced rate and longer expiratory time.
- Tension pneumothorax – treat with immediate needle decompression followed by insertion of a chest drain

#### *Hypoxaemia*

During and after attempted intubation hypoxaemia may be caused by a prolonged attempt without pausing for re-oxygenating the patient's lungs, unrecognised oesophageal intubation, intubation of a main bronchus (usually the right), and failed intubation.

## **Trauma**

During an intubation the lips, teeth, tongue, mandible, pharynx or cervical spine can be injured.

## **Ventilation**

Following successful intubation, ventilate the patient's lungs with high-concentration oxygen:

1. Use a self-inflating bag with a reservoir and an oxygen flow of  $15 \text{ l min}^{-1}$ 
  - a. Ventilate the lungs to produce visible chest rise at a rate of 12-15 breaths  $\text{min}^{-1}$ .
2. Use of a mechanical ventilator will free-up the practitioner to undertake other tasks and will provide a constant minute ventilation.
  - a. Set a tidal volume of 6-8 ml/kg at a rate of 12 breaths  $\text{min}^{-1}$ .
  - b. Set the pressure limit initially to 30 cmH<sub>2</sub>O.
  - c. Set the FiO<sub>2</sub> to 1.0 (100%, no air mix).
  - d. Switch on and attach to the tracheal tube.

The final determinant of the adequacy of ventilation is analysis of an arterial blood sample. A chest x-ray will also be required to confirm the position of the tracheal tube and to enable identification of chest injuries. Insert a large-bore gastric tube to empty the stomach - do not use the nasal route if a base of skull fracture is suspected.

## **Suction**

If the airway is soiled by blood or vomit, use suction to clear it but precede this with a period of pre-oxygenation. Insert into the tracheal tube a flexible, sterile catheter, less than half the diameter of the tracheal tube. Once in place, apply suction intermittently as the catheter is withdrawn slowly, taking no longer than 30 seconds. Once the suction catheter has been removed, ventilate the patient's lungs for at least one minute before repeating the procedure and monitor the ECG and arterial oxygen saturation throughout.

Suction may cause:

- hypoxia from a decrease in lung volume, particularly if suction is prolonged;
- arrhythmias from sympathetic and vagal stimulation;
- hypertension and an increase in intracranial pressure.

## **Drugs used to facilitate advance airway management**

The reflexes in the larynx must be abolished to facilitate intubation. In some severely-injured patients the airway reflexes are so depressed that the trachea can be intubated without drugs - these patients have a very high mortality rate. In most cases, drugs are needed to

abolish the airway reflexes and ensure the patient is unconscious; this is normally achieved with a combination of an anaesthetic drug followed by a neuromuscular blocking drug ('muscle relaxant'). Further doses or infusions are then given to maintain unconsciousness and enable positive pressure ventilation of the lungs. The other most commonly used drugs are analgesics, usually opioids. Anaesthetic drugs are also given to modify the cardiovascular response to intubation, for example, in the head-injured patient when such responses may increase intracranial pressure (ICP). Anyone using anaesthetic and neuromuscular blocking drugs must be trained in their use and competent to deal with any complications that arise.

Anaesthetic and neuromuscular blocking drugs will render the patient apnoeic and totally dependent on the airway nurse and doctor to secure the airway and maintain oxygenation and ventilation. If intubation is difficult or impossible and it is not possible to ventilate the lungs ('can't intubate, can't ventilate'), it will be necessary to establish a surgical airway under poor and hurried conditions. The ultimate disaster occurs when, under these circumstances, the cricothyroid membrane is inaccessible. Under some circumstances, if intubation is predicted to be very difficult, fibreoptic intubation (requires a cooperative patient and a bloodless field) or establishing a surgical airway (cricothyroidotomy or tracheostomy) with the patient awake are options. Alternatively, a needle can be inserted through the cricothyroid membrane and its location confirmed before giving induction drugs – in this way, oxygenation will be possible even if the intubation attempt fails.

## **Pharmacology**

### ***Drugs used for induction (hypnotics)***

The drugs used to induce loss of consciousness (hypnosis) are generally short-acting intravenous anaesthetics. These depress myocardial contractility and cause vasodilation or both, resulting in hypotension. This effect is exaggerated in hypovolaemic trauma patients.

#### **Propofol**

- Presentation: 20 ml ampoules,  $10 \text{ mg ml}^{-1}$  (1%). (50 ml syringes of 1% and 2% for infusion)
- Dose:  $0.15\text{-}0.3 \text{ mg kg}^{-1}$  but may be dramatically reduced in the injured
- Loss of consciousness after 40-60 seconds, duration 3-4 minutes
- Significant potential for causing hypotension, requires prior correction of any hypovolaemia
- Reduces cerebral blood flow, helping control ICP
- May cause pain on injection and involuntary muscle movement

## Thiopental Sodium

- Presentation: powder, reconstituted with water to yield a 2.5% solution (500 mg in 20 ml)
- Dose: 3-5 mg kg<sup>-1</sup> IV; 1.5-2 mg kg<sup>-1</sup> IV in unstable patients
- Loss of consciousness after 5-15 seconds onset, duration 5-15 minutes
- Neuro-inhibition (at barbiturate receptor as part of GABA –receptor complex)
- Cerebroprotective - because of dose dependant decrease in:
  - Cerebral metabolic oxygen consumption
  - Cerebral blood flow
  - ICP
- Maintenance of cerebral perfusion pressure
- Venodilation
- Myocardial depression
- Central respiratory depression

## Ketamine

- Presentation: 10 mg ml<sup>-1</sup> - 10 ml and 20 ml vials; 100 mg ml<sup>-1</sup> - 10 ml vial
- Dose: 1-2 mg kg<sup>-1</sup> IV; 5 mg kg<sup>-1</sup> IM
- Loss of consciousness after 15-30 seconds when given IV
- Excitatory phenomena
- Profound analgesia
- Sedation
- Dissociative state
- Amnesia (less than benzodiazepines)
- Central sympathetic stimulation leading to:
  - Increased heart rate
  - Increased blood pressure
- Increased cerebral cortical activity
- Bronchial smooth muscle relaxation
- Myocardial depression (in doses >1.5 mg kg<sup>-1</sup>)
- Respiratory depression – dose related
- Enhanced laryngeal reflexes, with potential for laryngospasm
- Secretions increased – pharyngeal & bronchial
- Emergence phenomena
  - Commoner in adults
  - Reduced by pre-treatment with midazolam

The use of thiopental or propofol in hypovolaemic patients necessitates considerable experience of these drugs, and a carefully considered dose reduction. Propofol is increasingly used in trauma patients because after induction it can be infused continuously to maintain unconsciousness. Infusion rates to maintain unconsciousness are variable in this group and will range from 200 – 600 mg h<sup>-1</sup> depending upon age, weight, cardiovascular status and the concurrent administration of other drugs, particularly opioids. Where time allows, the placement of an arterial line is invaluable during drug-assisted emergency intubation to provide accurate and continuous blood pressure measurement. Ketamine is a useful induction drug in some circumstances: it is a bronchodilator, causes less hypotension and respiratory depression than the other induction drugs, and is a potent analgesic. It can, however, cause hypertension and is relatively contraindicated in head injury.

#### *Neuromuscular blocking drugs*

THIS GROUP OF DRUGS ARE INJECTED TO ABOLISH LARYNGEAL REFLEXES AND TO FACILITATE LARYNGOSCOPY AND INTUBATION. NEUROMUSCULAR BLOCKING DRUGS ARE DIVIDED INTO TWO GROUPS ACCORDING TO THEIR ACTION AT THE NEUROMUSCULAR JUNCTION: DEPOLARISING (SUCCINYLCHOLINE, SUXAMETHONIUM) OR NON-DEPOLARISING (ATRACURIUM, ROCURONIUM, VECURONIUM, PANCURONIUM). SUCCINYLCHOLINE HAS THE FASTEST ONSET AND REMAINS THE NEUROMUSCULAR BLOCKING DRUG OF CHOICE FOR USE DURING EMERGENCY DRUG-ASSISTED INTUBATION.

#### Succinylcholine (Suxamethonium)

- Presentation: 2 ml ampoules containing 50 mg ml<sup>-1</sup>
- Dosage: bolus of 1.5 mg kg<sup>-1</sup> intravenously
- Following injection causes a transient, generalized fasciculation (twitching), followed by complete relaxation within 30-45 seconds
- Duration of action of 3-5 minutes - metabolised by a naturally occurring enzyme - pseudocholinesterase

Succinylcholine has several potential adverse effects:

- Repeated boluses cause vagal stimulation and bradycardia. Atropine must be readily available and always given before a second dose.
- May cause hyperkalaemia after major crush injuries, burns (high-risk period is 2 days to 6 months), major denervation injury and certain pre-existing neuromuscular disorders.
- Prolonged apnoea (3-24 hours) in patients who have very low pseudocholinesterase activity (1 in 3000).

- There is a risk of triggering malignant hyperpyrexia, a rare disorder of muscle metabolism.
- It needs to be stored at 4°C, as it breaks down at room temperature.

#### Atracurium

- Presentation: 2.5 and 5 ml ampoules containing 10 mg ml<sup>-1</sup>.
- Dose: 0.6 mg kg<sup>-1</sup>.
- Muscle relaxation allowing intubation occurs after 90-120 seconds; with smaller doses the time is progressively longer.
- Duration of action is also dose dependent, lasting about 30-40 minutes.
- Repeated doses of 0.1-0.2 mg kg<sup>-1</sup> to maintain relaxation and facilitate controlled ventilation.
- Indicated for maintenance of relaxation following intubation with suxamethonium or for the use in those situations where suxamethonium is contraindicated.
- No direct effect on the cardiovascular system.
- It should be stored in the fridge as it undergoes very slow spontaneous degradation at room temperature.

#### Rocuronium

- Presentation: 5 and 10 ml ampoules containing 10 mg ml<sup>-1</sup>.
- Dose: 0.6 mg kg<sup>-1</sup>.
- Muscle relaxation allowing intubation occurs after 90 seconds.
- Duration of action approximately 40 minutes.
- Repeated doses of 0.15 mg kg<sup>-1</sup> to maintain relaxation and facilitate controlled ventilation

If the initial dose is increased to 1.0 mg kg<sup>-1</sup>, the time to intubation can be reduced to around 60 seconds, but the duration is also significantly increased (at least 1 hour). This rapid onset makes it useful as a potential alternative in those patients where succinylcholine is contraindicated.

#### **Failed intubation**

Failure to insert a tracheal tube is not a disaster but failure to recognise incorrect placement or to allow the patient to become injured during further attempts to secure an airway is indefensible. Cease intubation attempts and re-oxygenate the patient's lungs once the SpO<sub>2</sub> decreases to 92% - this is the steep part of the oxyhaemoglobin dissociation curve and beyond this the oxygen saturation of arterial blood will decrease rapidly. Do not persist in attempts with intubation, try and insert a SGA device and if this fails a surgical airway must be created without delay.

## **WEBSITES**

[www.das.uk.com](http://www.das.uk.com)

The Difficult Airway Society of the United Kingdom. This society was formed in 1995 and aims to improve management of the patient's airway by anaesthetists and critical care personnel. Website and algorithms are available from the website.

[www.theairwaysite.com](http://www.theairwaysite.com)

This site is aimed at emergency physicians and orientated to American practice. It does however contain some useful information in the education section.

<http://gasnet.med.yale.edu/>

The best anaesthesia site on the web with free sign on and a virtual textbook of anaesthesia. This includes a good section on airway management.

<http://www.swsahs.nsw.gov.au/livtrauma/education/surgery/airway.asp>

Surgical airway management description with pictures.

<http://www.trauma.org/resus/moulagefour/moulagefour.html>

Cervical spine clearance moulage.

## **FURTHER READING**

1. Cranshaw J, Nolan J (2006). Trauma airway. *Brit J Anaesth CEPD*.
2. CARLEY SC, GWINNUTT C, BUTLER J ET AL (2002). RAPID SEQUENCE INDUCTION IN THE EMERGENCY DEPARTMENT: A STRATEGY FOR FAILURE. *EMERG MED J*: 19; 109.
3. CROSBY ED, COOPER RM, DOUGLAS MJ ET AL (1998) THE UNANTICIPATED DIFFICULT AIRWAY WITH RECOMMENDATIONS FOR MANAGEMENT. *CAN J ANAESTH* 45 (7): 757.
4. Gwinnutt CL and Nolan J (2003). Trauma Anaesthesia. In: Wylie and Churchill-Davidson's A Practice of Anaesthesia, 7<sup>th</sup> edition. (Eds Healy TEJ and Knight PR). Arnold, London, pp 689-706.
5. Mandavia DP, Qualls S and Rokos I (2000) Emergency Airway Management in Penetrating Neck Injury. *Ann Emerg Med* 35(3) 221.

## Chapter 4

# THORACIC TRAUMA

### Objectives

The objectives of this chapter are to teach the trauma team members:

- How to assess and manage thoracic injuries that are an immediate threat to life
- How to assess and manage thoracic injuries that are a potential threat to life
- The different investigations used in the management of thoracic trauma
- The assessment and management other non-life threatening thoracic injuries

Applied anatomy is covered in Appendix 4.1 and the pathophysiology of thoracic trauma in Appendix 4.2.

### Introduction

Chest trauma is the primary cause of death in about 25% of all trauma deaths and an associated factor in a further 50%, usually as a result of hypoxia and hypovolaemia. When the heart is not involved, the mortality of isolated penetrating chest trauma is low (<1%), but cardiac involvement increases mortality 20 fold. In an industrialised society, the commonest injury is to the chest wall, followed by pulmonary parenchymal injury, haemothorax, pneumothorax and flail chest. These conditions give rise to hypoxaemia and hypovolaemia that are often easily treatable with simple measures. Consequently the vast majority (approximately 85%) of thoracic trauma victims can be successfully treated without the need for thoracic surgical intervention. Unfortunately, thoracic trauma is associated with other injuries, particularly head injury, which may account for the overall high mortality in these patients. This chapter will discuss the principles of management of thoracic trauma. Although, the thoracic spine forms part of the thoracic cage, trauma to the spine is dealt with in Chapter 8.

### Assessment and management

This follows the well-defined protocol for all trauma victims used throughout this book, remembering that the primary survey and resuscitation are simultaneous events:

### Primary survey and resuscitation

This follows the same plan as described in Chapter 1 with the aim of identifying and correcting any immediately life-threatening conditions. In thoracic trauma there are six conditions that fall into this category:

- Airway obstruction or disruption
- Tension pneumothorax
- Open chest wound
- Flail chest
- Cardiac tamponade
- Massive haemothorax

## AIRWAY

Airway obstruction has been covered in Chapter 3. Members of the team responsible for the airway must assess, clear and secure the airway using whatever techniques are appropriate, whilst maintaining in line cervical immobilisation. A high-inspired oxygen concentration should be delivered.

Before applying the cervical collar take the opportunity to look and feel for clues in the neck that there could be an immediately life threatening condition:

<b>Sign</b>	<b>Life threatening condition</b>
Wounds	Laceration of the trachea, major vessels, oesophagus
Distended neck veins	Tension pneumothorax; cardiac tamponade
Tracheal shift	Tension pneumothorax;
Surgical emphysema	Breach of the main airway, tension pneumothorax
Laryngeal crepitus	Fractured larynx

## BREATHING

The patient's chest is exposed and inspected for:

- Marks & wounds
- Rate of breathing
- Effort of breathing - intercostal in-drawing; accessory muscle use
- Pattern – depth; symmetry

Rapid shallow breathing usually accompanies chest injury or developing hypoxia. Paradoxical chest wall movement during respiration (indrawing of the chest wall during inspiration and vice versa) suggests a flail segment.

Both sides of the chest are percussed and the note compared. Hyper-resonance suggests a pneumothorax, while a dull note indicates a haemothorax or possibility of a ruptured diaphragm. Surgical emphysema and crepitus are usually sought at this stage by quickly palpating the chest wall. Finally, the chest is auscultated on both sides, high in the axillae, to assess adequacy and equality of breath sounds.

### *Tension Pneumothorax*

This is caused by air entering the pleural cavity during inspiration (spontaneous or controlled ventilation) and failing to escape. The air can come from outside (open chest wound) or from the inner airways (lung trauma or rupture of an emphysematous bulla). For the tension to develop it is essential that the outer or inner breach acts as a ‘one way’ valve. Normally, the pleural cavity has a negative pressure with respect to atmospheric pressure, but if air accumulates over a period of time, the pressure becomes positive, causing collapse of the lung and mediastinal shift. The former causes hypoxaemia while the latter reduces venous return, cardiac filling and has a cardiac tamponade effect. These effects dramatically reduce cardiac output, causing hypotension and hypoperfusion that is exacerbated by the presence of hypovolemia. Compensatory mechanisms of increased catecholamine release and adrenergic discharge cause a tachycardia and peripheral vasoconstriction to try and maintain cardiac output and perfusion of the vital organs. Compression of the lung causes failure of ventilation and perfusion with rapidly increasing hypoxaemia. If the tension is not relieved, the compensation will fail causing ‘irreversible’ shock and ultimately cardiac arrest. The diagnosis is clinical **not** radiological and made by finding the signs shown in Box 4.1. Rarely, bilateral tension pneumothorax may occur. This should be suspected if there are no localising signs, profound hypoxia, increasing inflation pressures, surgical emphysema and cardiovascular collapse.

#### **BOX 4.1: SIGNS OF TENSION PNEUMOTHORAX**

- Anxiety
- Tachypnoea/respiratory distress/ Increasing effort of breathing
- Shock (hypotension & tachycardia)
- Hyper-resonance and decreased breath sounds in the same hemithorax
- Surgical emphysema (inconsistent)
- Deviation of the trachea away from the affected side (late)
- Engorged neck veins if no hypovolaemia
- Cyanosis (very late)

If a tension pneumothorax is suspected, it must be dealt with immediately by the insertion of a chest drain and if not immediately available by needle decompression (needle thoracocentesis) followed by the insertion of a chest drain (see below).

#### NEEDLE THORACOCENTESIS (FIG 4.1):

Needle decompression of a tension pneumothorax is an emergency life saving procedure; there is no time for local anaesthetic! It converts a tension pneumothorax into a simple pneumothorax.

- A large bore cannula (14 or 16g) is inserted in midclavicular line in the 2<sup>nd</sup> intercostal space just above the upper margin of the lower (3<sup>rd</sup>) rib.
- In most adults, the cannula will have to be inserted fully to reach the pleural cavity.
- Some advocate use of a syringe, containing a few ml of air, attached to the cannula. Once inserted the air is injected through the cannula to expel any tissue that may have blocked it on insertion.
- Upon removing the cap of the cannula or syringe, a rush of air is a positive sign, confirms the diagnosis and usually results in significant improvement of the patient's cardiovascular and respiratory status
- After removing the inner metal needle, the cannula may be fixed in place by adhesive tape
- Preparations must be made for an immediate definitive chest drain, as these cannulae easily become blocked by blood clots, bent or fall out, thus causing a recurrence of a tension pneumothorax.



**Fig. 4.1: Needle thoracocentesis**

**Fig. 4.1a: Diagnosis with saline in syringe**



**In up to 30% of patients, the length of common used IV cannulas (4.5 cm) may not be sufficient to fully penetrate the chest wall.**

In case of a misdiagnosis of tension pneumothorax (when there is no rush or passage of air), the clinicians will appreciate that a pneumothorax has now been created and needs to be dealt with as in simple pneumothorax.

If there is doubt about the diagnosis, a similar technique can be used as a diagnostic tool. A small diameter needle (eg. 21 G) is attached to a syringe, from which the plunger has been removed, and then partly filled with normal saline. Before inserting the needle, if the patient is being ventilated this must be stopped. The needle is then advanced into the chest and if there is gas under pressure in the pleural cavity, bubbling will be seen (Fig 4.1a). If negative, the small diameter needle will decrease, though not eliminate, the likelihood of an iatrogenic pneumothorax.

#### ***OPEN CHEST WOUND***

This results in a pneumothorax that is communicating with atmosphere. If the wound size is greater than two-thirds the diameter of the trachea, then during inspiration air will enter the pleural cavity via the defect rather than entering the lungs via the trachea. This compromises ventilation, causing hypoxia and if untreated, the lung will collapse. In some situations air can enter via the wound but not escape (sometimes referred to as a “sucking chest wound”) resulting in the development of a tension pneumothorax. This may be due to the shape of the wound or an inadequate dressing. The immediate management consists of removing any completely occlusive dressing to allow air to escape. An Asherman seal or new dressing, taped on three sides (to act as a flap valve), should be applied whilst preparation is made for the insertion of a chest drain via a separate route. The wound can then be formally redressed.

#### ***FLAIL CHEST***

This occurs when two or more ribs are fractured at more than one site along the rib. If conscious, the patient will be in severe pain with rapid, shallow breathing. Examination of the chest wall may reveal crepitus, abrasions or paradoxical movement of the flail segment. In the young adult the flail may not be obvious initially due to muscles splinting the fractured ribs, paradoxical movement only seen as the victim becomes exhausted. When the flail is large or central paradoxical movement will be noted early on – even in the young muscular adult.

In all cases management consists of high-flow, warmed, humidified oxygen, adequate fluid resuscitation and analgesia. This usually consists at this stage of opioids titrated intravenously. Intercostal or epidural analgesia should be considered during the secondary survey, depending on the expertise available. Some patients (Box 4.2) will require more aggressive management, as an increased inspired oxygen concentration fails to compensate for the progressive deterioration of ventilation, and increasing hypoxia as a result of the underlying lung injury. This will require tracheal intubation and controlled ventilation. In all patients arterial blood gases need to be monitored frequently to assess respiratory function. If intubated these patients should receive a lung protective ventilatory strategy (see Appendix 4.3) with advice from colleagues in critical care.

#### **BOX 4.2: INDICATIONS FOR TRACHEAL INTUBATION AND VENTILATION**

- Falling  $\text{PaO}_2$  or  $\text{PaO}_2 < 7 \text{ kPa}$  breathing air
- $\text{PaO}_2 < 10 \text{ kPa}$  on high flow oxygen
- Increasing  $\text{PaCO}_2$ , or  $> 6 \text{ kPa}$
- Exhaustion, respiratory rate  $> 30 \text{ breaths/min}$
- Associated head or abdominal injury

#### **Circulation**

The circulation doctor must make a quick assessment of the patient's colour, capillary return and presence of radial, femoral and carotid pulses while the nurse measures the pulse rate, blood pressure, pulse pressure and temperature. In the absence of a head injury, the level of consciousness gives an indication of organ perfusion supplemented later by urine output. The heart is auscultated and attention is paid to the presence of any murmurs. Neck veins will have been examined whilst the neck collar was off. Two large (14,16g) peripheral intravenous cannulae should be inserted and from one, 20 ml blood taken for investigations and cross-match or typing. Assuming a tension pneumothorax has already been ruled out or treated, the following life-threatening thoracic injuries must be looked for.

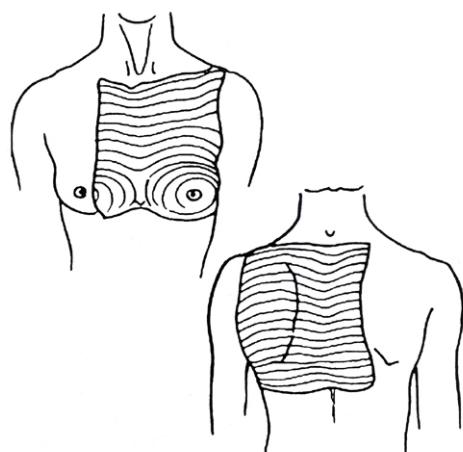
#### *Cardiac tamponade*

This should be suspected in any victim with a penetrating wound of the chest, neck or upper abdomen as these may involve the heart. In particular, a penetrating injury to the central chest area, from xiphisternum to clavicles and between the right nipple and lateral chest wall, anteriorly and posteriorly, (often referred to as 'the box' in Fig. 4.2) should immediately alert the clinician. A penetrating wound of the heart may result in bleeding into the 'intact' pericardial sac (smaller injuries of the pericardium may seal with a clot). Cardiac tamponade may also rarely be seen in blunt trauma and patients who are on anticoagulants. The

accumulated blood in the pericardial sac restricts ventricular filling during diastole, thus reducing the stroke volume during systole. This in turn results in a low cardiac output. As mentioned earlier, compensatory mechanisms come into play to maintain cardiac output (by increasing heart rate) and perfusion pressure (by increasing peripheral vascular resistance). If the pressure within the pericardial cavity is not released, cardiac output keeps falling and the compensatory mechanisms are not enough to maintain organ perfusion. Profound hypotension results, which in turn causes further myocardial injury (as a consequence of reduced coronary perfusion). If not relieved promptly, the patient will die from grossly inadequate cardiac function and perfusion. The signs of cardiac tamponade are shown in Box 4.3, but are only seen in approximately one-third of trauma patients.

#### BOX 4.3: SIGNS OF CARDIAC TAMPONADE

- Beck's triad
  - Shock
  - Raised JVP (impaired return to the right ventricle)
  - Decreased heart sounds (difficult in the resuscitation room)
- Pulsus paradoxus > 10 mmHg fall in pressure during inspiration
- Kussmaul's sign – raised JVP on inspiration



**Fig. 4.2: Sites of penetrating wounds associated with cardiac tamponade**

Initial management consists of augmenting venous return to maintain cardiac output by elevating the patient's legs and increasing the rate of intravenous infusion. The surgical management of choice is thoracotomy. Pericardiocentesis may be performed if the following criteria are met:

- No team member is competent to perform a thoracotomy.

- A surgeon is not available.
- The patient is in extremis (i.e. about to die).
- There is a very high degree of suspicion that tamponade is present

The blood often clots in the pericardium so there is a significant chance of having a negative tap even when a tamponade exists. Consequently pericardiocentesis is not a good idea in trauma patients because the patient who is moribund needs a thoracotomy, and the patient who is stable, but has a high certainty of having a tamponade, also needs a thoracotomy.

There are many risks of pericardiocentesis that include damage to organs (myocardium, lung, stomach, bowel, oesophagus, spleen, and kidney) and laceration of a coronary artery. Furthermore, performing a pericardiocentesis effectively commits a patient to a pericardial exploration. Ideally an urgent ultrasound should be organised while surgical help is obtained if there is suspicion of a tamponade.

If pericardiocentesis is required while arrangements are made for a thoracotomy the following should be followed:

- If time permits, prep and drape the skin, infiltrate local anaesthetic, ensure ECG monitoring
- The skin is punctured 1-2 cm below and left of the xiphoid process, using a long needle and cannula, at an angle of 45°
- While aspirating continuously, the needle is advanced towards the tip (inferior angle) of the left scapula
- The ECG monitor may show an injury pattern (ST depression or elevation) and arrhythmia (ventricular ectopics) indicating that the needle has advanced too far and is now touching the myocardium
- Withdraw the needle until a normal ECG is restored
- Once in the pericardial sac, as much blood as is possible should be withdrawn
- As the pressure on the myocardium decreases and its filling increases, the myocardium may move towards the needle tip and an injury pattern may be seen again

The cannula is taped in place, the metal needle is removed from within it and a three-way tap attached. Should the signs of tamponade recur, the pericardial sac can be reaspirated.

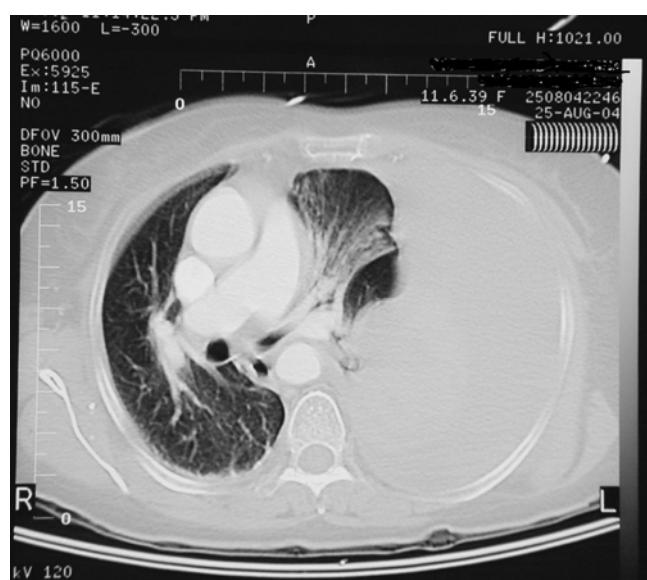
Cardiac tamponade is definitively treated by surgery to repair the cardiac laceration and evacuation of the clotted blood in the pericardial sac.

### *Massive haemothorax*

This condition is defined as greater than 1.5 litres blood in the thoracic cavity or continuing drainage of more than 200 ml/hour for four hours. It is usually secondary to a laceration of either the intercostal or internal mammary arteries. Accumulation of blood in the pleural cavity compresses the lung, impairs ventilation and causes hypoxia, while at the same time causing hypovolaemia. It may also occur following injury to one of the great vessels in the mediastinum for example a tear of the pulmonary hilum. Such injuries are commonly fatal unless victims reach hospital very rapidly and undergo emergency thoracotomy. The signs of a massive haemothorax are shown in Box 4.4. Immediate management will consist of securing venous access and commencing fluid resuscitation, with the early requirement for blood. Venous access should precede insertion of a chest drain as the latter may precipitate circulatory collapse, making cannulation extremely difficult. After the chest drain has been inserted and the diagnosis confirmed some of these patients will need a thoracotomy to control the bleeding.

#### **BOX 4.4: SIGNS OF A MASSIVE HAEMOTHORAX**

- Shock
- Dull to percussion over the affected hemithorax
- Decreased breath sounds over the affected hemithorax
- Raised JVP



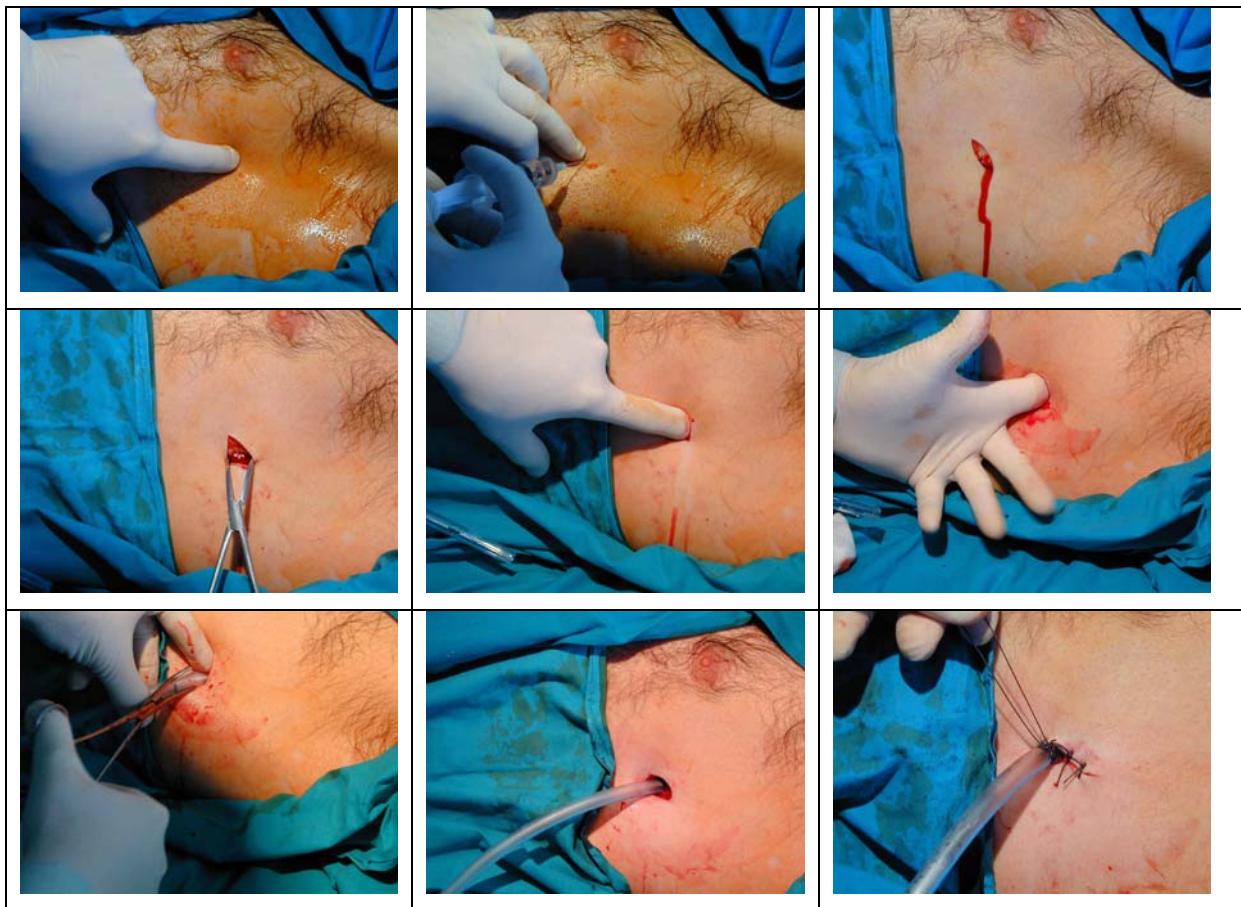
**Fig. 4.3: CT scan showing presence of a massive left-sided haemothorax. Note shift of the mediastinum.**

### **Insertion of a chest drain (Fig. 4.4)**

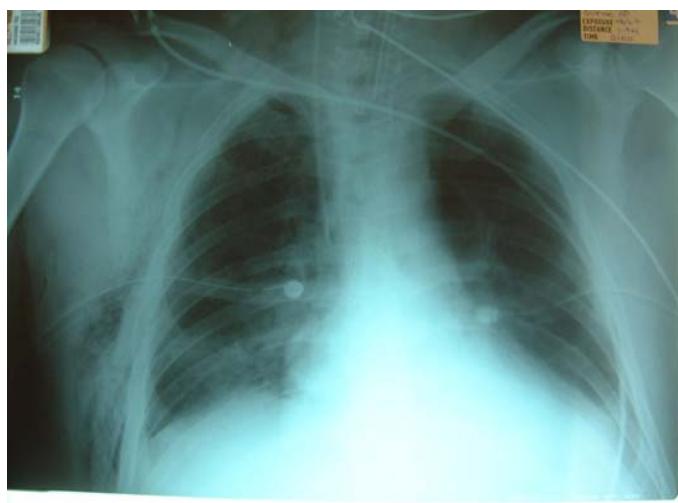
A chest drain is inserted in the 5<sup>th</sup> intercostal space just anterior to the mid-axillary line.

- The patient's arm is abducted and the 5<sup>th</sup> intercostal space identified, the 4<sup>th</sup> or even 6<sup>th</sup> space may be chosen if there is an injury at the site of 5<sup>th</sup> space
- The chest is cleaned and the area draped
- Just anterior to the mid axillary line, local anaesthetic is infiltrated into the skin, subcutaneous tissue, down to the pleura keeping just above the upper margin of the lower rib to avoid the neuro-vascular bundle
- A 3-5 cm transverse incision is then made along the upper margin of the rib through the anaesthetised area
- The track is continued through the intercostals muscles using blunt dissection down to the pleura
- The pleura is then pierced just above the upper margin of the rib using a curved clamp
- The operator then inserts a finger along the track into the pleural cavity and sweeps around the space to detect the presence of any adhesions or bowel (in case of a ruptured diaphragm)
- Remove the metal trocar from the drain (if present) and clamp the distal end of the drain. The tube is then directed through the incision into the pleural cavity (basal placement facilitates drainage of blood, while apical placement facilitates drainage of air)
- Fogging and condensation caused by warm air escaping down the drain confirms placement in the pleural cavity
- The proximal end of the drain is then connected to a suitable underwater seal. There may initially be a rush of air out into the underwater seal but this usually settles down and a raised fluid level in the tubing suggests establishment of intra-pleural negative pressure and nearly resolved pneumothorax
- The chest drain is then secured by anchoring sutures, and an appropriate dressing
- A chest radiograph, taken after the placement of the chest drain, confirms the tube placement and resolution of the pneumothorax (Fig. 4.5)
- The circulation nurse must continue to monitor the chest drain to ensure it is swinging with ventilation and to note any blood loss

**Fig. 4.4: Sequence of events when inserting a chest drain.**



Once the six immediately life-threatening conditions have been eliminated or resolved the primary survey should proceed along the lines already discussed.



**Fig. 4.5: Chest x-ray showing bilateral chest drains**

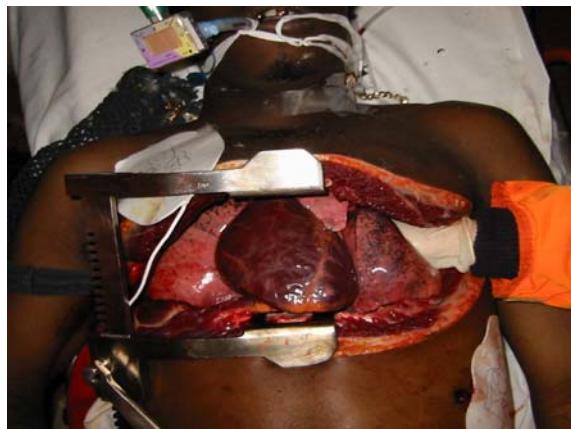
### **Emergency room thoracotomy**

This is a controversial topic. This strategy is a desperate attempt to save a patient who is in immediate danger of dying. Recently, it has been suggested that the procedure is justified

in patients with penetrating trauma, who displayed vital signs at the scene, where there is no alternative and the patient is rapidly deteriorating. However, those patients who do not have vital signs at the scene and patients with blunt injuries who have no vital signs on arrival in the Emergency Department should not undergo emergency room thoracotomy. The patients who most benefit from this procedure are those with penetrating cardiac injury who decompensate just before, or on arrival, in the emergency room. Because of the risks of damage to the thoracic organs, only a doctor with appropriate training should perform this procedure (Fig. 4.6).

Emergency thoracotomy allows:

- Pericardial incision and evacuation of pericardial blood causing tamponade.
- Direct control of exsanguinating thoracic haemorrhage.
- Open cardiac compression.
- Cross clamping of descending aorta to stop blood loss below diaphragm and allow heart filling to achieve cardiac and brain perfusion.
- Cross clamping of pulmonary hilum to control exsanguinating pulmonary haemorrhage.



**Fig. 4.6: Emergency Department thoracotomy**

## **Secondary survey**

Only when all the immediately life threatening conditions described above have been treated or eliminated, the remainder of the primary survey completed and the patient's condition has been stabilised, a detailed head-to-toe examination is carried out in the manner described in Chapter 1 to identify the potentially life threatening injuries (Box 4.5) and begin a plan for definitive care. It also includes investigations for example x-rays, ECG and arterial blood gases. Further invasive monitoring may require the patient to be transferred to a specialist area such as intensive care. If this happens before the secondary survey has been completed, this must be reported and recorded. Other sophisticated investigations may be

warranted as a result of the findings on preliminary chest radiograph, for example CT scan or angiography. It is also important to remember that many of the conditions sought in the secondary survey may develop relatively slowly and if the secondary survey is performed soon after the patient presents, signs or symptoms may be minimal or absent.

It must be emphasised that should a patient's condition deteriorate during the secondary survey, it is essential to return to primary survey and resuscitation in case a new life threatening condition has developed or an already existing one has been missed! The secondary survey should only proceed (or be completed) once the threat to life has been satisfactorily dealt with.

#### **BOX 4.5: POTENTIALLY LIFE-THREATENING THORACIC CONDITIONS**

- Pulmonary contusion/laceration (parenchymal injuries)
- Blunt cardiac injury
- Ruptured diaphragm
- Aortic rupture
- Ruptured oesophagus
- AIRWAY RUPTURE

#### **Potentially life threatening thoracic injuries**

##### *Pulmonary contusion/laceration (parenchymal injuries)*

There is a high incidence (>50%) of pulmonary parenchymal injuries in thoracic trauma the primary effect of which is to impair the patient's oxygenation. Direct impact causes haemorrhage in the underlying lung tissue and in adults there are almost always associated rib fractures and often an accompanying pneumothorax or haemothorax. The contusion is usually, but not always, localised to the area directly adjacent to of rib fractures. On examination ventilation is often rapid and shallow, there may be bruising or abrasions on the chest wall and tenderness on palpation. Percussion and auscultation may be normal, particularly early after the insult. As the pathophysiological effects of contusion progresses over 24-48 hours lung compliance is reduced and respiratory distress ensues. Serial arterial blood gas analysis is essential – the PaO<sub>2</sub> gradually falls and A-a gradient increases as the ventilation/perfusion mismatch increases (see Appendix 4.2). It is very important to carefully assess the chest x-ray for evidence of lung contusion bearing in mind that this may occur away from the area of the chest wall involved in direct impact. A series of x-rays may be needed as the condition may evolve over hours or even days (Fig. 4.7). Management consists initially of warmed, humidified oxygen; careful fluid resuscitation and observation preferably in a critical care area. Larger contusions cause respiratory failure and

hypoxaemia of various degree and these patients will require ventilatory support. Some patients with traumatic pulmonary contusions may be managed using non-invasive ventilatory support provided adequate oxygenation is ensured and effective analgesia is provided. Patients with refractory hypoxia and overt respiratory failure will require intubation and ventilation using a lung protective strategy (see below).

The criteria used to determine the need for invasive ventilation will vary between units and examples are given in Box 4.6. Blast lung injury carries a risk of systemic air embolism the risk of which may be increased by positive pressure ventilation, and this should be considered when managing patients who are severely injured following bombings.

**BOX 4.6: INDICATIONS FOR INVASIVE VENTILATION FOLLOWING PULMONARY CONTUSION**

- Decreased level of consciousness
- Elderly
- Poor analgesia and inability to clear secretions
- Increasing PaCO<sub>2</sub>
- Decreasing PaO<sub>2</sub>
- General anaesthesia required for surgery
- Transfer required
- Severe pre-existing lung disease



**Fig. 4.7: Extensive right-sided pulmonary contusion**

One of the most controversial questions in management of pulmonary contusions is fluid therapy. Fluid requirement varies from patient to patient and also depends upon loss of blood from other injuries. The contused lung tissue is also prone to damage by fluid overload. However, fluid and blood resuscitation is mandatory if the consequences of hypoperfusion are to be avoided. Recommendations for fluid infusions in such patients are varied and fluid management in these patients may best be guided by invasive haemodynamic monitoring

with a pulmonary artery catheter (PAC), oesophageal Doppler or echocardiography. Recently, attention has been drawn to the technique of small volume resuscitation with hypertonic saline (7.5%), which effectively supports the haemodynamic profile of these patients without the disadvantages of administering large fluid volumes.

#### *Pulmonary lacerations*

Pulmonary lacerations are usually the consequence of penetrating thoracic trauma but they can also be produced by blunt injuries with a heavy impact. These injuries are invariably accompanied by a pneumothorax, haemothorax or haemopneumothorax. The injury is managed using the principles already described. About 5% of these patients will require formal thoracotomy, especially if there is persistent bleeding, persistent pneumothorax, and haemoptysis or for evacuation of clotted haemothorax or failure of the lung to re-expand. Most patients can be managed by suturing of lacerations at operation; occasionally a lobectomy or pneumonectomy may be required.

#### **Blunt cardiac injury**

Blunt cardiac injury (BCI) is a common complication of chest trauma and is most commonly caused by motor vehicle crash. Depending on the various criteria for diagnosis, blunt cardiac injury can be expected to have an incidence in the range of 8-71% in patients sustaining blunt chest trauma. The true incidence remains unclear because there is no agreement on diagnostic standards.

Blunt cardiac injury represents a spectrum of conditions, which range from minor ECG or enzyme abnormality to septal, free wall or valvular rupture. The spectrum also includes injury with coronary artery thrombosis, myocardial dysfunction causing cardiac failure and injury with complex arrhythmia. The anterior right ventricle is the most common area involved in BCI, followed by the anterior interventricular septum and anterior-apical left ventricle. Blunt injury can also damage the conduction system resulting in arrhythmias and bundle branch block patterns.

Cardiac contusion specifically is a pathologic diagnosis, which lacks an accurate clinical definition. The findings on histology are of subepicardial and intramyocardial haemorrhage and disruption, inflammatory cell infiltration, and interstitial oedema. At post-mortem examination the hearts of patients who died of BCI show changes that resemble myocardial infarction (MI) but with a more abrupt demarcation between normal and abnormal, more haemorrhage, and more laceration.

The key clinical issues are; identifying patients at risk of clinically significant BCI, identifying the consequences of BCI, particularly arrhythmia and hypotension due to myocardial dysfunction and investigation and management to prevent adverse outcomes.

Patients at risk of a clinically significant BCI have been exposed to major forces and often have associated chest and other injuries, may have an abnormal ECG on admission and have haemodynamic instability that is not explained by other injuries. A normal ECG and haemodynamic stability virtually excludes significant BCI. Most haemodynamically unstable patients with multiple trauma are unstable because of haemorrhage and active haemorrhage from injuries to the abdomen, thorax, pelvis, retroperitoneum, long bones and body surface needs to be excluded. Objective assessment of cardiac injury is provided by echocardiography. The most common echocardiographic finding is right ventricular wall motion abnormality with some dilatation. Severe ventricular dysfunction without surgically correctable pathology is usually managed with careful fluid and inotropic management guided by assessments of cardiac filling and output (invasive or non-invasive). The use of intra-aortic balloon pump counterpulsation, after excluding aortic injury, makes physiological sense. It rests the damaged myocardium and has been used successfully in this situation, as does the use of a left ventricular assist device in selected cases. All of these strategies will require involvement of critical care physicians.

Coronary artery injury presents something of a dilemma in trying to work out which came first, the infarct or the trauma but is not of practical importance. These patients are unlikely to be candidates for thrombolysis in the presence of serious trauma but may benefit from other methods of revascularisation including percutaneous and open surgical techniques, while recognising there may be difficulties with anticoagulation during or after the procedure.

Echocardiography remains the most useful investigation for suspected BCI but is impractical for all patients. Therefore, ECG, cardiac enzyme measurement and the selective use of echocardiography are used to assess other patients who are more stable. A normal ECG helps to identify a very low-risk patient. ECG changes are neither specific nor sensitive for significant myocardial dysfunction but have a high negative predictive value for cardiac complications. Up to 60% of patients with significant chest trauma have an abnormal ECG and approximately half of these patients have ECG changes which include conduction abnormalities, ST depression or elevation  $>1$  mm, and T-wave inversion in two consecutive leads. Echocardiography will identify wall motion abnormalities but its role in the management of stable patients after chest trauma has been questioned because the presence of wall motion abnormality does not necessarily correlate with a worse outcome in patients with normal ECG and enzymes. Therefore, cardiac enzyme measurement is

commonly performed as a second screening test. Creatine kinase (CK) and its isoenzyme (CK-MB) are often raised in the presence of multi-trauma and are therefore of little value. Other markers of myocardial injury, troponin T and I, while more specific, may not predict worse outcomes and the need to measure these enzymes in blunt injury patients has caused controversy.

Although troponin has improved specificity when compared with conventional markers, it has low sensitivity as well as low predictive values in diagnosing myocardial contusion and is not good for diagnosing blunt cardiac injury in haemodynamically stable patients.

Current evidence supports the following recommendations:

- An admission ECG should be performed on all patients if there is suspected BCI.
- If the admission ECG is abnormal (arrhythmia, ST changes, ischemia, heart block, unexplained ST), the patient should be admitted for continuous ECG monitoring for 24 to 48 hours. Conversely, if the admission ECG is normal, the risk of having a BCI that requires treatment is insignificant, and the pursuit of diagnosis should be terminated.
- If the patient is hemodynamically unstable, an imaging study (echocardiogram) should be obtained. If an optimal transthoracic echocardiogram cannot be performed, then the patient should have a transoesophageal echocardiogram.

Late complications such as arrhythmia, aneurysm, and heart failure have been reported but are rare. Perhaps this reflects the fact that the majority of trauma patients are younger and in good health and that those with fatal injury die before reaching hospital or shortly after. As the underlying cause of the myocardial trauma is not an ongoing process when compared to atherosclerosis it is not surprising that these patients do much better than in those with myocardial infarction from coronary artery disease.

### Ruptured diaphragm

This may arise as a result of either blunt or penetrating trauma. Blunt injuries to abdomen or thorax that produce a sudden rise in pressure may cause a burst type of injury resulting in irregular tears, with herniation of abdominal viscera into the thoracic cavity causing respiratory embarrassment and occasionally an acute abdomen due to strangulation of bowel loops through the tear. The commonest causes of diaphragmatic injuries are motor vehicle crashes with side impact collisions causing three times more diaphragmatic disruption than frontal impacts. The left hemidiaphragm is more prone to injury than the right, probably because of the protective effect of the liver. Consequently, right-sided injuries can be very severe and usually involve the liver (Fig. 4.8).

Where the diaphragm is injured by penetrating trauma, 75% of cases are associated with intra-abdominal injury, whilst 20% of penetrating injuries of the thorax will involve the diaphragm. Again the left side is more involved than the right, probably because most assailants are right handed! As the diaphragm is always moving, these small penetrating wounds do not heal spontaneously and will enlarge over a period of time and may result in herniation of the abdominal contents years later.



**Fig. 4.8: Ruptured left hemidiaphragm.**

Many of these injuries are asymptomatic at presentation; the most suggestive sign of a diaphragmatic injury is the proximity of the penetrating wound. On examination, breath sounds may be decreased over the affected hemithorax and occasionally bowel sounds may be heard in the chest. Often the first suspicion comes from the appearance of the chest x-ray. The injured hemidiaphragm is elevated and there may be bowel in the pleural space. If a gastric tube has been inserted, this may be visible above the diaphragm. If diagnostic peritoneal lavage is performed, this may be positive if there is an intraabdominal injury, and if a chest drain is in the same hemithorax, the lavage fluid may appear in the drain. Diaphragmatic injuries may also be diagnosed with contrast gastrointestinal studies, ultrasonography, abdominal/thoracic CT or at laparoscopy or thoracoscopy. Diaphragmatic ruptures do not heal spontaneously and should always be surgically repaired.

### **Aortic rupture/great vessel injury**

These are found in victims of high-speed motor vehicle crashes and falls from great heights, and 85% of these injuries are due to blunt trauma. The majority (80-90%) of the patients die at the scene of the accident from massive blood loss. Of the patients reaching hospital alive, only 20% will survive without operation. The mortality remains high even after surgery.

In cases of aortic rupture, the clinical presentation depends upon the site of injury. Patients with injury to the intrapericardial portion of the ascending aorta will usually develop a cardiac tamponade. Extrapericardial ascending aortic injury produces a mediastinal haematoma and a haemothorax, usually on the right side. Injury to the aortic arch may remain undiagnosed initially if the adventitia remains intact and the damage is contained in the form of a mediastinal haematoma. Rapid deceleration is believed to be responsible for damage to the aorta that most commonly occurs in the region of ligamentum arteriosum, just distal to the origin of left subclavian artery. Here the descending aorta is relatively firmly fixed while the heart is mobile in the mediastinum, consequently it is the point of maximal shearing forces. Patients may show transient hypotension, which responds well to fluid therapy and further clinical signs may be absent. This may delay the diagnosis with catastrophic results should the aorta rupture completely. Thus a high index of suspicion and judicious use of appropriate investigations cannot be overemphasised.

Aortic disruption should always be suspected in patients with profound shock and who have no other external signs of blood loss and in whom mechanical causes of shock (tension pneumothorax and pericardial tamponade) have been excluded. In a patient who has suffered rapid deceleration, upper body hypertension (relative to lower body) should immediately arouse a suspicion of trauma to the aorta. Symptoms (if the patient is conscious) may include severe retrosternal pain, pain between the scapulae, hoarseness of voice (pressure from haematoma on the recurrent laryngeal nerve), dysphagia (compression of the oesophagus) and paraplegia or paraparesis (reduced perfusion of the vessels supplying the spinal cord). There may also be ischaemia or infarction of other areas, for example limbs and abdominal organs. There may be accompanying fractures of ribs or sternum as well. There are several features that may be seen on a plain chest x-ray that should make one think of the possibility of thoracic aortic rupture (Box 4.7).

**Box 4.7: Radiological features suggesting thoracic aorta rupture**

- Widened mediastinum (Fig. 4.9)
- Pleural cap (apical haematoma), especially on the left
- Compression and downward displacement of left main bronchus
- Fractured first or second ribs
- Trachea shifted to right
- Blunting of the aortic knuckle
- Raised right main bronchus
- Left haemothorax with no obvious rib fractures or other cause
- Deviation of the nasogastric tube to the right



**Fig. 4.9: Widened mediastinum**

With these symptoms and signs, investigations for aortic injury and consultation with thoracic surgeon are mandatory as delay may mean profound hypotension and death. The definitive investigation of choice is angiography or a CT angiogram of the aortic arch, the choice depending on local policy. Survival in patients who have their injury repaired surgically and who have remained haemodynamically stable during the repair is 90%. Minimally invasive repair using aortic stenting techniques are also being used.

#### *Oesophageal injury*

Oesophageal injuries are uncommon, as it is well protected in the posterior mediastinum. The cervical oesophagus is more prone to injury from penetrating trauma and following crush injury. The thoracic oesophagus may rupture following a severe blow to the epigastrium. On examination there is often shock and severe pain out of proportion to the apparent injuries. Pain on swallowing suggests oesophageal injury and must be investigated. A left-sided pneumothorax or effusion in the absence of trauma or rib fractures should raise suspicions. Surgical emphysema or signs of peritonitis, depending on site of rupture may develop with time. Patients should be kept nil by mouth and the oesophageal injury should be surgically repaired, ideally within 12 hours. A cervical oesophagostomy is performed to prevent soiling of the mediastinum, along with a gastrostomy. Surgical repair and restoration of oesophageal continuity is undertaken when mediastinal infection has settled and patient is stable. Mediastinitis is a very severe form of infection and may rapidly progress to multiple organ failure.

#### **Airway rupture**

Most patients with major airway injuries die at the scene due to asphyxia, intrapulmonary haemorrhage or aspiration of blood. However, survival is possible if the transection of the airway, even a major airway, is sealed off by soft tissue. These injuries produce severe surgical emphysema, pneumothorax, pneumomediastinum, haemothorax, pneumopericardium or even pneumoperitoneum. Diagnosis is by high index of suspicion, evidence of

pneumothorax, pneumomediastinum and confirmed by airway endoscopy. Injuries may be overlooked even on endoscopy if the vision is blocked by blood in the airway and complications increase if not properly diagnosed. Fractures, especially scapular, clavicular or ribs 1 - 3 indicate high impact injury and airway injury must always be suspected in such patients. Treatment of these injuries is almost always surgical. An urgent consultation with thoracic surgeon is mandatory in all such cases.

## **Imaging and investigations in thoracic trauma**

### **Chest radiograph**

This is the most important basic investigation performed in a patient with thoracic trauma and is mandatory in these patients. Although an erect posteroanterior (PA) view is the best option, in practice, an anteroposterior (AP) film with the patient lying supine is often all that is possible.

Fracture of first three ribs indicates a high-energy transfer and should prompt a search for other underlying injuries for example an aortic injury or severe pulmonary contusion. Fractures of the lower ribs should prompt a search for injury to the abdominal organs, spleen and the kidneys. Other fractures may also be obvious, sternum, clavicle and or scapula, which may suggest the presence of a flail segment. It has been suggested that only 50% of rib fractures are evident on initial chest radiographs.

When a pneumothorax is suspected, a chest radiograph in full expiration is ideal as the reduced air in the lungs provides a better contrast between the air in the pleural cavity and the lung parenchyma. Pneumothoraces are identified by a rim of complete translucency on the lateral side of the lung, without any lung markings distally. A rim of about 1 cm on the radiograph corresponds to a pneumothorax of 10% of total lung volume. A typical apico-lateral pneumothorax is often "invisible" on the initial supine chest x-ray in as many as 30% of cases because the air will be lying anterior to the lung. CT is playing an increasingly important role in the diagnosis of pneumothorax (see below). In the treatment of pneumothoraces, the chest radiograph is only a guide; the treatment of the pneumothorax itself is dependent on the clinical condition of the patient. Surgical emphysema does not need a chest radiograph for diagnosis but its presence is important; it may make visualization of a pneumothorax difficult but also, it can be an indirect sign of a pneumothorax. Consequently, the CXR must be examined carefully in these patients.

A haemothorax, in an erect radiograph, can be diagnosed by obliteration of the costophrenic angle (requiring presence of 300-400 ml of blood). In supine patients, this amount of blood may not be immediately obvious. Careful observation of the chest radiograph may reveal

presence of decreased radiolucent ‘veiling’ in one hemithorax (massive haemothorax will result in a unilateral whiteout). A haemopneumothorax will have an air-fluid level.

Pulmonary parenchymal injuries may be masked by the presence of a pneumothorax, a haemothorax, or both, along with other pathology (rib fractures etc). The appearances range from a small infiltrate, an involved lung segment or lobe, to a complete ‘white-out’ of the whole lung. Contusions usually resolve over a period of days so serial radiographs are important in assessing progression. Lung lacerations are accompanied by pneumothoraces or small opacities denoting lung haemorrhage and haematomas. Lung haematomas can also be difficult to diagnose and they may appear as pulmonary infiltrates because of the extravasation of blood into the surrounding lung tissue.

Blast injuries can give rise to diffuse bilateral infiltrates in the lung field, often with associated pneumothoraces and/or pneumomediastinum. A similar picture is seen in patients with traumatic asphyxia but here the radiographic appearance is due to diffuse interstitial haemorrhage along with pulmonary oedema.

Injuries to trachea and bronchi are suspected in the presence of fractures to first two to four ribs and presence of mediastinal emphysema, pericardial air and subcutaneous emphysema. These injuries can be difficult to diagnose and a quarter of the chest radiographs in these patients are reported as normal.

### **Computerised tomography (CT)**

CT is becoming increasingly used in the assessment of patients with major trauma as the information obtained is often very specific and there is a growing expertise available. The disadvantages of CT are delay in organising the procedure and transport of the patient to a specific area, often away from a resuscitation room. Hence this procedure can only be performed in stable patients in whom life-threatening conditions have been effectively managed. However, recent innovations in CT angiography (spiral, multi-slice technology) make CT imaging of trauma victim easier and quicker; this is becoming popular in many countries as the imaging of choice in trauma victims.

Notwithstanding the disadvantages, valuable information may be obtained about occult (or difficult to diagnose) pneumothoraces (Fig. 4.10), mediastinal haematomata, small haemothoraces and pericardial fluid. One such small haemothorax is the ‘pleural cap’ or minimal apical haemothorax; the presence of which should prompt the clinicians to look for injuries such as a ruptured diaphragm or aortic injury.



**Fig. 4.10:** CT showing small pneumothorax.

The most significant contribution of CT scanning is in the diagnosis and identification of aortic and great vessel injury. It also distinguishes other causes of 'widened mediastinum' (congenital abnormalities, anatomical variations) from that caused by aortic injury. In a recent study, 22% of aortic injuries were diagnosed only after CT imaging. CT also detects small haematomas that may not be obvious on chest radiographs and the presence of such, especially in the mediastinum may indicate the need for an aortogram to rule out aortic injury.

### Magnetic resonance imaging (MRI)

MRI offers a number of potential advantages over CT; solid structures can be differentiated from blood vessels and fluid collections without the use of intravenous contrast; image quality is superior and there is also no danger of radiation. However, at present MRI has limited utility in early trauma management as the scans take a long time and isolate critically ill patients for an unacceptable period of time. Furthermore most currently used ventilators and monitoring are not compatible with the strong magnetic field of MRI scanners. MRI is currently being evaluated in diagnosis of cardiac injuries as well as small diaphragmatic injuries.

### Endoscopy

Several forms of endoscopy are used in thoracic trauma some of which may be performed in the emergency or intensive care departments by the patient's bedside.

Bronchoscopy may be the only diagnostic tool useful in the diagnosis of life-threatening injuries such as tracheal or bronchial tears. It is indicated in thoracic trauma victims when there is:

- Haemoptysis
- Subcutaneous emphysema without obvious pneumothorax
- Persistent pneumothorax

- Persistent pneumomediastinum
- Massive air leaks through a chest tube (suspicion of ruptured bronchus).

Oesophagoscopy is indicated in diagnosis of oesophageal injury, often when contrast swallows are negative or cannot be performed in presence of clinical suspicion of oesophageal injuries.

### **Echocardiography**

Two types of echocardiography are available; *transthoracic echocardiography (TTE)* and *transoesophageal echocardiography (TOE)*. Whereas TTE is commonly available, TOE may not be available in all institutions.

TTE can provide useful information about the ventricular septal and wall movement abnormalities (hypokinetic segments), valvular integrity and presence of pericardial effusion as well as mural thrombi. TTE can also calculate ventricular ejection fraction (a measure of overall ventricular function). TOE is a more accurate instrument in the diagnosis of cardiac abnormalities and useful in patients when large areas of surgical emphysema or mediastinal emphysema preclude effective examination by TTE. TOE provides information about valvular injuries, intracardiac shunts, septal defects and great vessel injuries. It is rapid, safe and its diagnostic ability by the bedside makes TOE a potential diagnostic tool in suspected aortic injuries.

### **Angiography**

Angiography is still considered the ‘gold standard’ in imaging for suspected aortic or great vessel injury in thoracic trauma or any other vascular trauma. A widened mediastinum on the initial chest radiograph is regarded as a standard indication for angiography, despite a high false positive rate. Various forms of vessel injury may be identified for example, laceration (from penetrating trauma), avulsion, internal flap formation, obstruction (from blunt trauma). Rarely, an aortogram may appear completely normal (some times in blast injury) despite the presence of a serious injury as the intima may not be breached.

### **Other investigations**

ECG, cardiac enzyme analysis and arterial blood gases (ABG) are non-specific but easy bedside procedures and give valuable information about cardiorespiratory dysfunction and may alert the clinician to evolving problems. New Q waves or heart block on ECG usually indicates a significant myocardial injury. Cardiac enzymes are of limited usefulness as

discussed above. ABGs, like chest radiographs, are mandatory for any trauma victim but more specifically for thoracic trauma.

## **OTHER INJURIES IN THORACIC TRAUMA**

### *RIB FRACTURES*

Rib injuries and fractures are common in thoracic trauma. Multiple rib fractures are usually associated with contusion of the underlying lung to a varying degree. Apart from injury to the underlying lung, pain from rib fractures impairs breathing by restricted movement and inadequate cough. This in turn leads to accumulation of secretions, infection, collapse and consolidation and sometimes, frank respiratory failure. Consequently, most patients with more than two fractured ribs will need admission to hospital for analgesia and monitoring of respiratory function. Patients with isolated rib injuries, or the elderly with osteoporosis who may suffer 'simple' isolated rib fractures with little or no injury to the underlying lung parenchyma, will require adequate analgesia. Strapping of the chest wall as a form of 'therapy' has now been abandoned.

Despite genuine concerns about opiates, especially in the elderly, their careful use is beneficial especially when combined with supervision and monitoring, adequate humidification of oxygen, and chest physiotherapy. A useful way of administering opiate analgesia is by patient controlled analgesia (PCA), in consultation with an acute pain service (Chapter 16). Problems of inadequate analgesia should be vigorously pursued and may need consultation with senior medical staff. There is no justification for allowing patients to breath and cough inadequately for want of adequate analgesia.

Should these simple measures prove unsatisfactory, alternative methods of pain relief should be considered. These include epidural analgesia, intercostal nerve block (Chapter 16) and intrapleural analgesia. Epidural analgesia consists of the administration of a continuous infusion of local anaesthetic agent, sometimes containing an opioid (morphine or fentanyl), into the epidural space via a fine catheter inserted percutaneously. It is a specialised technique, particularly in the thoracic vertebral region and should only be administered by those with experience and preferably with the involvement of the acute pain service. Because of the risk of complications, particularly hypotension, such patients should be cared for in a high dependency area. Epidurals are contraindicated in presence of an open wound at the intended site of the epidural, thoracic vertebral column fractures, infection at or close to the site of epidural and coagulation abnormalities.

Alternative modalities of providing analgesia are via indwelling intrapleural or paravertebral catheters. This form of therapy has been used when epidurals are contraindicated because of open wounds, fractures of the thoracic spine or coagulopathy. Details of these techniques are beyond the scope of this text.

#### ***SIMPLE PNEUMOTHORAX***

Patients with a simple pneumothorax will present with sharp pain in the chest, particularly on inspiration (rib fractures present in the same way!). Respiratory distress varies in degree, depending upon the extent of pneumothorax, accompanying rib fractures and presence or absence of lung contusion. A pneumothorax may be suspected from bruising or a penetrating wound and decreased movement of the ipsilateral chest wall. Hyper-resonant percussion note may be present on the affected side and breath sounds may be decreased or absent. There may be tachycardia but if there has been no loss of blood, the patient should not be hypotensive.

#### ***HAEMOTHORAX***

This may be diagnosed on an erect chest film and should be drained, as a radiologically visible haemothorax is at least 500 ml in quantity. This will also allow monitoring of the rate of blood loss from the pleural cavity. Large size drains (32F) are necessary otherwise the blood will clot in the drain. Failure of the haemothorax to drain may also suggest clotted blood in the pleural cavity or a collection of subpleural blood. In either case, a thoracotomy will eventually be required to drain the clots and/or blood. Continued blood loss from a chest drain may indicate a clotting abnormality, usually due to dilutional thrombocytopenia or diluted coagulation factors following large blood transfusions. These should be treated accordingly.

#### ***Air embolism***

Four percent of patients with major thoracic trauma are said to have air embolism associated with bronchopulmonary-venous fistulae. This is a life-threatening problem occurring either in blunt or penetrating trauma and is a particular feature of blast injury to the lung. Unfortunately, this condition is not easy to diagnose. It can affect the left or right sides of the heart. Left sided embolism usually presents as sudden cardio-vascular collapse shortly after initiation of mechanical ventilation as air is forced into the pulmonary vein by positive pressure. Expansion of the intravascular space by fluid resuscitation, increasing systemic arterial pressure by inotropes (and/or vasopressors), ventilation with 100% oxygen and reduction of tidal volume of the ventilator to reduce intra-thoracic pressure are the holding measures until a thoracotomy can be organised. Mortality remains high, particularly with the

neurological consequences of air entering the cerebrovascular system. Patients with a patent foramen ovale may have paradoxical systemic embolism following right-sided air embolus.

#### *Thoracic duct injury*

Usually occurs from blunt trauma due to a fall, compression, hyperextension or hyperflexion spinal injuries. It presents itself as a pleural effusion subsequently found to be chyle (chylothorax), which may occur soon after the injury or be delayed. Milky white fluid draining from the chest drain is highly suspicious and analysis of the fluid showing chylomicrons will confirm the diagnosis. Consultation with a thoracic or general surgeon is essential in management of these patients as there are varying opinions as to the best course of management for these patients.

### **Summary**

Thoracic trauma is common and contributes directly to death in about a quarter of all trauma deaths. Most deaths are due to hypoxaemia and hypovolaemia, both conditions that should be aggressively treated. Most of the thoracic injuries (85%) can be managed with simple measures of oxygenation, fluid and blood resuscitation and chest tube placement. Surgical intervention is required in but a few patients.

## **Appendix 4.1: Applied anatomy**

### **Chest wall**

The chest wall consists of a bony skeleton and associated soft tissue that acts as a cage to protect the thoracic and upper abdominal contents. The bony skeleton comprises of 12 semicircular ribs on either side, which are attached posteriorly to the thoracic vertebrae, and anteriorly by the costal cartilages to the sternum, except for ribs 11 and 12 that are free anteriorly. The sternum consists of a short upper manubrium, a larger body, and a small xiphoid process. The sternum protects the heart and the great vessels that lie directly posterior to it while giving attachment to the various neck, abdominal and pectoral muscles. The skeletal elements are covered with muscles, fascia, subcutaneous tissue and skin. Overlying the upper ribs are the clavicles and scapulae. The inside of the thoracic cavity is lined by the parietal pleura. The neurovascular bundle lies along the lower border of each corresponding rib, a fact that is important to remember when inserting a chest drain in order to avoid unnecessary injury. The area inferior to the axillae, usually the 5<sup>th</sup> intercostal space and anterior to the mid-axillary line, is the thinnest portion of the chest wall and an ideal site for insertion of chest drains.

A fracture of the 1<sup>st</sup> or 2<sup>nd</sup> ribs usually indicates significant energy transfer, for example a fall from height or high-speed impact. This should alert the trauma team to look for other associated injuries. When several ribs are fractured in two places, the chest wall will show *paradoxical movement* during respiratory cycle, i.e. the broken section will be pulled inwards during inspiration and bulge out during expiration. This is called a *flail segment* and will result in inadequate ventilation. There is usually an associated underlying lung contusion and the combination of inadequate ventilation and lung injury may lead to severe hypoxaemia and hypercarbia.

### **Diaphragm**

This is the main muscle of respiration and consists of a central tendon with radially orientated muscle fibres forming two domes (right and left) or hemidiaphragms. Several structures traverse the diaphragm; the aorta, the thoracic duct, the azygos vein, the oesophagus, the vagus nerves and their branches and the vena cava. During normal quiet breathing, the diaphragm moves about 1.5-2 cm but this can increase to 10 cm during deep breathing. During deep exhalation, the dome of diaphragm can ascend to the 5<sup>th</sup> intercostal space, an important point to remember while inserting chest drains. This also explains diaphragmatic and intra-abdominal injuries following penetrating injuries high on the chest wall. Injuries of the liver and spleen in particular should be considered in the presence of lower rib fractures.

## **Trachea and bronchi**

The trachea extends from the cricoid cartilage, at the level of 6<sup>th</sup> cervical vertebra to the carina. On each side lie the jugular veins, common carotid arteries and the vagus nerves. It bifurcates at the level of lower border of 4<sup>th</sup> or upper border of 5<sup>th</sup> thoracic vertebra, into the main bronchi, the right being shorter, straighter and less angulated in relation to the trachea than the left. This is why a tracheal tube will tend to enter the right main bronchus when advanced too far. The right main bronchus lies posterior to the right main pulmonary artery while the left main bronchus is adjacent to the aortic arch, posterior to the left atrium. The trachea is loosely fastened in the neck and superior mediastinum and can be displaced laterally by external pressure.

## **Lungs and pleurae**

The right lung has three lobes; upper, middle and lower, divided by transverse and oblique fissures. The left lung has two lobes, upper and lower, divided by the oblique fissure. Both lungs are divided into broncho-pulmonary segments corresponding to the bronchial branches of the right and left pulmonary arteries. Both lungs are drained by superior and inferior pulmonary veins.

The surfaces of lungs are lined by the visceral pleura and in the healthy state there is only a potential space between the visceral and parietal pleura. However, as a result of the opposing forces of the chest wall (pulling out) and the elastic recoil of the lungs (pulling in), there is a negative pressure in this potential space that prevents the lung from collapsing. If there is a break in either pleural membrane, then air and/or fluid can enter the cavity and a 'space' is created. If air enters the cavity a *pneumothorax* exists. If a 'one way valve' forms into the pleural cavity, then air enters but cannot leave and results in a potentially life threatening condition of *tension pneumothorax*. Accumulation of blood in the pleural cavity is called a haemothorax.

## **Mediastinum**

This contains all the thoracic viscera except the lungs and extends from the spine posteriorly to the sternum anteriorly, with the heart occupying the middle mediastinum. The heart is invested by the pericardium, a tough, non-elastic membrane that is attached to the diaphragm inferiorly and extends along the two branches of the pulmonary artery as well as the ascending aorta. If blood collects between the pericardium and heart, the chambers of the heart are compressed and cardiac output is reduced (cardiac tamponade).

The thoracic aorta is divided into an ascending part, the arch and the descending part. The main branches of the aorta are brachiocephalic (innominate) artery, which divides into right

subclavian and right common carotid arteries just behind the right sternoclavicular joint, the left common carotid and left subclavian arteries which arise further downstream (separately) from the arch just before it becomes the descending aorta. The descending part continues downwards, behind the heart to pass through the diaphragm into the abdominal cavity.

The oesophagus lies in the posterior mediastinum and extends from the pharynx in the neck to the gastro-oesophageal junction and is extra pleural. The thoracic duct ascends through the aortic hiatus into the thorax and lies on the right side of the vertebral bodies. It empties into the venous system at the junction of the left subclavian and internal jugular vein.

The major consequences of thoracic trauma are pulmonary and cardiac dysfunction resulting in hypoxaemia and decreased cardiac output respectively. Jointly or separately, they cause a reduction in oxygen delivery to the tissue, hypoperfusion, organ failure and death. For clarity the two disturbances are considered separately, but there is considerable overlap and disturbance of one system may profoundly affect the function of the other.

## **Appendix 4.2: Pathophysiology**

The main functions of the lungs are oxygenation of blood and removal of carbon dioxide. To achieve this:

- Adequate air has to reach the alveoli (ventilation)
- An adequate circulation is required around the alveoli (perfusion)
- Transfer of gases must occur in both directions between the alveoli and blood (diffusion)

If there is no problem with diffusion, oxygenation of the blood is primarily a function of the inspired oxygen concentration and removal of carbon dioxide is a function of alveolar ventilation. Any disruption one or more of these processes can lead to hypoxaemia or hypercarbia.

### **Ventilation**

The amount of air taken into the lungs with each breath is the tidal volume ( $V_T$ ) and during quiet periods of breathing, is normally about 7-8 ml/kg (about 500 ml in a 70 kg adult). The volume of air inspired into (or expired from) the lungs each minute is the minute volume and under normal circumstances is approximately 7 litres/minute (500 ml/breath at a rate of 14 breaths/min).

Not all of the tidal volume takes part in the respiratory gas exchange. Only about 70% of  $V_T$  (350 ml) reaches the alveoli; the rest fills the mouth, trachea and the bronchi. This is called the dead space or more accurately *anatomical dead space*, as it is not involved with gas exchange. Failure of gas exchange also occurs in areas of lung distal to the bronchi that are ventilated but not perfused with blood. When added to the anatomical dead space, this is called the *physiological dead space*. In health, these are almost identical as most areas of the lung are both ventilated and perfused. The volume of ventilation that actually takes part in the gas exchange is called *alveolar ventilation*, and is approximately 5 litres/min (350 ml x 14 breaths/min).

At the end of a normal expiration there is a considerable volume of gas remaining in the lungs, termed the *Functional Residual Capacity (FRC)*. This is the result of the two opposing forces between the chest wall (outwards) and the lung (inwards). Approximately 15% of the FRC is replaced with fresh gas during each tidal volume breath. The FRC acts as a reservoir, keeping alveoli open at the end of expiration and ensuring that sudden changes in gas composition within the lungs and blood are avoided.

### **Perfusion**

The lungs are perfused (Q) with approximately 5 litres blood per minute. The pulmonary circulation is a low-pressure system and as a consequence, gravity influences the flow of

blood in different parts of the lungs. In turn this effects oxygenation of the blood circulating in that part. In the erect position, apical alveoli are less well perfused than basal alveoli, giving rise to dead space (see above). In turn the basal alveoli are overperfused in relation to ventilation and this leads to some of the blood not taking up oxygen nor giving up carbon dioxide. This latter effect is termed *shunting* (the blood is literally shunted past alveoli with no exchange) and in health this amounts to less than 5% of the cardiac output.

## Diffusion

Gas exchange occurs by diffusion a process dependent upon:

- Partial pressure gradient of the gas
- Solubility of the diffusing gas
- The thickness of this membrane
- Area of the membrane across which diffusion is taking place

**The partial pressure of a gas is dependant on the percentage it contributes to a mixture, multiplied by the total pressure. For example, oxygen comprises 21% of the atmosphere (pressure 100 kPa), therefore the partial pressure of oxygen in air is  $21/100 \times 100 = 21$  kPa.**

Gases diffuse down a partial pressure gradient (ie from high to low). As the partial pressure of oxygen in the alveoli is high and in the pulmonary capillary blood low, oxygen diffuses from alveoli to blood. Conversely, carbon dioxide diffuses from blood to alveoli. However, carbon dioxide is 20 times more diffusible than oxygen so despite a lower gradient, diffuses across the alveolar-capillary membrane far more rapidly.

The alveolar-capillary membrane is ideally suited for diffusion as it is only 0.0005 mm thick and has a large surface area  $50\text{ m}^2$ . It follows then, that an increase in the thickness of this membrane (e.g. pulmonary oedema) or a reduction in its size (collapse, consolidation, pneumothorax etc) will impair diffusion, particularly of oxygen and cause hypoxaemia.

## Ventilation/perfusion ratio

In health, ventilation ( $V$ , 5l/min) and perfusion ( $Q$ , 5l/min) are well matched (despite minor variations at the extremes of the apices and bases) and the ventilation/perfusion ratio ( $V/Q$ ) is approximately 1. Oxygen in the alveoli diffuses into blood, saturating all the haemoglobin molecules and as a result, the partial pressure of oxygen in the blood ( $\text{PaO}_2$ ) leaving the lungs is approximately 13 kPa. In addition a very small amount of oxygen is dissolved in the plasma.

If ventilation exceeds perfusion, for example in shock (low cardiac output,  $V/Q > 1$ ), there is not enough blood circulating to accept all the oxygen available. Oxygen is “wasted” as once the haemoglobin is fully saturated the arterial content cannot be increased any further, except for a small increase in oxygen directly dissolved in plasma.

Conversely, when perfusion exceeds ventilation for example a lung contusion (reduced ventilation,  $V/Q < 1$ ) there is an inadequate amount of oxygen to fully saturate the haemoglobin. Consequently there is a reduced oxygen content in the blood leaving these areas of the lung. This has the same effect as “shunting” described earlier, that is the blood seems to ‘have bypassed’ the lung without being oxygenated.

After trauma areas of normal, high and low V/Q co-exist in the lungs and the final oxygen content of arterial blood depends upon the combined influence of all three. The small rise in oxygen dissolved in plasma from areas of high V/Q cannot compensate for the larger deficit in the oxygen content from areas of low V/Q. As the latter tend to predominate this results in hypoxaemia. Once 30% or more of the blood in the pulmonary circulation passes through area of low V/Q, the hypoxia cannot be corrected by simply increasing the oxygen content of the inspired gas. Not surprisingly, V/Q mismatch is responsible for many deaths in trauma victims.

$PaO_2$  - partial pressure of oxygen in arterial blood is determined by the partial pressure of oxygen in the alveoli ( $PAO_2$ ). The relationship between the  $PAO_2$  and the fractional inspired oxygen concentration ( $FIO_2$ ), is described by the alveolar gas equation. As can be seen from the equation, the  $PAO_2$  is always slightly less than the inspired partial pressure of oxygen due to the presence of water vapour and carbon dioxide in the alveoli.

$$PAO_2 = FIO_2 (P_b - P_{H2O}) - PACO_2/R$$

$PAO_2$  = Alveolar oxygen pressure

$FIO_2$  = Inspired oxygen fraction

$P_b$  = Atmospheric pressure

$P_{H2O}$  = Water vapour pressure

$PACO_2$  = Alveolar  $CO_2$  pressure

R = Respiratory quotient

When breathing air at an atmospheric pressure of 100 kPa (760 mmHg, sea level), the partial pressure of inspired oxygen is 20.9 kPa. As the air moves through the respiratory tract, it becomes fully saturated with water vapour and the partial pressure of oxygen falls to 19.5 kPa. At the alveolus, oxygen is taken up and replaced by  $CO_2$ , which reduces the  $PAO_2$  to 14.5 kPa. Under normal circumstances, the  $PaO_2$  is always slightly lower than that the  $PAO_2$ .

due to physiological shunt. When breathing air, the normal  $PaO_2$  is 12.5 kPa at the age of 20 years and 10.8 kPa at 65 years due to the normal effects of aging.

An estimate of the  $PAO_2$  can be made as follows:

$$PAO_2 = \text{inspired } pO_2 - PACO_2 / R$$

If we assume that R is constant at 0.8, then:

$$PAO_2 = \text{inspired } pO_2 - PACO_2 \times 1.2$$

As  $PACO_2$  is approximately equal to  $PaCO_2$ , then:

$$PAO_2 = \text{inspired } pO_2 - PaCO_2 \times 1.2$$

The Alveolar-arterial (A-a) gradient

This is the difference between the calculated  $PAO_2$  and the measured  $PaO_2$  (A-a gradient = alveolar  $pO_2$  - arterial  $pO_2$ ). It provides a means of determining if the  $PaO_2$  is appropriate for the inspired oxygen concentration. As can be seen above, the  $PAO_2$  is normally 2-4 kPa higher than the  $PaO_2$ . If the difference is greater than this it implies an impairment of oxygenation and in the trauma patient this is usually due to ventilation/perfusion mismatch.

### **Appendix 4.3: Lung protective ventilation strategy**

Patients suffering thoracic trauma are at risk of Acute Lung Injury (ALI) and Acute Respiratory Distress Syndrome (ARDS). There are strict definitions for the diagnosis of ALI and ARDS. The triad of hypoxia, low lung compliance and widespread infiltrates on CXR should be accompanied by a known precipitant of the syndrome and a normal left atrial pressure estimated from the pulmonary artery occlusion pressure (PAOP). A  $\text{PaO}_2 / \text{FiO}_2 \leq 40 \text{ kPa}$  (300 mmHg) defines ALI and a  $\text{PaO}_2 / \text{FiO}_2 \leq 27 \text{ kPa}$  (200 mmHg) defines ARDS.

Patients suffering severe trauma and thoracic trauma are at high risk of developing respiratory failure with both ALI and ARDS. The principles of mechanical ventilation are to provide adequate oxygenation and  $\text{CO}_2$  removal while minimising the risk of barotrauma and volutrauma. Cyclical over distension and collapse of alveoli induces damaging shear forces and increases levels of systemic inflammatory cytokines. The use of low tidal volumes and moderate positive end expiratory pressure (PEEP) will reduce these shear forces and minimise damage. A protective ventilation strategy using low tidal volume and low inspiratory plateau pressure improves survival in patients with ARDS. The use of low tidal volumes may cause hypercarbia that may be problematic in patients with head injury. A popular, pragmatic approach is to use tidal volumes of  $6-8 \text{ ml kg}^{-1}$  predicted body weight and to keep the plateau pressure  $\leq 35 \text{ cm H}_2\text{O}$ . If chest wall compliance is poor, for example, in the presence of severe oedema or abdominal distension, it is appropriate to allow the plateau pressure to increase. This is because the amount of stretch applied to the lungs is dependent on the transpulmonary pressure and not simply the inspiratory pressure. Recruitment manoeuvres, such as continuous positive airway pressure of  $40 \text{ cm H}_2\text{O}$  applied for 40 secs, will inflate collapsed dependent alveoli (recruitment) and a PEEP of  $5-15 \text{ cm H}_2\text{O}$  will help to maintain inflation. Prolonged inspiratory times (i.e., inverse ratio ventilation) will increase intrinsic PEEP ( $\text{PEEP}_i$ ) and oxygenation; however, there is a risk of over inflation of the lungs and the impairment to venous return can reduce cardiac output dramatically. Prolonged use of a high, inspired oxygen concentration is thought to induce lung injury. The  $\text{FiO}_2$  should be reduced to  $\leq 0.5$  as soon as possible by setting targets for acceptable oxygenation. The optimum level of PEEP in relation to the  $\text{FiO}_2$  is unknown. In the early stages of ARDS, ventilation in the prone position improves oxygenation but this is usually impractical in the trauma situation. Extracorporeal membrane oxygenation with  $\text{CO}_2$  removal (ECMO) has been used with anecdotal benefit in trauma patients but the requirement for anticoagulation is often prohibitive.

Management strategies for these patients should begin **upon arrival** at the trauma centre/emergency department by initially identifying who is most likely to develop severe respiratory insufficiency. The goal is to institute therapies early (eg. “open lung” or

“protective” lung ventilation) in the emergency room, operating room and in the intensive care unit in an effort to lessen the degree or to prevent the formation of atelectasis and /or parenchymal ventilator associated damage to the lung.

The following general guidelines should be applied:

- Limit mechanical ventilation tidal volumes to 6 – 8 ml/kg
- Apply PEEP (at least 5 cm H<sub>2</sub>O) to keep the ‘lung open.’ Measurement of optimal PEEP level (with reference to the lower inflection point of pressure volume curves) is impractical in initial management.
- Limit peak/plateau airway pressure to < 35 cm H<sub>2</sub>O
- Adjust I:E ratio and respiratory rate as needed to achieve above
- Wean FiO<sub>2</sub> to obtain oxygenation saturation of >93%
- Early conversion to pressure-limited modes of ventilation

## **Further reading**

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## Chapter 5

# SHOCK

### Objectives

At the end of this chapter the reader should understand:

- The definition and classification of shock
- The causes of shock
- How to estimate the degree of hypovolaemia
- The initial assessment and management of the shocked patient

Cardiovascular physiology, the cellular effects of shock, invasive monitoring, the use of crystalloids and colloids and intravenous access are covered in Appendices 4.1 – 4.5 respectively at the end of the chapter.

### DEFINITION AND CLASSIFICATION OF SHOCK

Shock is an expression used frequently but inexactly by lay people and the media. Medically, the term is defined as inadequate oxygen utilization by organs/tissues and it is in this restricted sense that the word is used throughout this book.

In shock there is a miss-match between the delivery of oxygen to cells and what they need. Typically we think of a state of poor delivery due to blood loss but it is important to be aware that shock can exist when the oxygen delivery has increased but not enough to cope with the rise in cellular consumption (e.g. following sepsis). This inadequate oxygen utilization can be global or affect only specific organs.

Shock results from a number of diverse pathophysiological cardiovascular disorders with distinctive causes (Box 5.1). When shock is due to major trauma, cardiac output is usually decreased, but it may be normal or even increased in neurogenic and septic shock.

Regardless of the cause there is impaired oxygen utilization, that will eventually lead to cellular hypoxia, dysfunction and cell death if left untreated. The key aim for the clinician is to detect and treat the condition as soon as possible so that the cell death can be minimised.

#### Box 5.1: Classification of shock

Type	Examples of causes	Effect on cardiac output
Hypovolaemic	Haemorrhage, dehydration	Decreased
Cardiogenic	Acute myocardial infarction, myocardial	Decreased

	contusion,	
Neurogenic	High spinal cord injury (usually above T5)	Decreased
Septic	Pneumonia, bowel perforation, as a late complication of major trauma, delayed resuscitation	Normal or increased initially
Anaphylactic	Acute allergic reaction (type I hypersensitivity)	Decreased

Often the trauma patient will have shock due to a number of causes; for example, following a traffic accident, the victim may have both hypovolaemia as a result of blood loss and a tension pneumothorax, reducing venous return. However, after trauma, shock usually has a hypovolaemic component. This is the most easily managed and should be identified and treated before shock is attributed to any other causes.

#### Hypovolaemic shock

This is by far the commonest cause of shock in trauma patients. Injury disrupts the vasculature within soft tissues (e.g. muscles), solid organs (e.g. liver) and bones and results in haemorrhage. Venous return to the heart is reduced decreasing cardiac output and, depending on the volume lost, arterial pressure. In addition to haemorrhage, the intravascular volume is also depleted as a result of leakage of plasma into the interstitial spaces and this can account for up to 25% of the circulating blood volume reduction following blunt trauma. Burns can lead to greater leaks and so larger losses of circulating volume.

A tension pneumothorax or cardiac tamponade have a similar effect on the circulation in that they both impair venous return to the heart by a pressure effect on the right ventricle. In both cases however there may be relatively little loss of the circulating blood volume.

The normal blood volumes in adults and children are shown in Box 5.2

#### **Box 5.2: Blood volumes**

- Adult: 70 ml per kilogram ideal body weight (approximately 5l in 70 kg person)
- Child: 80 ml per kilogram ideal body weight

#### **Cardiogenic shock**

Cardiogenic shock may be due to myocardial trauma and/or ischaemia. The compensatory sympathetic response is often ineffective in restoring cardiac output as the dysfunctional left

ventricle is unable to increase its contractility and arterial blood pressure falls despite the development of a tachycardia. Attempts to maintain arterial blood pressure with a low cardiac output occur by a progressive increase in the SVR. Unfortunately, both a tachycardia and increased SVR increase myocardial oxygen demand and a vicious circle develops with further myocardial ischaemia and dysfunction occurring. Additionally each time the tissues are subjected to hypoxia / hypoperfusion with cellular destruction, mediators are liberated into the circulation increasing the risk of SIRS (Appendix 5.2). Cardiac failure is therefore frequently complicated by autonomic, endocrine and inflammatory responses.

In patients with pre-existing ischaemic heart disease or poor cardiovascular reserve, and in all patients in the advanced stages of sepsis, the situation is aggravated by toxins exerting negative inotropic effects on the myocardium. The relatively high cardiac output typical of septic shock is now compromised and a vicious cycle develops, accelerating the demise of the patient.

### **Neurogenic shock**

The sympathetic outflow comes from the spinal cord between the levels T1-L3. The vasoconstrictor supply to the blood vessels arises from all these levels, and the heart receives its sympathetic innervation from levels T1-T4. A spinal cord injury will impair the sympathetic outflow below the level of the injury, the higher the lesion, the more pronounced the disturbance. Lesions above T4 will result in generalised vasodilatation (reduced SVR), at the same time denervating the heart and preventing any increase in rate to try and maintain cardiac output. The clinical picture is one of hypotension, systemic vasodilatation and a relative bradycardia.

### **Estimating volume of blood loss**

Estimating blood loss is difficult. It must be remembered that haemorrhage can be overt, when it is often overestimated, or concealed and usually underestimated. Concealed haemorrhage occurs into the cavities of the thorax, abdomen and pelvis, or into potential spaces, for example the retroperitoneal space, muscles and tissues around long bone fractures. The greater the volume lost, the more severe the state of shock. The standard method for estimating the degree of hypovolaemia is to observe the changes in a number of physiological parameters:

- heart rate
- blood pressure
- respiratory rate
- capillary refill time
- skin colour

- urine output
- conscious level.

Haemorrhage results in a sympathetically mediated response aimed at maintaining blood flow and oxygenation of the vital organs (heart, brain, kidneys etc). The classical clinical picture of a shocked patient is one who is pale with cool peripheries, a tachycardia, and tachypnoea. If haemorrhage continues, the blood pressure will fall along with a reduction in urine output and level of consciousness. However, these observations are based upon the changes seen when young, healthy animals are subject to controlled haemorrhage under strict laboratory conditions. The closest we come to seeing this response in trauma patients is the young, fit adult with isolated penetrating trauma with little tissue damage. More frequently, the patient has blunt trauma, is anxious, frightened and in pain, all of which may modify the physiological response. The situation may be further complicated by the presence of drugs or pre-existing comorbidites, particularly in the elderly, that modify their ability to respond to haemorrhage. These factors may lead to a serious risk of over or underestimation of blood loss (Box 5.3). Management of the shocked patient must therefore take into account the type and nature of injuries sustained, the individual characteristics of the patient being treated, and their response to treatment.

#### *Tissue damage*

The sympathetically induced circulatory response to hypovolaemia described above are markedly affected by the degree of tissue damage. With minimal tissue damage (e.g. a stab wound to a major vessel) the tachycardia and vasoconstriction (also referred to as the systemic vascular resistance, SVR) continue up to the point where around 30% (1.5l) of the circulating blood volume has been lost. Continued blood loss further reducing venous return leads to stimulation of the cardiac C-fibre reflex. This inhibits the vasomotor centre resulting in a vagally mediated reduction in heart rate and loss of sympathetic tone (particularly in skeletal muscle & kidneys). As a consequence, there is a profound fall in blood pressure. The reason for the need for such a reflex is not clear, but it is strongly suspected that it may have evolved to protect the body against excessive blood loss by reducing the pressure and thereby minimising the risk of dislodging fresh clots that have formed at the point of injury. Patients with isolated penetrating trauma therefore demonstrate the signs of shock with relatively small haemorrhages.

In contrast, when there is predominantly tissue damage (e.g. blunt trauma with long bone fractures) the cardiac C-fibre reflex is impaired. This results in prolonged sympathetic stimulation and marked vasoconstriction in non-essential organs for example the gut and kidneys. Consequently, the blood pressure is maintained despite a greater loss of the

circulating blood volume, even more than 40% (21). These patients will therefore compensate with relatively larger haemorrhages and so demonstrate the signs of shock relatively late.

**Box 5.3: Patients with a risk of over- or underestimation of blood loss**

Type of patient:

- Elderly (decreased cardiovascular reserve)
- Drugs/pacemaker
- Pregnancy
- Athlete

Environment/pre-hospital:

- Hypothermia
- Delay in resuscitation
- Type of injury

*The elderly*

The elderly usually have a reduced cardiorespiratory reserve and are less able than a younger (fitter) trauma victim to compensate for acute hypovolaemia. Blood pressure therefore falls at smaller volumes of blood loss. Consequently reliance on blood pressure alone can lead to an overestimation of blood loss. Patients with a low fixed cardiac output (eg aortic stenosis) behave similarly. As a corollary to this, it should also be noted that very young patients will compensate for hypovolaemia extremely well and hypotension is a late sign and presages impending cardiovascular collapse.

*Drugs and pacemakers*

Various medications alter the physiological response to blood loss, a good example being  $\beta$ -blockers. Even after losing over 15% of the circulating volume, these drugs may prevent the development of a tachycardia and also inhibit the normal sympathetic response. This can lead to underestimation of blood loss if relying on heart rate alone. Similarly, hypotension will develop with loss of smaller volumes of blood because of the impaired compensation. Many illicit drugs can also affect or mask the normal physiological response (e.g. cocaine). An increasing number of patients have pacemakers fitted. Depending on their complexity and sophistication, these devices may only pace the heart at a constant rate (approximately 70-100 beats per minute), irrespective of volume loss or arterial blood pressure. Therefore they may give rise to errors in estimation of acute blood loss.

*The pregnant or athletic patient*

The pregnant patient will undergo a variety of physiological changes which may complicate the assessment of blood loss including, increased blood volume, faster heart rate and respiratory rate (Chapter 11). The resting heart rate in a trained athlete may be less than 50 beats per minute. Therefore a compensatory tachycardia indicative of significant acute blood loss may be less than 100 beats per min. An increase in blood volume of 15-20% as a consequence of training may constitute a further possible reason for underestimation of blood loss.

#### *The patient with hypothermia*

Hypothermia (core temperature < 35°C) will reduce arterial blood pressure, pulse and respiratory rate in its own right, irrespective of any blood loss. If this is ignored, hypovolaemia may be overestimated. It has also been found that hypovolaemic, hypothermic patients are often 'resistant' to appropriate fluid replacement. Estimation of the fluid requirements of these patients may therefore be very difficult and invasive haemodynamic monitoring is often required (Chapter 15).

#### *Delay in resuscitation*

The longer the time the patient spends without resuscitation (especially in the young), the longer the normal compensatory mechanisms will have to work. This will lead to improvements in blood pressure, respiratory rate and heart rate. Underestimation of blood loss may then occur.

### **Assessment and management of the shocked patient**

Patients cannot remain indefinitely in a state of shock, they either improve or die. For the latter shock can be viewed as "a momentary pause on the way to death", a pause that gives the trauma team time and opportunity to prevent further deterioration and death of the patient. As previously stated, the state of shock is defined as inadequate utilization of oxygen by the tissues. It therefore follows that the detection of this condition is dependent on monitoring parameters which are produced as a result of poor oxygen utilization. Furthermore the treatment of shock consists of restoration of an adequate delivery of oxygen and not simply the restoration of a normal blood pressure.

### **Primary survey and resuscitation**

The same plan described in Chapter 1 is used, with members of the team carrying out their tasks simultaneously. The priorities are; to clear and secure the patient's airway, ensure adequate ventilation with a high inspired oxygen concentration while at the same time, the spinal column in general, and the cervical spine in particular, are immobilized if the mechanism of trauma suggests the potential for injury. At the same time, the breathing

personnel should exclude, or if present treat, any immediately life-threatening thoracic injuries. If the patient is shocked, it is presumed to be due to hypovolaemia until proved otherwise. The circulation personnel are responsible for stopping any overt bleeding by direct pressure and inserting two large bore peripheral IV lines (14 or 16g). Short, wide cannulae should be used as flow is inversely proportional to length and directly related to radius (Box 5.4).

**Box 5.4: Relationship between cannula length, radius and flow**

Cannula size	Flow rate (ml/min)
14g short	175-200
14g long	150
16g short	100-150
16g long	50-100

Immediately following successful venous cannulation, 20 ml blood is taken for estimation of serum electrolytes, glucose, full blood count (FBC), grouping /cross-matching and pregnancy test (in females of appropriate age). At the same time, basic monitoring should start and a record of the vital signs made (Box 5.5).

A central vein is commonly used in adults, if peripheral venous access is unavailable or fails. Occasionally it may be necessary, depending upon skills and facilities of the team, to obtain intraosseous access or perform a venous cut-down. These techniques are covered in Appendix 5.4

**Box 5.5: Vital signs that must be monitored in trauma patients**

- Respiratory rate
- Peripheral oxygen saturation
- Heart rate, arterial blood pressure, pulse pressure
- Capillary refill time
- Urine output
- Glasgow Coma Scale score
- ECG via chest leads (rhythm and waveform)
- Temperature, core and peripheral

By the time the cannulae are in place, the team leader should have quickly assessed the patient to try and differentiate the main cause of shock and the possibility of any confounding issues. Patients can initially be categorised into one of three main groups:

## **1. Penetrating trauma**

Patients with penetrating trauma and minimal tissue damage (e.g. a stab wound), usually have ongoing haemorrhage into a body cavity that cannot be controlled externally. They rapidly become tachycardic, hypotensive, pale, cold and sweaty with a gradual reduction in urine output and conscious level. Once more than 30% blood volume is lost, there may be a slowing of the heart rate, especially when bleeding is intraperitoneal. At this point the patient will develop cardiovascular collapse and cardiac arrest (PEA) is imminent. Although aggressive resuscitation with rapid infusion of a large volume of fluid tends to raise arterial pressure, there may be adverse effects including dislodgement of thrombus and a dilutional coagulopathy. These lead to further haemorrhage necessitating even greater fluid resuscitation – a vicious circle develops making optimisation of such patients difficult, if not impossible. The priority in these patients is *emergency surgical or radiological haemostasis*. Very few or even no investigations are required and therefore the period of severe shock before control of bleeding is usually of short duration. Therefore, providing that there is no head injury, fluid resuscitation prior to any intervention should be limited to achieving an arterial blood pressure sufficient to maintain organ viability in the short term. This is now regarded as being a systolic pressure around 70mmHg.

## **2. Blunt trauma**

This is most often soft tissue and/or bony injury. Patients with this type of trauma therefore usually require radiological investigations (x-rays, CT scan) and delays of 1-2 hours before surgery are not uncommon. As discussed previously this group of patients are able to compensate to a much greater degree by diverting blood from non-essential organs to maintain perfusion of the vital organs. The systolic blood pressure is therefore often well maintained despite the loss of 30% of the blood volume (Box 5.6). This can lead to underestimation of the severity of shock, despite the fact that these patients still appear pale and have cool peripheries. Consequently, the presence of shock in this group of patients needs more active treatment in order to limit cellular damage and prevent the later development of SIRS and MOF, in a manner that causes no harm. Intravenous fluids and possibly blood are therefore usually required, the question is, “how much?”. Too little fluid is associated with high mortality rates, but too much fluid also increases mortality. This is a result of; a dilutional coagulopathy, particularly when excessive volumes of crystalloids are given, and exceeding a blood pressure threshold beyond which bleeding increase as the consequence of loss of vasoconstriction and disturbance of clots. This appears to be at a systolic blood pressure of around 90mmHg . As a result several guidelines now recommend an upper limit of 90mmHg as a guide to fluid resuscitation in blunt trauma patients without head trauma.

### **3. Head injury**

Patients with isolated traumatic brain injury (TBI) represent another group of patients where different specific goals are now used to guide resuscitation efforts. Some patients will present with hypertension and a relative bradycardia. This elevation of the blood pressure is an indication of raised intracranial pressure and an attempt by the body to maintain cerebral perfusion. No action should be taken to lower it. Isotonic fluids should be given at a normal maintenance rate.

### **The complex patient**

The problem arises when a patient has both TBI and penetrating or blunt trauma. The decision as to the resuscitation regime used will depend upon the speed with which bleeding can be arrested. Where there is ongoing, significant, uncontrollable haemorrhage, it is reasonable to aim for a systolic blood pressure of around 90mmHg as mortality is significantly increased in patients with TBI who have even brief periods below this level. The aim should then be to achieve surgical control of the bleeding in the shortest time possible. Once achieved, the resuscitation of the TBI takes precedence and the blood pressure can be increased. Although a precise target for blood pressure in patients with TBI has not yet been defined, the Brain Trauma Foundation Guidelines and the European Guidelines for the treatment of severe BTI recommend a higher blood pressure than for other types of injury. Most authorities now agree that in patients with TBI, resuscitation should aim for a systolic blood pressure of 110 mmHg.

<b>Box 5.6: Comparison of physiological changes in blunt and penetrating trauma</b>	Approximate volume of blood loss by % total blood volume	
Physiological changes	Penetrating trauma With little tissue damage	Blunt trauma
Heart rate < 100/min		
Blood pressure normal		
CRT < 2 sec	<15% TBV	15-30% TBV
Respiratory rate normal		
Urine output normal		
Fully orientated		
Tachycardia 100-140/min		
Systolic BP reduced		
CRT > 3 sec		
Tachypnoea	15-30% TBV	30-40% TBV

Reduced urine output		
Obtunded		
Tachycardia >140/min or bradycardia		
Severe hypotension, SBP<70mmHg		
CRT absent		
Tachypnoea/gasping respiration	30-40% TBV	>40% TBV
Anuria		
Unconscious		

### Type of fluid for resuscitation

While blood and colloids once infused remain mainly in the vasculature, isotonic crystalloids diffuse throughout the extracellular space and as a result less than 1/3 remains in the intravascular compartment. Consequently, much greater volumes need to be given than the volume of blood lost, generally 3-4 times as much. Hypotonic solutions (e.g. glucose 5%) diffuse into both the extracellular and intracellular spaces, and little remains intravascular. Furthermore, by entering the intracellular space, this causes or exacerbates cellular swelling, a particularly dangerous situation in patients with TBI as it will lead to increases in intracranial pressure.

Although there is currently little evidence of superiority between crystalloids and colloids, the most recent Cochrane review demonstrated a trend towards a lesser mortality in patients with TBI treated with crystalloids. Clearly the type of fluid or colloid used will be dictated by local policy. Further details are given in Appendix 5.4.

### **How much fluid?**

This will depend on the predominant type of injury and the derangement of physiological signs or severity of shock. As can be seen in Box 5.6, patients with blunt trauma will require a greater volume of fluid for any given degree of shock. They are also more likely to require greater volumes of fluids because the target blood pressure is relatively higher and blunt trauma is associated with a greater degree of tissue oedema, fluid that is effectively “lost” from the extracellular (intravascular) space.

Whichever fluid is given, crystalloid or colloid, it should be warmed before administration to prevent iatrogenic hypothermia. A simple way of achieving this is to store them in a warming cupboard, thereby eliminating the need for warming coils which increase resistance to flow and slow the rate of fluid administration. In appropriate situations an alternative is using a level 1 device which can deliver large volumes of warm fluid rapidly.

Typically fluids are given to shocked adult patients as boluses of 500-1000ml depending on the severity of shock and the response of the patient assessed. Because crystalloids are predominantly the first fluids used, it is not unusual for patients with a loss of up to 30% of their blood volume to require 3-4 l depending on the target pressure.

Red cell replacement is a secondary consideration, becoming more important with progressively larger blood loss (remember the advantageous effect of a reduced haematocrit on blood viscosity and flow). In the majority of trauma cases who require blood in the resuscitation room, type-specific blood is used, i.e. the recipient and donor blood are checked for ABO and Rhesus compatibility. Most laboratories can provide this within 10 minutes. Occasionally, exsanguinating haemorrhage will require immediate administration of blood. In these cases, uncrossmatched blood (O negative) is used initially until typed blood is available. These patients rapidly develop a coagulopathy both as part of the response to injury and also as a result of the dilution of clotting factors by administered fluids, the release of tissue factors and minimal amounts of clotting factors in stored blood. It is now recognised that platelets and clotting factors should be given early in conjunction with the use of packed red blood cells (PRBCs). A typical regime is given in Box 5.7.

#### **Box 5.7: Massive haemorrhage transfusion policy**

In major trauma patients with one or more of:

- Loss of one whole blood volume
- ISS >30
- PT/APTT 1.5 x control:
  - Give 1 unit FFP ± platelets per unit PRBCs
- When PT>18, APTT>55 and bleeding:
  - Give 1.5 units FFP per PRBCs

Ensure that hypothermia is corrected

By following the guidelines above the aim is to ensure that by a combination of the body's compensation and the resuscitation performed, the function of vital organs will be maintained. However patients are often complex, they may not fit neatly into the categories described or they may not respond as predicted. As a result, there is an increasing trend to monitor markers of cellular hypoxia (Appendix 5.3) including; serum lactate, pH, base excess and, in patients with invasive monitoring, oxygen extraction ( $SvcO_2$ ). Early detection and intervention helps to guide therapy and minimise the degree of shock and cellular damage.

## **Measuring blood pressure**

Although non-invasive methods of measuring blood pressure are widely used, they are increasingly unreliable at low pressure and in patients with severe vasoconstriction. Consequently, there is an increasing use of invasive arterial pressure monitoring at an early stage in the resuscitation of trauma patients. Not only does this allow more accurate and continuous monitoring of the patient's blood pressure, it also allows repeated sampling of blood for various investigations, in particular acid-base balance and blood gas analysis.

Accurate measurement of urine volume will obviously require the insertion of a urinary catheter with the volume recorded whenever the other vital signs are measured.

The rest of the primary survey is completed as previously described in Chapter 1. At the end, the team leader must ensure that the required tasks have been or are being carried out. An arterial blood sample may be sent at this stage. Metabolic acidosis is invariably a result of anaerobic metabolism in poorly perfused tissues. Appropriate management consists of increasing cardiac output by fluid administration, optimising  $\text{PaO}_2$  and ensuring that the patient is not hypercapnic (increased  $\text{PCO}_2$ ) to ensure adequate delivery of  $\text{O}_2$  to the tissues. Sodium bicarbonate is reserved for cases of immediately life-threatening acidosis where the pH is less than 7.0 and adequate perfusion is assured and ventilation guaranteed. In such cases it is preferable that the patient is intubated and ventilated to assist in elimination of  $\text{CO}_2$  via the lungs and prevent a respiratory acidosis.

The principles of treating sepsis syndrome and SIRS are the provision of support for failing organ systems (e.g. optimisation of oxygenation of arterial blood, mechanical ventilation, fluid resuscitation, inotropic and vasopressor drugs, haemofiltration, clotting factors), and targeting any infecting organism with appropriate antibiotic therapy and/or surgical drainage. This may require repeated blood cultures and analysis of specimens of sputum, urine, wound and catheter sites to identify the organism responsible. If a collection of pus within the thorax, abdomen, pelvis or elsewhere is a clinical possibility, it must be sought using ultrasound scanning, CT scan or laparotomy. Such patients do not recover if surgical drainage is not undertaken.

## **Secondary survey**

After the detailed head to toe assessment of the patient has been carried out, the team should have a reasonable estimation of the blood loss and its source. They should also know the patient's allergic history, current medication, past medical history, time of last meal and the mechanism of injury (remember 'AMPLE').

Pain relief is usually necessary to relieve suffering, increase the patient's ability to compensate for any hypovolaemia and to decrease myocardial workload by reducing catecholamine secretion. Care is required in doing this because the analgesia can block any neuro-adrenergic mechanism present compensating for the shock. As a result hypoxia and hypotension can increase following analgesia unless preventative steps are taken. A diluted opioid analgesic may be given intravenously, until satisfactory analgesia is achieved (e.g. morphine in 1-2 mg increments). An appropriate dose of an antiemetic agent should also be given. There is a wide therapeutic dose range for analgesic drugs amongst patients, depending on their age, premorbid fitness, comorbidity and physical status post-injury. Consequently, a wide dose range may be required to achieve satisfactory analgesia (Chapter 16). Analgesia should never be given by the intramuscular route as initially there is only limited systemic uptake due to the poor perfusion of the patient's muscles. Subsequently, once perfusion has improved after resuscitation, a large bolus of opioid analgesia may be absorbed rapidly into the bloodstream with profound effects on conscious level, respiration and arterial blood pressure.

In the time it takes an efficient trauma team to reach this stage in the resuscitation, the first fluid challenge will have been completed. The original estimated blood loss can be compared with the patient's response to the fluid volume provided. Essentially, there are three possible outcomes:

**(1)The patient is improving**

This suggests that the intravascular volume deficit is less than 20 - 30% and that the rate of fluid input is greater than the rate of fluid loss. Such patients may require blood later but one can afford to wait for a full crossmatch. Vital signs should be monitored closely and the team leader informed of any sudden deterioration (see below).

**(2)The patient initially improves, then deteriorates**

In these cases the rate of bleeding has increased, either because of a new source of bleeding or loss of haemostasis at the original site. The latter may occur with the rise in blood pressure following resuscitation. The majority of these patients will require surgery or interventional radiology and early involvement of the appropriate clinical team. Blood is also required, the choice being between typed or uncrossmatched, unless fully crossmatched blood has already been prepared. The decision will depend on the clinical state of the patient (as above).

### **(3)The patient does not improve**

These patients are either bleeding faster than blood or other fluids are being supplied or they are not suffering from hypovolaemic shock alone. The former group patients will have lost over 40% of their blood volume and therefore require urgent surgery, or embolisation, with ongoing fluid resuscitation.

An alternative explanation that must always be considered is other co-existing causes of shock; cardiogenic, neurogenic or septic, either alone or in combination with hypovolaemia. Aspects of the history, examination and vital signs are essential to distinguish between these possibilities.

### **Cardiogenic shock**

Obstructive causes of shock, such as cardiac tamponade and tension pneumothorax should be rapidly excluded because these conditions can quickly kill the patient (Chapter 4). If a cardiac cause for the shock is suspected, it is essential to discover the past medical history and current medication. In addition to the more usual signs of shock, there may be evidence of chest trauma, dysrhythmias, crackles on auscultation of the chest or a raised CVP suggested by engorged jugular veins. These patients are also less able to compensate for any hypovolaemia and their management is complex. Early involvement of the Intensive Care team is essential as more accurate haemodynamic assessment is usually required (Appendix 5.3). Depending on the system used, the filling pressure of the left side of the heart or cardiac output can be estimated. Along with a combination of mechanical ventilation, vasodilators, inotropes and expansion of circulating volume, cardiac output and oxygen delivery can be manipulated to satisfactory levels.

### **Neurogenic shock**

Conscious patients with neurogenic shock will have a history and physical findings suggestive of spinal cord damage (Chapter 8). It is more difficult in unconscious trauma victims as the neurological information is often insufficient to reliably exclude spinal cord damage. In these cases a high level of suspicion, and proper image studies, should be considered until spinal damage and neurogenic shock have been excluded. It is important that patients with neurogenic shock are neither under- nor over-transfused. The former may lead to poor perfusion of the spinal cord and exacerbate injury, the latter to pulmonary oedema. In patients with no previous heart or lung disease, the CVP and LVEDP have a close correlation. Therefore in the early stages, CVP measurement will be useful in estimating fluid requirements and response to treatment. However, the patient may require more intensive and accurate fluid monitoring at a later stage on the ICU.

## **Septic shock**

The more profound and prolonged the tissue hypoxia and destruction (e.g. necrosis, crushing) the bigger the probability of a strong systemic response. The dominant clinical picture is manifestations attributable to vasodilatation, generalized oedema, signs of uncoupling of oxygen delivery and consumption (rising lactate and falling SvcO<sub>2</sub>) and impaired organ function. These mechanisms are early contributors to the distributive component of shock.

In the early phase of shock, compensation is heralded by autonomic response, a profile similar in response to that of hypovolaemia. However as soon as reperfusion takes place, the inflammatory mediators are liberated and contribute to the clinical picture with a wide pulse pressure and warm skin due to dilated peripheral blood vessels. Cardiac output may be in the normal range or raised. The patient is often agitated, pyrexial and hypoxic due to the development of acute respiratory distress syndrome (ARDS). Coagulopathies such as disseminated intravascular coagulation may be seen and frequently complicated by massive blood loses (coagulation factors consumption and coagulation factor dilution). This abnormality may be life-threatening and manifests initially as blood oozing from wounds and cannula sites.

Trauma patients with delayed resuscitation, massive tissue damage or being transferred from another hospitals with bowel perforation are all at risk of developing a systemic inflammatory response syndrome (SIRS), severe sepsis or even septic shock. The management of these patients is generally the domain of the Intensivist, but the Trauma Team should be able to recognise the signs and symptoms of septic shock to allow them to participate in the care of patients who may arrive in the resuscitation room following transfer from another hospital.

## **Summary**

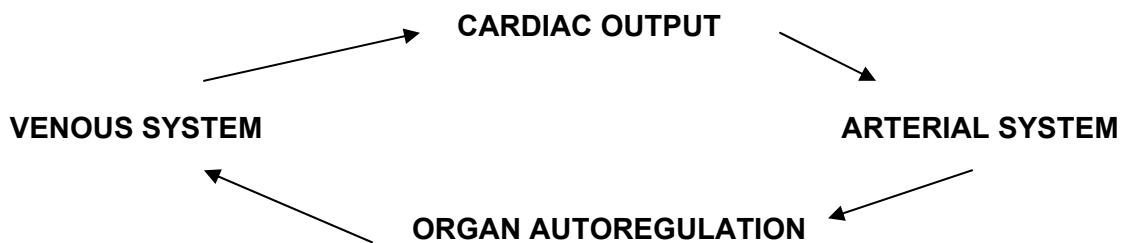
All members of the trauma team must recognize and initiate treatment in shocked patients as early as possible. In trauma cases the cause of the shock is frequently multifactorial but will invariably have a hypovolaemic component. Tissue hypoxia is minimised by early assessment, constant monitoring and appropriate interventions. Regular reassessments is also important because any subsequent deterioration needs to be detected quickly and treated. In addition as the patient improves, other problems may become apparent.

## **Appendix 5.1: Cardiovascular physiology**

To appreciate the rationale for treatment of the shocked patient, it is necessary to understand the normal physiological control of cardiac output, arterial blood pressure and regional organ perfusion. For the reader who is particularly interested, the formulae for calculating the various physiological variables discussed are listed in Appendix 5.1 at the end of this chapter.

### **Circulatory control**

Optimal organ perfusion is dependent upon the following system being intact:



### **Venous System**

The main determinants of venous return to the heart are blood volume and venous tone. The venous system is capable of acting as a reservoir for over 50% of the circulating blood volume and is therefore often referred to as a capacitance system. The amount of blood stored at any one time is dependent on the size of the vessel lumen. This is controlled by sympathetic innervation and local factors (see later) which can alter the tone of the vessel walls. If the veins dilate, more blood remains in the venous system and less returns to the heart. Should there be a requirement to increase venous return, sympathetic stimulation increases, reducing the diameter of the veins and the capacity of the venous system. A change from minimal to maximal tone can increase the venous return by approximately 1 litre.

**The main physiological determinants of venous return to the heart are blood volume and venous tone.**

### **Cardiac Output**

Cardiac output (CO) is the product of the volume of blood ejected with each heartbeat (stroke volume, SV) and heart rate (beats per minute). It is expressed in litres per minute:

$$\text{Cardiac Output} = \text{Stroke Volume} \times \text{Heart Rate} (\text{l min}^{-1})$$

To enable meaningful comparisons between patients of different sizes, CO can related to body surface area. This is termed the cardiac index (CI) and is measured in litres per min per m<sup>2</sup>:

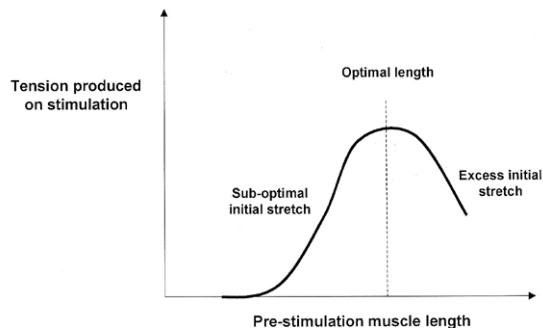
$$CI = CO / \text{body surface area} (\text{l min}^{-1} \text{m}^{-2})$$

### Factors affecting stroke volume

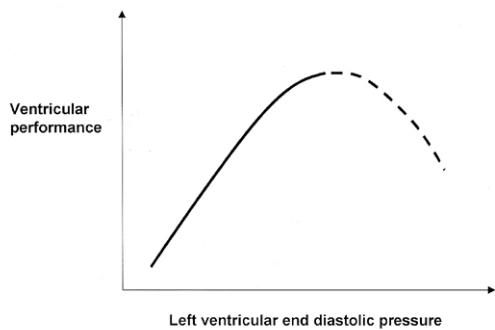
The three principle determinants of stroke volume are; (i) preload, (ii) myocardial contractility, (iii) afterload.

#### (i) Preload

This is the force with which a muscle contracts is dependent on its resting length (Fig. 5.1); the more it is stretched beyond its resting length, the more forcefully it contracts. This also applies to the myocardial muscle fibres, the force of contraction is dependent on the degree of stretch during diastole by the blood returning from the venous circulation. The greater the stretch during diastole, the more forcibly they will contract during systole. The result is more blood is expelled into the systemic and pulmonary circulations providing that the optimal level is not exceeded (Starling's Law, Fig. 5.2). Clearly, it is not possible to measure the length of individual myocardial fibres. Instead an estimate may be obtained by measuring pressure at the end of diastole because in the normal heart there is close relationship between the two. In routine clinical practice central venous pressure (CVP) gives an estimate of right ventricular end diastolic pressure (RVEDP), and pulmonary artery occlusion pressure (PAOP), or wedge pressure, an estimate of left ventricular end diastolic pressure (LVEDP).



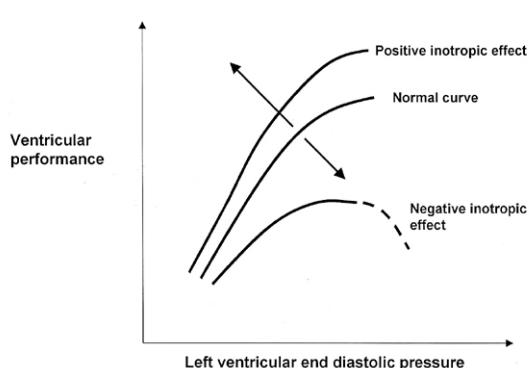
**Fig. 5.1: Effect of increasing resting length on tension during contraction for a single muscle fibre.**



**Fig. 5.2:** Starling's Law expressed as the effect of increasing LVEDP on ventricular performance (cardiac output).

### (ii) Myocardial contractility

Substances that increase myocardial contractility for a given resting length (proportional to end diastolic volume or pressure) are termed positive inotropes. This action can be represented as shifting the Starling curve to the left (see Fig. 5.3). Stimulation by the sympathetic nervous system has a positive inotropic action, as does circulating catecholamines such as adrenaline and dobutamine. Many drugs e.g. anti-dysrhythmics, anaesthetics, sedatives, severe hypoxia and acidosis have a negative inotropic effect.



**Fig. 5.3:** Effect of positive and negative inotropes on Starling curve.

### (iii) Afterload

This is the resistance faced by the ventricular myocardium to ejection of the blood. It is significantly different for both ventricles:

- Left ventricular afterload is mainly due to the resistance offered by the systemic arterial blood vessels (and aortic valve) and is termed the systemic vascular resistance (SVR).
- Right ventricular afterload is mainly due pulmonary blood vessels (and pulmonary valve) and is termed the pulmonary vascular resistance (PVR).

When afterload is reduced (e.g. following arterial vasodilatation) and preload maintained, the ventricles contract more quickly and extensively and thereby increase stroke volume and cardiac output. A good example of this is seen in the patient with the early phase of septic

shock. Patients are pink, warm (vasodilated and normovolaemic) with a bounding pulse because of the increased cardiac output.

### **Factors affecting heart rate**

Increases in heart rate are mediated by the sympathetic nervous system both directly and indirectly. It is termed a positive chronotropic effect. Conversely, the parasympathetic nervous system (via the vagus nerve) causes a decrease in heart rate, a negative chronotropic effect. In the resting state, the rate averages 60-100 beats per minute, depending on the age and physical fitness of the patient and is usually subject to dominant vagal activity.

An increase in heart rate usually increases CO (see above). However, the increased rate occurs largely as a result of shortening of diastole, during which ventricular filling (and perfusion of the myocardium) occurs. If heart rate continues to increase, diastole is eventually reduced to the point that filling is compromised and stroke volume and cardiac output decrease. Similarly, as heart rate decreases, there comes a point where no more filling can occur and CO falls. In a “normal”, fit individual, stroke volume is relatively unaffected between 40 and 150 beats per minute. This range may be considerably reduced by age, disease and medication so that a heart rate well within this range may not be tolerated. In summary the main factors affecting the cardiac output of the left ventricle are listed in Box 5.8.

#### **Box 5.8: Factors that affect left ventricular cardiac output**

Preload (or LVEDP)

Myocardial Contractility

Afterload ( or SVR)

Heart Rate

### **The arterial system**

#### *Systemic arterial blood pressure*

This is the pressure within the arterial blood vessels. Systolic pressure is the maximum pressure generated in the large arteries during each cardiac cycle and diastolic pressure is the minimum. The pulse pressure is the difference between systolic and diastolic pressures and mean arterial pressure (MAP) is the average pressure during the cardiac cycle. It is approximately equal to the diastolic pressure plus one-third of the pulse pressure. MAP is the product of cardiac output and systemic vascular resistance and is therefore affected by all the factors discussed above.

## **MAP = CO x SVR**

Arterial blood pressure is normally tightly controlled by neural, humoral and metabolic mechanisms to maintain adequate perfusion pressure and blood flow to organs and tissues. Following a fall in arterial blood pressure a number of mechanisms operate to restore pressure including:

- Baroreceptors located in the aortic arch, carotid sinus and heart. These send impulses to the vasomotor centre within the brainstem, increasing sympathetic activity. This leads to a rise in heart rate, contractility and arteriolar and venous constriction
- Selective arteriolar and pre-capillary sphincter constriction in non-essential organs (eg skin, gut). This helps to maintain perfusion of vital organs (eg brain, heart)
- Reduction in renal blood flow is detected by specialised cells within the juxtaglomerular apparatus of the kidney releasing rennin which leads to the formation of angiotensin II (a vasopressor and stimulator of aldosterone production)
- Aldosterone secreted from the adrenal cortex and antidiuretic hormone (vasopressin) released from the pituitary, increase reabsorption of sodium and water by the kidney, reducing urine volume to help maintain the circulating volume

In addition, insulin and glucagon are released which promote the supply and utilisation of glucose by the cells. The liver also attempts to enhance circulating volume by releasing osmotically active substances that increase plasma oncotic pressure, thus reducing the osmotic gradient causing extravasation of fluid from the circulation through leaky capillaries. As a result, cardiac output and SVR rise in an attempt to restore arterial blood pressure back towards its homeostatic set point.

Normally, an isolated fall in either CO or SVR results in an increase in the other to maintain arterial blood pressure. However, if hypovolaemia leads to a marked fall in CO, arterial blood pressure will eventually fall despite sympathetic activity causing positive inotropic and chronotropic effects on the heart and vasoconstriction. The clinical picture is of a pale, cold and clammy patient with a weak or absent peripheral pulse.

**Following major trauma arterial blood pressure will fall even though increased sympathetic activity is causing positive inotropic and chronotropic effects on the heart and vasoconstriction. Clinically, the patient is pale, cold and clammy with a weak or absent peripheral pulse.**

## **Organ autoregulation**

Blood flow through many organs, particularly the kidneys and brain remains almost constant over a range of blood pressures as a result of a process termed autoregulation. It is due to relaxation of the smooth muscle within arteriolar walls and precapillary sphincters. This allows the arteriolar diameter to increase, and cellular flow to be maintained, as the pressure falls. Furthermore, accumulation of metabolic products such as H<sup>+</sup> ions, CO<sub>2</sub> and hypoxia cause a direct relaxant effect on the arterioles. Although not often a problem in the trauma patient, hypertension causes vasoconstriction in an attempt to protect the organs from high pressure.

## **Oxygen delivery**

The delivery of oxygen to the tissues is critical if shock is to be prevented. It is dependent on:

- A. transfer from pulmonary alveoli into blood flowing through the pulmonary capillaries
- B. transport in the blood to the tissues
- C. release from the blood to the tissues.

### **A. Transfer from alveoli to blood**

The factors affecting this, namely ventilation, perfusion, diffusion and V/Q ratios have already been discussed in Chapter 4.

### **B. Transport to the tissues**

The amount of oxygen transported from the lungs to the tissues in arterial blood (DO<sub>2</sub>) is dependant on the oxygen content of arterial blood (CaO<sub>2</sub>) and the cardiac output. It is normally 500-720 ml min<sup>-1</sup> m<sup>-2</sup>:

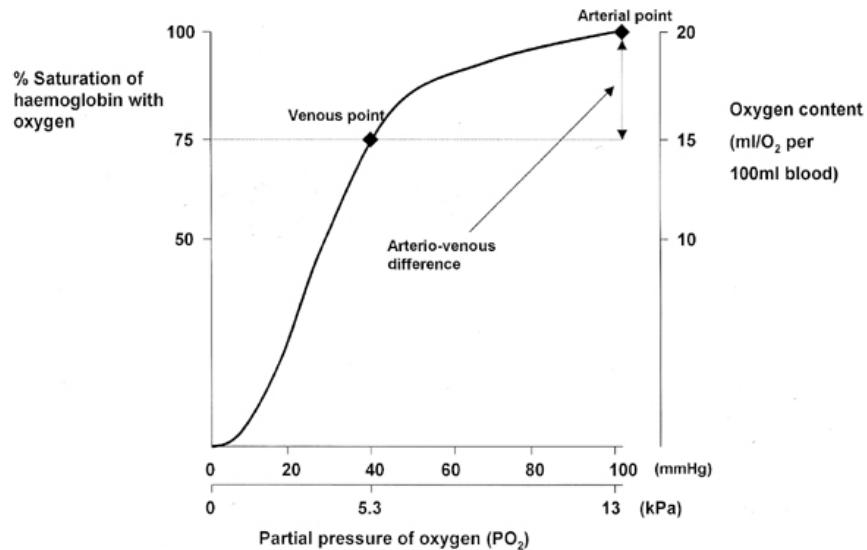
$$DO_2 = CaO_2 \times CO$$

#### *(i) Oxygen content of arterial blood (CaO<sub>2</sub>)*

The vast majority (98.5%) of oxygen is carried in the blood reversibly bound to haemoglobin. The rest is dissolved in plasma. The oxygen content of arterial blood (CaO<sub>2</sub>) is therefore dependent on the haemoglobin concentration and its saturation with oxygen. The relationship between the PaO<sub>2</sub> and oxygen uptake by haemoglobin is not linear but assumes a sigmoid curve (Fig. 5.4), with haemoglobin virtually fully saturated at a PaO<sub>2</sub> of 13 kPa (100 mmHg). Increasing the PaO<sub>2</sub> further therefore has little effect on oxygen transport. At a PaO<sub>2</sub> below 8 kPa (60 mmHg) saturation falls steeply, rapidly reducing content. The affinity of haemoglobin for oxygen at a particular PaO<sub>2</sub> is decreased by an acidosis (increased H<sup>+</sup> conc), raised PCO<sub>2</sub> and pyrexia. Remember, during exercise, active muscles require more oxygen than when at rest. Due to the increased metabolism they generate lactic acid, CO<sub>2</sub> and heat all of which will assist in the release of oxygen from haemoglobin. Conversely, the

opposite of these factors increases the affinity and shifts the curve to the left; an alkaline environment (decreased  $H^+$  conc), decreased  $PCO_2$ , a low concentration of red cell 2, 3 diphosphoglycerate (as found in transfused blood), and a fall in temperature.

Although increasing haemoglobin increases the oxygen carrying capacity of blood, this will also increase blood viscosity. In turn this impedes blood flow, increases myocardial workload and SVR, thus negating any advantage. The normal haemoglobin concentration is somewhat above the point for optimal oxygen transport so a modest reduction will reduce viscosity and myocardial work, improve flow through the microcirculation and therefore increase oxygen delivery. It is certainly seldom necessary to strive for a haemoglobin concentration above 100 g  $l^{-1}$  (haematocrit 30%) during resuscitation and levels as low as 8 g  $l^{-1}$  are often well tolerated.



**Fig. 5.4: The oxyhaemoglobin dissociation curve.**

(ii) Cardiac output has already been discussed.

### C. Oxygen release to the tissues

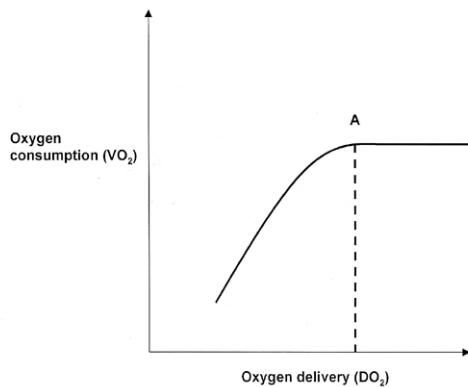
At tissue level, there is a partial pressure gradient driving oxygen from capillaries to cells:

- The  $PaO_2$  of blood at the proximal end of capillaries is approximately 13 kPa
- At the distal capillaries the  $PO_2$  has equilibrated with the interstitium which is approximately 4.3 kPa
- Intracellular  $PO_2$  normally averages only 3-3.5 kPa and may fall as low as 0.8 kPa
- A  $PO_2$  of 0.1-0.6 kPa is required for full support of all oxidative intracellular metabolic processes

Any metabolic activity that increases demand for oxygen results in local changes that act to allow  $O_2$  to be released more readily (shifting the curve to the right).

### Oxygen consumption

In the normal resting subject, total consumption of oxygen per minute ( $VO_2$ ) is 100-160 ml  $min^{-1} m^{-2}$  and remains constant over a wide range of oxygen delivery (normal value of  $DO_2$  500-720 ml  $min^{-1} m^{-2}$ , Fig. 5.5). Though oxygen uptake by different organs varies, overall only 20-25% of the oxygen delivered is taken up. This is termed the oxygen extraction ratio (OER). Clearly under normal circumstances, there is potential for the tissues to extract more oxygen from the circulating blood if required.



**Fig. 5.5: Relationship between oxygen delivery and consumption**

Trauma results in an early increase in oxygen consumption, despite the fact that the delivery of oxygen falls because of a reduction in haemoglobin and cardiac output (see above). Initially, the increase in consumption is achieved by increasing the extraction of oxygen, but this mechanism only operates while the delivery of oxygen is greater than approximately 300 ml  $min^{-1} m^{-2}$ . Below this rate the tissues cannot increase oxygen extraction any further because oxygen extraction is at its maximum. Oxygen consumption is therefore limited because it is now directly dependent on the rate of oxygen delivery to the tissues.

## **Appendix 5.2: Cellular effects of shock**

Normally after injury there is a proportional, compensatory anti-inflammatory response. However this balance can be lost in severe, repeated or prolonged shock. It is also now realised that certain people are more genetically predisposed to manifest these effects. This imbalance can lead to an exaggerated response, called a Systemic Inflammatory Response Syndrome (SIRS).

Shock can give rise to SIRS because ischemia, and reperfusion of previously ischaemic tissue, cause the release of toxic mediators (e.g. cytokines, complement, kinins, prostaglandin, leukotrienes and free radicals). In addition there can be translocation of gut flora into the circulation due to breakdown of the normal gastrointestinal mucosal barrier following splanchnic vasoconstriction.

In SIRS there is co-existing inflammatory and anti-inflammatory responses. This leads to further endothelial damage, disseminated intravascular coagulation, microvascular disturbances and accumulation of tissue leucocytes. These changes cause stagnation of blood flow, sludging of red cells and a further impairment of tissue perfusion. In addition, the hydrostatic pressure within the capillaries increases because blood can still perfuse the capillaries but cannot escape. As a consequence, further intravascular fluid is lost as it diffuses through the leaky capillary wall into the interstitial space. Generalized oedema is a good clinical marker of the presence of an inflammatory response and increased capillary permeability.

These changes compromise the oxygen delivery to cells. Cellular oxygen consumption can be further reduced by mitochondrial dysfunction. The end result of all these derangements is imbalance between oxygen supply and demand, anaerobic metabolism and lactic acidosis.

The body tries to reduce tissue damage by inactivating some of the unnecessary cells by a mechanism known as apoptosis (programmed cell death). This mechanism is not responsible for the increment of the inflammatory response but the presence of cell destruction (necrosis) is. Necrosis plus ischemia and reperfusion are major causes of cell, tissue and organ parenchyma destruction and the development of multiple organ failure (MOF). This condition is one of the commonest causes of late death after trauma. There is also an increase susceptibility to infection.

<p><b>The likelihood of MOF occurring is increased if resuscitation and correction of circulatory shock is inadequate or delayed.</b></p>
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When a sufficient cell mass has been damaged, the shocked state becomes irreversible and death of the patient is inevitable. Fortunately the body has several compensatory mechanisms that control the circulation in an attempt to maintain adequate oxygen delivery to the essential organs of the body and help prevent this stage being reached. The compensatory response is mediated by a number of mechanisms including; neurogenic (autonomic nervous system), endocrine (renin, angiotensin, vasopressin, catecholamines) and inflammatory (cytokines, NO, free radicals etc as already described above).

As a result of these neural effects cardiac output and SVR rise in an attempt to restore arterial blood pressure back towards its homeostatic set point. In addition sympathetic stimulation of arterioles supplying skeletal muscle actually causes vasodilatation (acting through  $\beta_2$  receptors), an appropriate response for “fight or flight”

### **Appendix 5.3: Invasive monitoring**

Traditionally to measure many of the indices below would require the insertion of a multi-lumen, pulmonary artery floatation catheter (PAFC, or Swan-Ganz catheter). However, minimally invasive methods of monitoring the cardiac output are being developed, examples being:

- Trans-oesophageal Doppler probe: flow through a cylinder (aorta) is proportional to its cross sectional area and the velocity of the fluid. The cross-sectional area of the aorta is estimated using a nomogram based on the patient's age, weight and gender. The velocity of blood flow along the aorta is determined according to the Doppler shift that occurs as ultrasound waves from the probe are reflected back from the moving bloodstream, hence stroke volume and cardiac output can be monitored.
- PiCCO: this uses a combination of transpulmonary thermodilution and arterial pulse contour analysis to assess cardiac function (output) and volume status.
- LiDCO: this calculates the power of each heart beat and calibrates this against the actual stroke volume as derived from an independent indicator dilution using lithium chloride. Stroke volume, cardiac output and systemic vascular resistance can be monitored.

#### **Cardiac output (CO)**

The CO is measured by a PAFC using a thermodilution technique (an application of the indirect Fick principle) by rapidly injecting 10 ml of cold crystalloid solution into the right atrium via a proximal lumen of the PAFC. This causes a reduction in blood temperature, monitored at the tip of the catheter by the thermistor. The reduction in temperature is inversely proportional to the extent of dilution of the injectate which is itself directly proportional to the CO. More sophisticated (and expensive) PAFCs are now available that use a variation of the thermodilution principle to provide a continuous readout of CO. The distal portion of the catheter proximal to the thermistor is surrounded by a heating coil that warms the blood slightly as it flows past, causing the temperature detected by the thermistor to rise.

#### **Left ventricular end diastolic pressure (LVEDP)**

The LVEDP cannot be measured directly and is estimated from the pulmonary artery occlusion or wedge pressure (PAOP). This is the pressure at the tip of the PAFC with the balloon inflated and wedged against the walls of the pulmonary artery. As distal flow is interrupted, there is a direct communication between the tip and the left atrium. At end diastole, the pressure within the left atrium approximates to the LVEDP which itself is usually an accurate reflection of left ventricular (LV) preload. Normal values range between 5-15 mmHg.

#### **Systemic vascular resistance (SVR)**

The SVR is a derived variable and not directly measured. It is calculated from the mean arterial pressure (MAP), the central venous pressure (CVP) and the cardiac output (CO) (Box 5.9). It is usually increased in hypovolaemic and cardiogenic shock and decreased in septic, anaphylactic and neurogenic shock.

#### **Box 5.9: Calculating the systemic vascular resistance**

MAP = Diastolic pressure + 1/3 (systolic pressure – diastolic pressure) (Approx)

SVR = (MAP-CVP) x 80/CO (normal range = 800-1400 dyne/s/ cm<sup>5</sup>)

#### **Oxygen content of arterial blood**

Accurate measurement of haemoglobin oxygen saturation ( $SaO_2$ ),  $PaO_2$  and haemoglobin concentration [Hb] are needed to calculate the oxygen content of arterial blood ( $CaO_2$ ), which includes both oxygen bound to haemoglobin plus that dissolved in plasma.

The amount bound to Hb = [Hb] (g/dl) x  $SaO_2$  (expressed as a decimal fraction) x 1.34

(when fully saturated, 1g Hb binds 1.34 ml O<sub>2</sub>)

The amount dissolved in plasma =  $PaO_2$  x 0.003

(ie 0.003 ml/dl oxygen dissolves in plasma for each mmHg  $PaO_2$ .)

Therefore:

$$CaO_2 \text{ (ml/dl)} = ([Hb] \text{ (g/dl)} \times SaO_2 \times 1.34) + (PaO_2 \times 0.003)$$

**It can be seen that the amount of oxygen dissolved in plasma physiologically is only 1-2% of the total and is relatively insignificant.**

#### **Delivery of oxygen**

The delivery of oxygen to the tissues ( $DO_2$ ) is dependent on the content of oxygen in the blood ( $CaO_2$ ) and the rate at which blood is reaching the tissues (CO).

$$DO_2 \text{ (ml/min)} = CO \times CaO_2 \times 10 \quad (\text{normal value } 500-720 \text{ ml/min})$$

The factor of ten is needed to express  $CaO_2$  in units of ml/l.

$DO_2$  is often indexed to body surface area in the same way as CO when it is designated  $DO_{2 \text{ INDEX}}$ .  $DO_{2 \text{ INDEX}}$  is calculated from the CI and  $CaO_2$

#### **Consumption of oxygen**

In order to assess the oxygen consumption ( $VO_2$ ) of the cells and tissues of the body, it is necessary to measure the amount of oxygen left in venous blood returning to the heart. If this is low (less than approximately 70%), it indicates that the tissues are extracting larger amounts of oxygen than normal from arterial blood as it passes through the capillary network

(ie the oxygen extraction ratio is high) from which it may be deduced that regional blood flow through the tissues is suboptimal.

The metabolic activity of organs and tissues differs and hence the oxygen extraction ratio varies. However, pulmonary artery blood is a homogenous mixture of the venous blood returning from all the organs and tissues and a sample of blood is taken from the distal lumen of a PAFC which lies in the pulmonary artery. This is often termed a mixed venous sample. Some modern types of PAFC incorporate a miniature oximeter within the tip of the catheter, thereby permitting a continuous readout of mixed venous oxygen saturation.

The oxygen content of a mixed venous blood sample ( $CvO_2$ ) is calculated using an analogous formula to that used to calculate the  $CaO_2$ :

$$CvO_2 \text{ (ml/dl)} = ([Hb] \text{ (g/dl)} \times S_vO_2 \times 1.34) + (P_vO_2 \times 0.003)$$

$S_vO_2$  and  $P_vO_2$  represent the oxygen saturation and oxygen partial pressure of the mixed venous sample.  $VO_2$  is also often indexed to body surface area in the same way as CO when it is designated  $VO_2$  INDEX.

A number of “targets” have been suggested as indicators of ensuring that adequate amounts of oxygen are being delivered and utilised in critically ill patients. These are shown in Box 5.10.

**Box 5.10: Optimal goals in securing adequate oxygen transport (normal ranges in brackets)**

Cardiac index (CI) > 4.5 l/min/m<sub>2</sub> (2.8-3.6 l/min/m<sub>2</sub>)

Oxygen delivery ( $DO_2$ ) > 600 ml/min/m<sub>2</sub> (500-720 ml/min/m<sub>2</sub>)

Oxygen consumption ( $VO_2$ ) > 170 ml/min/m<sub>2</sub> (100-160 ml/min/m<sub>2</sub>)

Pulmonary artery occlusion or wedge pressure (PAOP) 18 mmHg (5-15)

Mixed venous oxyhaemoglobin saturation ( $S_vO_2$ ) > 70% (70-75%)

Whole blood lactate concentration = <2 mmol/l (<2 mmol/l)

## **Appendix 5.4: Colloids versus crystalloids**

Much has been written about which type of fluid is most appropriate in treating shocked patients. Advocates for colloids argue that rapid replacement of intravascular volume is of primary importance. The proponents of crystalloids consider that fluid is required to restore the deficit from the entire extracellular space (e.g. intravascular and interstitial spaces).

### **Colloids**

Colloid solutions are usually isotonic and can be used to replace an intravascular loss up to one litre, on a 1:1 basis. Greater degrees of blood loss usually require packed cells to be added so that the haematocrit does not fall below 30%. Colloids are either plasma derivatives (5% albumin and human plasma protein fraction (HPPF)) or plasma substitutes (gelatins, dextrans, hydroxyethyl starches).

The two gelatin preparations in common use are Haemaccel and Gelofusine. They are derived from alkaline hydrolysis of bovine collagen. The average molecular weight of the molecules is approximately 30-35 000 daltons and they have a half life within the circulation of two to four hours during which time the gelatin is eliminated completely by filtration in the renal glomeruli and hepatic metabolism. These fluids do not adequately replace the interstitial loss but they do produce less tissue oedema than crystalloids. However cardiac failure has been reported more often in patients receiving inappropriately large volumes of colloids. Haemaccel has a higher calcium and lower sodium concentration than Gelofusine and the former may therefore produce flocculation (clumping) of red cells if Haemaccel and blood are administered via the same giving set. Worldwide data shows that modified gelatins have a higher rate of allergic reaction than dextran.

Dextrans are polysaccharides produced with differing ranges of molecular weight, that is used to describe the solution, for example Dextran 70 (average molecular weight 70 000 daltons). This is the only type used for trauma resuscitation. Although the clinically effective intravascular half life of dextran 70 is about six hours, higher molecular weight components can be detected days or even weeks later. Dextran solutions also interfere with both crossmatching and coagulation due to effects on platelet function and fibrin formation. Although dextran diluted blood can still be used for crossmatching purposes, it is more time consuming for the laboratory and as a consequence, dextran solutions tend not to be used during resuscitation.

Another type of colloid solution are the hydroxyethyl starches. Hetastarch (Hespan, 6% starch in isotonic saline) has an average molecular weight of 450 000 daltons. Accordingly it has a much longer circulatory half life than the gelatins and the clinical effect may even

extend beyond 24 hours. Care must therefore be taken to avoid fluid overload when blood is added later to restore the haematocrit. Pentastarch differs from hetastarch only in its degree of hydroxyethylation. It is available as 6% and 10% solutions in normal saline and has an average molecular weight of 250 000 daltons.

Starch colloid solutions have a low incidence of acute allergic reaction.

### **Crystalloids**

The most commonly used crystalloid solutions are Hartmann's solution (Ringer's lactate) and 0.9%N (physiologically normal) saline. The former may be preferred because it contains a lower concentration of sodium and chloride ions and may therefore reduce the risk of producing hyperchloraemic acidosis in the shocked patient. Hartmann's solution is closer to the ionic composition of extracellular fluid. It contains lactate ions that are metabolised in the liver to produce bicarbonate, although this process may be inhibited in the shocked patient.

Both crystalloids have an intravascular half-life of only 30-60 minutes before they diffuse throughout the extracellular fluid compartment. Over 60% of the volume infused is taken up by the interstitium under normal conditions and this may be increased to 90% in the shocked patient. Consequently at least three times the estimated intravascular loss has to be infused as crystalloid to maintain intravascular volume. This becomes a major problem when there is large volume loss. It is difficult to infuse such large volumes of crystalloid quickly (>5 litres) and tissue oedema may result. This is of particular importance in acute brain and lung injury when further cerebral swelling or pulmonary oedema may be produced. Renal complications may also occur, particularly in elderly patients receiving large volumes of crystalloids. The advantages of using crystalloids over colloids are that they restore intracellular and interstitial fluid loss, they are cheap, convenient, have an extremely low incidence of allergic reactions and a long shelf life. Despite the problems associated with the use of large volumes of crystalloid, fluid resuscitation using only Hartmann's solution and blood is a technique commonly used in the United States.

Recently hypertonic-hyperosmotic crystalloid solutions have been advocated for initial resuscitation of hypovolaemia. Although it is suggested that they may be superior to isotonic crystalloid or colloid solutions, they are not in routine use. There is currently no evidence that they are significantly better clinically but they do cut down on the volume given and so may be of use when storage space is lacking – such as in ambulance transfers and military situations.

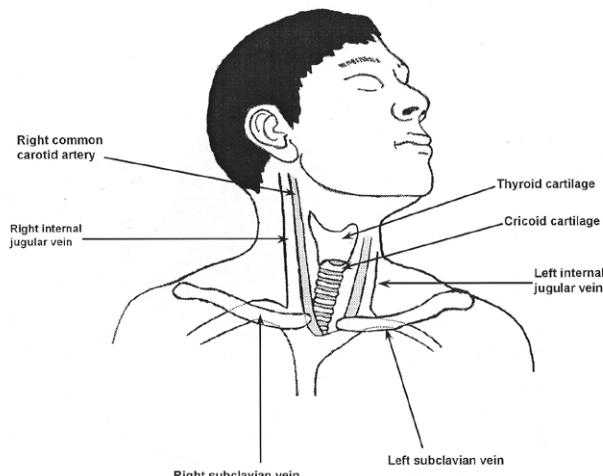
When deciding on fluid replacement, the most appropriate fluid for the affected body space should be chosen. Blood should be given as early as possible for patients with massive haemorrhage. For less severe haemorrhage, the intravascular volume and haematocrit can be maintained with colloids. (Crystalloid, in adequate volumes can be given as a substitute.) At a later stage, crystalloids will be needed to replace the interstitial loss.

## Appendix 5.5: Venous access

In adults, there if a peripheral site for venous access is not available then either a central vein or the intraosseous route can be used. For both, an aseptic technique must be used along with infiltration of local anaesthesia when appropriate.

### Central line

This technique involves the insertion of an appropriate cannula (14 or 16g) into a central vein, usually the subclavian, internal jugular or femoral vein, using the Seldinger technique (see below). The procedure should be carried out only by experienced staff because it has potential for damaging the vein and neighbouring structures. The anatomy of the central veins is shown in Fig. 5.6. A list of equipment that will need to be prepared is given in Box 5.11.



**Fig. 5.6: Anatomy of the internal jugular and subclavian veins.**

### Box 5.11: Equipment required for central venous cannulation

- Skin preparation solution
- Swabs
- Sterile sheets
- Sterile gowns and gloves for the nurse and doctor
- Local anaesthetic
- Syringe and needle for administering the anaesthetic
- Scalpel and blade
- Suture and sterile scissors
- Central line pack:
  - Syringe

- Large bore needle
- Guide wire
- Dilator
- Cannula
- Three way taps
- Giving set attached to intravenous fluid for infusion
- Opsite™ or other transparent adhesive sterile dressing
- Monitor and appropriate connecting tubing

### *The Seldinger technique*

Using a needle attached to a syringe, the central vein is initially punctured percutaneously, confirmed by the ability to aspirate blood. The syringe is removed, the flexible guide wire passed down the needle, 4-5 cm into the vein and the needle carefully withdrawn leaving the wire behind. The dilator is then loaded onto the wire and whilst holding the proximal end of the wire, advanced into the vein using a rolling or twisting motion. A small incision in the skin may be required to facilitate insertion of the dilator. The dilator is withdrawn leaving the wire in the vein and then the cannula is introduced into the vein in a similar manner. The wire is then removed, the syringe reattached and blood aspirated to confirm the cannula lies in the vein. If difficulty is encountered inserting the wire, the needle and wire must be withdrawn together to avoid damaging the wire on the needle tip.

### The subclavian vein

This vein can be cannulated via both the supra- and infraclavicular approach.

#### Relative contra-indications

Avoid using in cases of coagulopathy as compression of the vessel is not possible.

### Procedure

The following is a brief description of one of many approaches to the vein.

1. The patient is placed supine, arms at his side, head turned away and if safe 10° head down.
2. The operator stands on the same side as that to be punctured and identifies the midclavicular point and the suprasternal notch.
3. The needle is inserted 1 cm below the midclavicular point, advanced horizontally, postero-inferior to the clavicle towards the “tip” of a finger in the suprasternal notch, aspirating on the syringe.
4. When the needle tip enters the vein, usually at a depth of 4-6 cm, blood is easily aspirated, the syringe is removed and the cannula introduced as described above.

5. The cannula is secured, a sterile dressing applied and a chest x-ray taken to exclude a pneumothorax and confirm correct positioning of the cannula.

#### Complications

- Pneumothorax
- Haemothorax
- Puncture of the subclavian artery
- Injury to mediastinal structures
- Air embolism
- Infection
- Thrombosis

#### *Internal jugular vein*

Absolute contra indication

Patients with suspected neck injuries

Relative contra-indications

Avoid using in cases of head injury as cerebral venous return can be impaired leading to a further rise in ICP

#### Procedure

The following is a brief description of one of many approaches to the vein. The right side is usually chosen as there is a straight line to the heart, the apical pleura is not as high, and the main thoracic duct is on the left.

1. The patient is supine, head turned slightly away from the side of approach and if safe 10° head down.
2. The carotid artery is identified at the level of the thyroid cartilage with the tips of the fingers of the left hand.
3. With the fingers still marking the position of the artery, the needle is introduced 0.5 cm lateral to the artery, towards the medial border of the sternomastoid muscle, aspirating on the syringe.
4. When the needle tip enters the vein, usually at a depth of 2-3 cm, blood is easily aspirated, the syringe is removed and the cannula introduced as described above.
5. The cannula is secured, a sterile dressing applied and a chest x-ray taken to exclude a pneumothorax and confirm correct positioning of the cannula.
6. If the vein is not entered on first attempt, a further attempt can be made slightly more laterally.

#### Complications

- Haematoma
- Puncture of the carotid artery
- Pneumothorax
- Air embolism
- Infection
- Thrombosis

#### *Femoral vein*

Access to this vein may be easier during resuscitation, however sterility is more difficult to maintain. Because of the risk of deep vein thrombosis, the cannula should be used for the minimum time possible.

#### Relative contra-indications

Avoid using in cases of pelvic or severe lower limb trauma as interstitial leakage of the infusing fluid is likely

#### Procedure

- The patient is placed in a supine position and the inguinal ligament identified.
- If possible tip the patient 10-20° head up
- Locate the femoral artery just below the ligament.
- With a finger on the artery, the needle is introduced 1cm medially at an angle of 45° cranially, aspirating on the syringe.
- The vein is usually entered at a depth of 3-4 cm and the syringe is removed and the cannula introduced as described above.
- Secure the cannula and apply a sterile dressing.

#### Complications

- Haematoma
- Arterial puncture
- Deep vein thrombosis
- Infection
- Injury to the femoral nerve

#### **Intraosseous infusion**

This technique is carried out when it is not possible to cannulate a peripheral vein in a child and the expertise to cannulate a central vein is unavailable. It is simple to learn and has a

low incidence of complications. Osteomyelitis and local soft tissue infection may occasionally occur when the needle has been left in place for several days or a hypertonic solution has been infused.

Ideally a purpose-designed intraosseous infusion needle should be used, but spinal and bone marrow aspiration needles are suitable alternatives. Whichever type is available, the needle must have a trocar to prevent it becoming obstructed as it traverses the bony cortex. The commonest site for needle insertion is 2-3 finger breadths below the tibial tuberosity on the anteromedial surface of the tibia.

A leg without a fracture proximal to the insertion site is chosen and the site cleaned. The needle is then pushed into the bone at 90° to the skin's surface. Steady pressure is maintained until there is a sudden fall in resistance, indicating that the needle is in the bone marrow. This position must be checked by firstly removing the trocar and aspirating marrow and secondly, noting a free flow of fluid into the bone without the development of a visible subcutaneous leak.

The aspirated marrow should not be discarded but instead sent for blood typing. The choice and quantity of fluid needed to resuscitate children is described in Chapter 10.

## Websites

<http://www.ccmtutorials.com/cvs/index.htm>

Interactive shock tutorial with cases to work through.

<http://www.pacep.org/>

Pulmonary artery catheter resource with haemodynamic monitoring tutorials.

<http://www.americanheart.org/presenter.jhtml?identifier=9181>

The American Heart Association website scientific statements section has numerous up to date guidelines.

<http://www.blood.co.uk/start.html>

Facts about blood transfusion in the UK.

<http://www.shot.demon.co.uk/>

UK national database on serious hazards of transfusion (SHOT).

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## Chapter 6

# ABDOMINAL AND PELVIC TRAUMA

### **Objectives**

At the end of this chapter the reader should understand:

- The importance of suspecting intraabdominal injury
- The importance of the mechanism of injury
- The principles of assessment and management of abdominal and pelvic trauma

The clinical anatomy of the abdomen and pelvis are covered in Appendix 6.1 and interpretation of a pelvic x-ray in Appendix 6.2 at the end of the chapter.

### **Introduction**

The two main causes of death after major trauma are traumatic brain injury (TBI) and major haemorrhage. The latter is often the result of abdominal and or pelvic trauma and apart from the consequences of blood loss alone it will significantly worsen the outcome from other unrelated injuries, particularly traumatic brain injury. Perforation of hollow viscera and peritoneal contamination resulting in sepsis are important contributors to late deaths after abdominal trauma.

Even though diagnostic abilities have become more sophisticated throughout the past decade, undetected or underestimated abdominal injury still remains a leading cause of preventable death (Box 6.1). This is because clinical assessment of these patients is not always straightforward, for the following reasons:

- Pain and peritoneal irritation are not reliable indicators of intraabdominal injury. These are often masked by an alteration of mental state, consumption of drugs and/or alcohol and painful distracting injuries elsewhere. Many severely injured patients, even though fully conscious, fail to complain of pain, probably as a result of intrinsic protective mechanisms e.g. release of endorphins.
- Early clinical manifestation of intraabdominal bleeding and peritoneal contamination is often subtle. The abdominal wall, trunk and pelvic ring often show no significant signs of injuries. Nevertheless disruption of intraabdominal organs or the pelvis may be present
- Young trauma patients have a remarkable tolerance of hypovolaemia. Blood pressure and heart rate are unreliable indicators of shock and may be relatively normal even with significant blood loss.
- Peritoneal irritation caused by blood or intestinal contents leads to vagal stimulation that in turn inhibits the reflex rise in heart rate expected in hypovolaemia.

- Patients with solid organ injuries may present initially to the emergency department in a stable haemodynamic state and subsequently deteriorate due to rupture of a subcapsular haematoma.
- Underestimation of shock and poor resuscitation frequently contribute to death in potentially salvageable patients.

**Box 6.1: Causes of morbidity and mortality in abdominal / pelvic trauma**

- Uncontrolled bleeding
- Secondary bleeding
- Unrecognised injury
- Over reliance on pain as a diagnostic marker
- Misinterpretation of false negative diagnostic procedures
- Hollow viscera perforation leading to delayed infection and sepsis
- Associated severe head injury

For the reasons outlined above clinical assessment alone is unreliable. Therefore it is important to maintain a high index of suspicion and to reassess a patient regularly if the diagnosis is in doubt.

The key requirements for diagnosing and managing abdominal and pelvic trauma are a clear understanding of the mechanism of injury, relevant clinical anatomy and interpretation of investigations in order to make an early diagnosis of intraabdominal injury. Bedside investigation using ultrasound is playing an increasingly important role in identifying the source of shock and assists in deciding whether emergency laparotomy is required. The key question is “does this patient need early surgical intervention” rather than trying to determine the specific diagnosis. If there are signs of shock and abdominal injury, early laparotomy and / or immediate reduction and stabilisation of the pelvis should be undertaken as part of the resuscitative efforts. In those in whom there is no immediate indication for laparotomy, but a high degree of suspicion, transfer for specialised investigations, such as CT scanning, should only be done when the patient is haemodynamically stable.

A suggestive mechanism of injury should lead to a strong suspicion of abdominal / pelvic trauma

### **Mechanism of injury**

Understanding the mechanism of injury is crucial in the management of trauma patients. Often patients show few or no external injuries. Therefore attention should be paid to the

amount of kinetic energy to which they have been subjected. This is best determined by reference to the mechanism of the injury. This will also alert the team leader to the potential injuries that may exist. Therefore in the early assessment as much information as possible about the mechanism of injury must be collected from the EMS personnel or from persons who first attended the scene (Fig. 6.1).



**Fig. 6.1: Severe frontal high speed impact**

The broad types of mechanisms of injury are **blunt** and **penetrating**.

The type of accident and the direction of forces in blunt trauma can indicate the structures that may be injured. In penetrating trauma visible signs of the injury may be discrete, while severe injuries to organs may exist. An example is the bullet that is deflected by a bone resulting in injuries in unsuspected areas. Furthermore it should be appreciated that one mechanism of injury may lead to another. For instance blunt trauma to the bony pelvis may lead to penetrating trauma of the bladder from the fractured bone.

### **Blunt abdominal and pelvic trauma**

In Europe, this is the commonest mechanism of injury to these regions (>90%), usually as a result of a Road Traffic Accident (RTA) or fall. Abdominal damage results from deceleration or compression forces on solid organs and shear-effects or closed-loop phenomenon on mesentery and bowel. The application of blunt force to the abdomen is usually over a wide area, with the exception of localised blows to the renal angle (assault) or anterior abdomen (for instance bicycle handlebars).

Injuries to the chest and pelvis should suggest that a significant abdominal injury is highly likely, given that much of the contents of the abdomen are within either the bony chest or

pelvis. Equally wherever there is evidence of hypovolaemia, or its consequences, abdominal injuries should be suspected and sought.

**In cases of blunt injury to the trunk, the abdomen must be considered injured, until proven otherwise**

The spleen, liver and kidneys are the commonest solid organs injured by blunt trauma. The hollow viscera may rupture due to the closed-loop phenomenon - in particular the small intestine and bladder. Shear forces may tear the mesentery in falls, leading to bowel ischaemia.

There may be minimal evidence of injury over the exterior of abdomen in blunt trauma. The diagnosis of visceral damage is notoriously difficult, as clinical assessment of abdomen is unreliable. Painful distracting injuries and an altered mental state compound these difficulties. The conventional clinical signs of peritoneal irritation; tenderness and guarding often cannot be seen. Absent bowel sounds should raise suspicion, but may also be an expression of a high sympathetic tone. Often clinical signs are not present where injuries are suspected and the use of special investigations are necessary when there is no clear indication for a laparotomy.

Blunt injury to the pelvis causes haemorrhage with associated skeletal and visceral damage. Clinical assessment of pelvic injury may be unreliable even in the conscious patient. Diagnosis is therefore dependent on having a high degree of suspicion based on the mechanism of injury, particularly when there has been high energy transfer. Once an unstable pelvic injury is suspected, stress testing for pelvic stability ("springing of pelvis") should be avoided as it may exacerbate soft tissue injury within the pelvis and increase bleeding. Plain radiography of the pelvis in the resuscitation room is necessary in all multiply injured patients. Pelvic fractures confirm severe forces and necessitate a search for other major injuries and for appropriate resuscitative measures.

**Penetrating abdominal and pelvic trauma**

Penetrating trauma accounts for a high proportion of abdominal trauma in the United States of America and other countries such as South Africa. In Europe such injuries are much less common. Injuries can result from low velocity objects, such as a knife, or high velocity objects, most commonly bullets. The injury will, of course, not respect anatomical boundaries. The pattern of injuries arising from stab wounds to the abdomen may, to some extent, be predicted from the entry site. However organs can be injured, even though they apparently lie away from the site of the wound. This may be as a result of the ricochet of a

bullet within the body or the posture of the patient at the time of the attack being different from that found when lying supine on a trolley in the resuscitation room. The use of a long-bladed weapon will also affect deep structures away from the wound.

The abdomen is divided into anterior, posterior, flank and lower chest areas. The anterior area is bordered by the anterior axillary lines laterally and the nipple line above. The posterior area is bordered by the posterior axillary lines laterally and the tips of the scapulae above. The flanks are the areas between these. The lower chest area is a special area overlapping with these areas, lying between the nipple line and the costal margin. Stabs wounds in the anterior and flank areas are more likely to penetrate the peritoneum. Local exploration of such wounds by a surgeon will be necessary to determine the extent and depth of underlying tissue damage. If there is doubt, double contrast CT may help. Laparoscopy and ultrasound have limited benefits for assessment of these patients. If all such wounds are explored by laparotomy up to half will not show any significant injury. Stab wounds to the lower chest area, nipple line to costal margin, may damage thoracic and/or abdominal structures.

Gunshot injuries can result in a path that is neither straight nor short. Abdominal injuries may thus arise from remote entry points. Furthermore damage in these injuries may be more extensive due to the cavitation effect of high velocity missiles. This results from the pressure wave that spreads radially from the missile trajectory. The cavity is larger than the bullet and contaminated by debris sucked in the path. As a result, extensive debridement will be required.

The incidence of serious internal damage is much higher for gunshot wounds to the abdomen. The presence of hypotension or signs of peritonism should mandate laparotomy. As outlined above the pattern of such injuries is unpredictable and usually extensive. A low threshold for laparotomy is appropriate. Diagnostic peritoneal lavage and laparoscopy are not reliable in such injuries. In some cases of low velocity stab or gunshot wound, a selective non-operative policy may be safe when managed by an experienced surgical team.

Open pelvic trauma is a life threatening condition. It is often accompanied by major blood loss and laceration of abdominal organs. Early surgical input is essential in caring for patients with such injuries.

### **Assessment and management**

The initial assessment and management of patients with abdominal and or pelvic trauma should follow the system already described in Chapter 1. Wherever possible, the admitting

hospital should receive notification of the impending arrival of these patients to ensure that appropriate resources are immediately available on their arrival in the emergency department. In addition, the mechanism of injury, injuries sustained, signs and symptoms, treatment given and the patient's response are extremely valuable to the trauma team leader.

### **Primary survey**

The purpose of the primary survey is to identify and treat life-threatening injuries rapidly and the main threat is from haemorrhage. After treating airway and breathing problems, the abdomen must be considered as a source of haemorrhage. In addition, shock may be due to peritonitis from perforation of the bowel. An abdominal or pelvic injury must be considered if the mechanism of injury is appropriate or the patient presents with:

- abdominal pain
- bruises, laceration of the abdominal wall, scrotal haematoma, flank haematoma
- tenderness, guarding, or pelvic instability

However, in severely injured trauma victims a **reliable clinical assessment** is impossible.

The classical signs may be masked by several factors:

- high doses of analgesics given prior to assessment
- the patient has been anaesthetised on scene
- altered mental status (TBI, alcohol, drugs, hypothermia, shock)
- other distracting, painful injuries (long bone fractures)
- a spinal cord injury

The key question in abdominal trauma is:

- Is an intraabdominal injury the cause of the patient's shock?

### **Identifying abdominal and pelvic haemorrhage as cause of shock**

Clinical assessment alone may be difficult as the classical signs of the acute abdomen may not be present. Focused Abdominal Sonography in Trauma (FAST) is the first line diagnostic test to confirm or to rule out intraperitoneal haemorrhage as a source of shock as part of the primary survey. However, in the shocked patient who fails to respond to the initial fluid resuscitation and an intraperitoneal abdominal source of shock is likely, immediate laparotomy should be the rule. It is crucial to understand that resuscitation may include laparotomy and that correction of hypovolaemia may not be possible or desirable before surgical control of bleeding is achieved. Nevertheless, in severely compromised patients transfusion should be started before surgery commences because opening the peritoneum may cause significant hypotension as the tamponading effect is lost and bleeding increase.

If the source of bleeding is most likely within the *retroperitoneal* space surgery may be withheld, since opening of the retroperitoneum may lead to disastrous instability, especially in pelvic fractures. In these patients closure and stabilisation of the pelvic ring should be attempted immediately in order to control of haemorrhage. Significant scrotal or labial swelling on admission (Fig. 6.2) indicates arterial bleeding within the pelvis that may not be controlled by closure of the pelvic ring only.



**Fig. 6.2: scrotal haematoma due to arterial bleeding in complex pelvic fracture**

Initially, temporary measures such as; the application of a pneumatic anti-shock garment (P.A.S.G.), a vacuum splint, the application of a compressing belt (Fig. 6.3) or a bed-sheet around the pelvis, may suffice. External fixation using a “C-clamp” is effective for haemostasis, if the bleeding is venous. Angiography and selective embolisation may be needed to achieve haemostasis with arterial bleeding in about 20% of complex pelvic fractures and is successful in 90% of the cases when the source can be identified. If this fails, extraperitoneal packing can be lifesaving.



**Fig. 6.3: Application of a pelvic compression belt**

If an unstable pelvic fracture and intraabdominal free fluid are present at the same time a balance must be made. It seems to be sensible to stabilize the pelvis before the laparotomy is carried out.

Some patients can bleed excessively during laparotomy. This can be either due to the nature of the injury (liver rupture), or to dilutional coagulopathy as a result of the fluid given, compounded by hypothermia. These patients benefit from a staged surgical approach: the primary laparotomy only aims at haemorrhage control that is achieved by packing of the abdomen and provisional wound closure. The definitive repair is carried out after correction of blood loss, acidosis, coagulopathy and hypothermia usually in a critical care environment.

### **Secondary Survey**

Once the patient is haemodynamically stable and normal and the primary survey completed, the abdomen must be re-evaluated as part of secondary survey. A head to toe examination looking to identify potentially life-threatening injuries is now performed as described in Chapter 1. The abdomen is assessed for possible internal damage using the system of LOOK – LISTEN – FEEL.

#### **Look**

The patient must be completely exposed and the abdominal surface inspected for wounds, bruising and imprints from contact, for example, tyre marks. This must include the back and the perineum that will require the patient to be log rolled. Care must be taken to prevent hypothermia. Look for scrotal or labial swelling as it may indicate bleeding within the pelvic retroperitoneum.

#### **Listen**

Auscultation of the abdomen to detect bowel sounds adds very little to the assessment, and may be difficult in the resuscitation room.

#### **Feel**

The abdomen should be gently palpated to assess distension, tenderness and guarding. All these signs may be subtle or totally absent in the early stages. Shoulder-tip pain may be an indicator of subphrenic irritation from intraperitoneal blood. Percussion may elicit rebound

tenderness more reliably than direct palpation. It is important to remember that injuries to the chest or the pelvis can produce abdominal signs.

Rectal or vaginal examination should be performed to detect the presence of blood and bony fragments, assess anal sphincter tone and the position of the prostate, as appropriate. This can be done supine or during the log roll. If examination of the rectum and external genitalia does not suggest urethral injury, a urethral catheter should be passed to measure urine output, decompress bladder and detect haematuria. A nasogastric tube helps decompress the stomach since most of the patients will have some degree of gastroparesis after trauma. As explained above, the practice of “springing” the pelvis to elicit instability is best avoided. On suspicion of a pelvic injury, a plain x-ray is the quickest and safest way to establish the diagnosis.

It should be mentioned that it is both humane and physiologically appropriate to give a conscious patient effective analgesia prior to secondary survey. This does not compromise abdominal assessment and may help to reveal clinical signs. The most common, simple and effective method of initially providing analgesia for the patient with abdominal and pelvis trauma is to give strong opioids titrated in small aliquots by the intravenous route. Further details and alternative techniques are discussed in Chapter 16.

**If the secondary survey reveals the signs of an acute abdomen urgent surgical referral is required.**

### **Investigations**

This section outlines the commonly available modalities that are used in the assessment of patients with abdominal and pelvis trauma. Although other specialised investigations are available, local expertise and protocols must guide their use.

#### Blood tests

During the primary survey, blood samples should be sent for a baseline full blood count, assessment of clotting, biochemistry including amylase and cross matching. Pregnancy testing must be undertaken in all female patients of childbearing age. An arterial sample should be sent for blood gas and acid base analysis. A severe coagulopathy on admission indicates an endangered patient. Maximum efforts have to be made without delay to control bleeding and to restore blood volume as well as to normalise haemostasis. A persistent negative base excess indicates that resuscitative efforts are insufficient and that the patient is still shocked.

### Radiography

X-rays of the chest and pelvis are requested early in the primary survey and should be performed in the resuscitation room. The former may reveal rib fractures or evidence of diaphragmatic injury (Appendix 6.1). The presence of gas under the diaphragm may not be visible on supine films. A system for interpreting the pelvic x-ray is given in Appendix 6.1. A plain film of abdomen is rarely useful, except to show the position of a residual foreign body such as a bullet or shrapnel. Much more useful in abdominal trauma is a pelvic film. Interpretation must be undertaken in a systematic fashion to avoid missing important findings (Appendix 6.2).

### *Focused abdominal sonography for trauma (FAST)*

The availability of ultrasonography in the resuscitation room should be the standard procedure on admission in all trauma patients with suspicion of abdominal trauma for many reasons:

- it is a rapid and repeatable investigation
- it provides information about the volume status of the patient:
  - An empty bladder after fluid resuscitation suggests ongoing fluid loss
  - An obviously filled bladder after fluid resuscitation suggest sufficient fluid replacement or retention
  - Inspiratory collapse of the vena cava suggests volume depletion.

The FAST protocol concentrates on finding fluid within the abdomen. This examination can detect as little as 50 ml of fluid in the subphrenic spaces, the subhepatic space and the pelvis (and pericardium). It may be repeated at frequent intervals without side-effects. The FAST examination can achieve almost 90% sensitivity, but it is operator dependent. An experienced sonographer is usually able to complete an examination within two minutes and can reliably determine if there is intraabdominal free fluid. However, it has low specificity in terms of determining the source of any fluid identified. Limitations of this investigation are missed injuries, especially solid organ damage without rupture of the capsule and retroperitoneal injuries.

### *Computed tomography (CT)*

Modern CT scanners can produce high-resolution images rapidly and the use of intravenous, oral or rectal contrast enhances the images resulting in an investigation with a high sensitivity and specificity. CT allows imaging of both the viscera and musculo-skeletal structures, making it ideal for most abdominal trauma. It is however less reliable for injuries to the diaphragm, bowel or pancreas. Nevertheless, it has superseded many other investigations,

for example intravenous urography for renal trauma. If a multi-slice CT scan is available, the mandate that only resuscitated patients who are haemodynamically stable are suitable for this diagnostic procedure is relative, as a total body CT-scan can be performed within few minutes. Nevertheless to be safe in the CT room it is good practice to ensure that patients have responded to resuscitation, be stable for a period of time and accompanied by suitably trained and equipped staff.

#### *Diagnostic peritoneal lavage (DPL)*

Since the introduction of bedside sonography, DPL has virtually disappeared from clinical practice. Nevertheless, if other modalities are not available, it is a highly accurate bedside test for the presence of abnormal fluid within the peritoneum; blood, bowel content or urine. It is a highly sensitive but has low specificity.

The indications for DPL are:

- suspected haemoperitoneum in a hypotensive patient.
- diagnosis of blood or hollow viscus contents in the peritoneal cavity.
- FAST not available

DPL is contra-indicated if there is a clear clinical indication for laparotomy. Previous scars, obesity, coagulopathy and pregnancy are relative contra-indications. It will, of course, not given any indication of injuries in subcapsular haematomas or in the retroperitoneum, unless there is peritoneal involvement. While the complication rate of DPL is low (1%) it is clearly invasive, time consuming and must be performed by a person with surgical skills. It cannot be repeated as part of the constant re-evaluation of the patient (as can ultrasound).

#### *Local wound exploration*

A stab wound may be explored locally to determine whether there is a peritoneal breach. Penetration of the transversalis fascia or inability to find the end of the tract constitutes a positive exploration and the patient will require a laparotomy or further diagnostic evaluation. Stab wounds in the posterior abdomen and flank are more difficult to explore due to the thickness of the muscles.

#### *Definitive care*

Definitive care is provided after complete resuscitation of the patient. It will be based upon the response of the patient to the treatment administered, the results of all the investigations and the extent of all the abdominal injuries identified. Many patients with solid organ injury (SOI) from blunt trauma may be managed without operation. This is usually only possible if the organ capsule is not ruptured. Observation of vital parameters in a ward with continuous monitoring is mandatory. Frequent control of haemoglobin and haematocrit is necessary as

well as repeated sonography. Surgical intervention must be possible at any time. A summary of the management of injuries to intraabdominal organs is given in Box 6.2.

**Box 6.2: Summary of the management of injuries to abdominal organs**

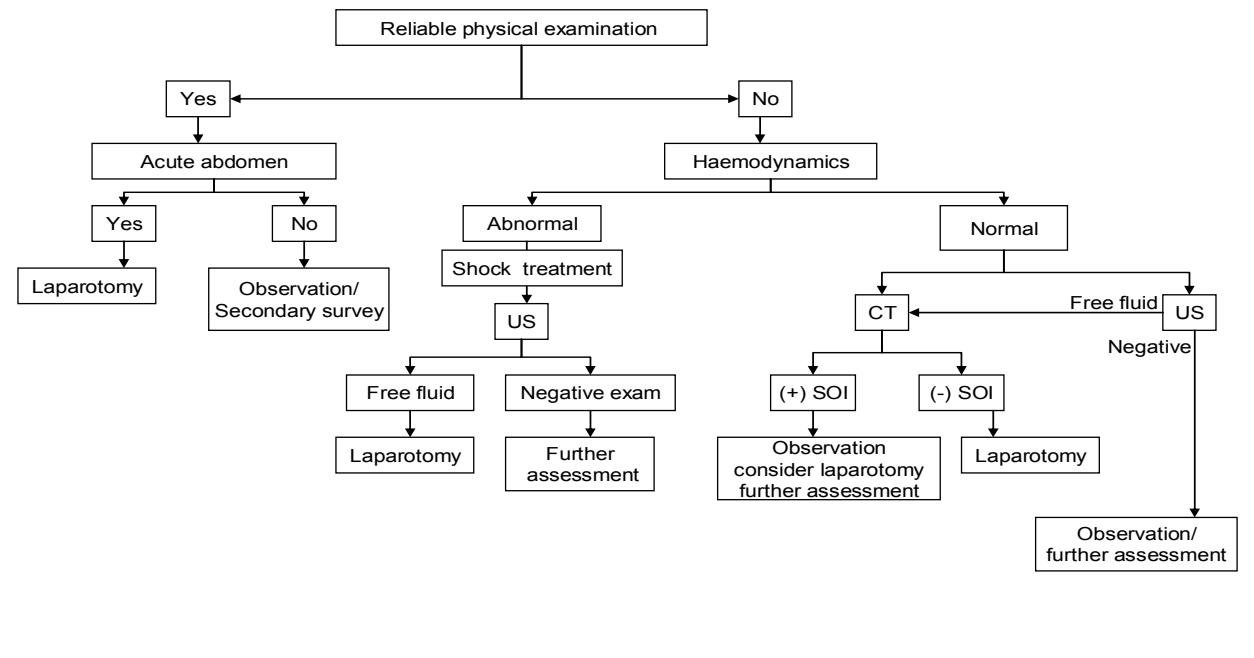
Organ	Haemodynamically Unstable	Haemodynamically stable
Diaphragm	Surgical repair	Surgical Repair
Liver	Laparotomy repair/resection /packing/conservative management	Non-operative management/repair
Spleen	Splenectomy or Repair	Non-operative management/splenectomy/repair
Stomach	Repair	Repair
Duodenum &	Repair	Repair
Small Intestine		
Biliary Tree	Repair & drainage	Repair & drainage
Pancreas	Debridement/excision/drainage	Non-operative management /debridement/excision/drainage
Large Intestine	Repair/Colostomy	Repair/colostomy
Bladder	Repair	Catheter decompression/repair
Kidney	Repair/partial nephrectomy	excision/ Non-operative management/ drainage
Vascular Injury	Repair	Repair

**Summary**

Injuries to the abdomen and the pelvis can vary immensely in magnitude. The presence of significant injury may not always be obvious on presentation. The clinical signs may add to the confusion. The trauma team must be suspicious of the existence of abdominal trauma in all patients of multiple injuries and must have a clear understanding of the mechanism of injury. It is best to assume that abdominal trauma exists unless proven otherwise.

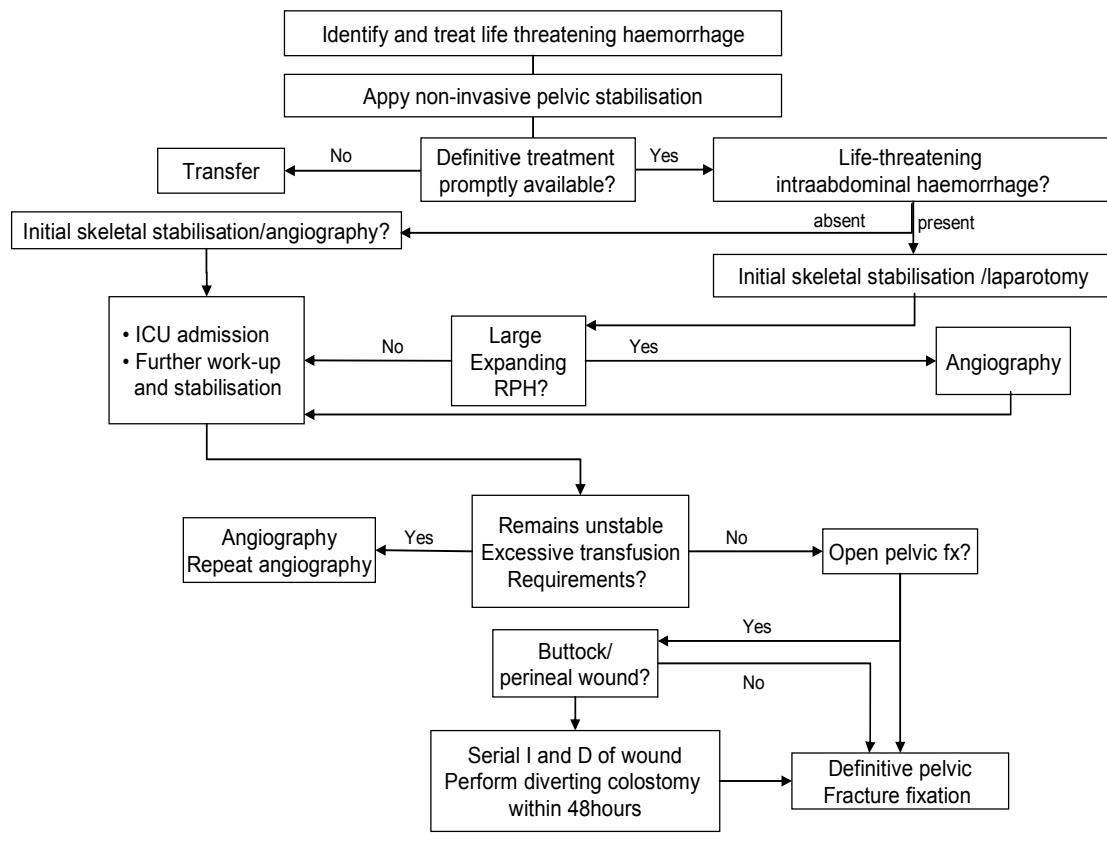
The investigations available to establish a diagnosis all have their roles and must be used liberally according to the index of suspicion. Transfer of patients for such investigations must only be done with haemodynamically stable patients. Such heightened awareness and a policy of aggressive investigation will help reduce unexpected findings in the abdomen and the avoidable deaths that still occur due to abdominal trauma.

## Management of Blunt Abdominal Trauma



From: Davi G. Jacobs, James F. Kellam, Michael A. Gibbs, Kenneth A. Egol *Initial assessment and stabilization of the hemodynamically unstable pelvic fracture patient in Initial Management of Injuries: Evidence Based Approach.* Blackwell BMJ Books 2001

## Initial Assessment and stabilization of the haemodynamically unstable patient with pelvic fracture



*From: Davi G. Jacobs, James F. Kellam, Michael A. Gibbs, Kenneth A. Egol Initial assessment and stabilization of the hemodynamically unstable pelvic fracture patient in Initial Management of Injuries: Evidence Based Approach. Blackwell BMJ Books 2001*

## **Appendix 6.1: Clinical Anatomy**

In the following sections the applied anatomy is combined with a discussion of the nature of injuries and their management.

### **The abdomen**

The abdomen is bounded by the diaphragm superiorly and the pelvic floor inferiorly. The pelvic rim divides the abdomen from the pelvis, although the two are contiguous. Posteriorly are the vertebral column and paravertebral muscles, antero-laterally from above downwards are; the bony rib cage, the abdominal muscles and the bony pelvis. The contents of the abdomen occupy the following regions:

1. Peritoneal cavity
2. Retroperitoneum
3. Pelvis

Organs that are largely covered with peritoneum (see below) are called intraperitoneal and those which are not, extraperitoneal. The pelvis contains both intraperitoneal and extraperitoneal structures. The retroperitoneum contains extraperitoneal structures and is continuous below with the extraperitoneal part of the pelvis.

### **The peritoneum and peritoneal cavity**

The peritoneum is a continuous serous membrane that covers certain organs in the abdomen. It is a closed sac and so has two layers that contain a potential space containing a small volume of serous fluid. The visceral layer covers the organs and the parietal layer lines the abdominal wall. These two layers are closely applied to each other and the cavity becomes apparent only when it is opened or if fluid (blood, bowel content or urine) collects in it. Fluid tends to accumulate in certain deep pockets in a supine patient, which may be examined by imaging. Free fluid in the *hepato-renal space (of Morrison)*, the *recto-vesical space (of Douglas)* or the *space between left diaphragm and spleen (of Koller)* are easily identified by ultrasound or CT-scan.

Chemical or bacterial irritation of the peritoneum leads to peritonitis, which eventually causes the clinical signs and symptoms of pain, muscle guarding and rebound tenderness. In early peritonitis these signs and symptoms are often subtle and may appear too late to be relied upon for diagnosis. The presence or absence of bowel sounds does not help identify or exclude an intra-abdominal lesion or peritoneal irritation.

Obvious signs of peritoneal irritation may be a late feature of abdominal trauma – but when present indicate the need for laparotomy as part of the resuscitative efforts

## *1. The peritoneal cavity*

When considering possible injuries in trauma patients, the peritoneal cavity can be considered in three parts.

The upper **intrathoracic** part is contained within the bony rib cage

The lower **pelvic** part lies within the bony pelvis

The **abdominal** part lies between these two.

The intrathoracic part may extend up to the nipple line in peak expiration so that trauma to the lower chest can lead to injury to intraperitoneal and as well intrathoracic organs. Damage to the intraperitoneal structures in the pelvis, mainly the small intestines, by trauma to the bony pelvis is unlikely as it is much more stable than the thorax. In contrast, extraperitoneal structures that are in close communication with the pelvic bones (e.g. bladder) are more often injured.

### Contents of peritoneal cavity

Intrathoracic	Abdominal	Pelvic
<ul style="list-style-type: none"><li>• Diaphragm</li><li>• Liver</li><li>• Spleen</li><li>• Stomach</li></ul>	<ul style="list-style-type: none"><li>• Small intestine</li><li>• Large intestine</li><li>• Omentum</li></ul>	<ul style="list-style-type: none"><li>• Intestines</li><li>• Bladder</li><li>• Uterus &amp; ovaries</li></ul>

Some of these structures pass through both the intra and extra-peritoneal regions. For instance the colon is retroperitoneal in its ascending and descending parts but intraperitoneal in its transverse and sigmoid parts. In other structures their containment within the peritoneum may vary. The urinary bladder extends more and more into the peritoneal cavity as it distends. Rupture into the peritoneum is thus more common when the bladder is distended and injured by a compressing force.

### Diaphragm

On each side, the diaphragm forms a dome shaped muscular structure separating the thorax from the abdomen. The muscular root fans out from attachments to the upper three lumbar vertebrae becoming tendinous over the dome. Peripherally it is attached to the sternum and ribs. Diaphragmatic injury is rare after abdominal trauma and is usually associated with penetrating trauma. Blunt trauma to the abdomen may cause a sudden increase in intra-abdominal pressure resulting in rupture of the diaphragm and herniation of the abdominal contents into the chest. A considerable force is needed to do this and there are, therefore, a number of significant associated injuries. Splenic trauma occurs in 25% of patients and pelvic

disruption in 40%. Other associated injuries include long bone fractures and closed head injury.

**Diaphragmatic injury occurs in only 3% of all abdominal trauma. It is relatively more common in penetrating trauma especially where there are penetrating left-sided thoraco-abdominal wounds**

Diagnosis can be difficult, a penetrating chest injury should alert the team leaders to the possibility. The typical features of disproportionate chest/abdominal pain, dyspnoea, reduced air entry/dullness to percussion at the bases, are often equivocal in the resuscitation room. Radiographic signs indicating the presence of abdominal structures in the chest, for instance bowel gas patterns or presence of a naso/orogastric tube are diagnostic. Change to the contour of the diaphragm is an indicative but not diagnostic radiographic sign. Other modalities including ultrasonography, CT scanning and diagnostic peritoneal lavage are all unreliable. Diagnostic laparoscopy has recently been shown to be accurate and reliable in stable patients, where there is no clear indication for laparotomy. However in patients with TBI it is essential to ensure that hypercarbia from the use of carbon dioxide for insufflation does not occur because of the risk of increasing ICP.

At laparotomy for trauma a diaphragmatic injury should always be sought by careful examination. A tear may not be obvious particularly as herniation through a defect may be prevented by positive pressure ventilation. Repair should be undertaken at operation.

#### Liver

The liver is situated in the right upper quadrant of the abdomen and is almost entirely covered by the abdominal rib cage. It is mostly intraperitoneal, very vascular and is connected to the inferior vena cava by large hepatic veins. Liver injury may be associated with biliary tree damage and/or massive bleeding. After the spleen, the liver is the second commonest abdominal organ damaged in blunt or penetrating trauma. Bleeding may remain undetected and is an important cause of preventable death. Systems exist to grade liver injury to help decide the most appropriate management. They depend on the size of haematomata and the degree of parenchymal disruption. It is important to remember that the first signs of liver injury may only occur after the start of mechanical ventilation, as the increase in venous pressure causes severe haemorrhage.

The presence of hypovolaemic shock, in the absence of an alternative source, should be assumed to be due to hepatic or splenic injury

Liver injury may only be diagnosed at laparotomy performed for non-specific signs of intra-abdominal bleeding or peritonitis. It is, however, important to recognise that some severe disruptive injuries are best managed conservatively as surgery may lead to sudden overwhelming haemorrhage. Liver damage may be repairable but when severe will need to be packed, the patient stabilised and arrangements made for transfer to a specialist centre.

Haemodynamically normal and stable patients should be investigated by CT scanning or ultrasonography. While even large intrahepatic haematomas may be difficult to identify by ultrasonography, CT scanning has the advantage of accurate delineation of the extent of liver damage, which is vital for non-operative management.

#### Gall bladder and biliary tract

These organs are located under the right lobe of liver and are often damaged in association with liver injury (50%) and occasionally pancreatic injury (17%). Clinical features depend on the presence of blood or bile in the peritoneum. Endoscopic retrograde cholangio-pancreatography (ERCP) may be necessary to evaluate damage, where there is no clear indication for laparotomy. The patient must be haemodynamically normal and stable to tolerate the transfer and procedure. Surgical treatment of extra-hepatic biliary damage can involve cholecystectomy and biliary drainage, diversion or reconstruction.

#### Spleen

The spleen lies under the seventh to eleventh ribs in the left upper quadrant of the abdomen. It is a very vascular organ and forms part of the reticuloendothelial system.

**Failure to diagnose and promptly treat splenic injury is a significant cause of preventable death following trauma, as massive bleeding can occur**

As with the liver, splenic injury may only be diagnosed at laparotomy done for signs of intra-abdominal bleeding following trauma. The early signs of intraperitoneal bleeding may be subtle and hidden by painful distracting injuries such as rib fractures. Left upper quadrant pain, left lower rib fractures or changes to the contour of the left hemidiaphragm on plain radiography are suggestive but non-specific features. Ultrasonography or CT scanning may show the injury and can help to determine whether a non-operative approach is possible.

Diagnostic laparoscopy may be useful in assessing a stable patient with penetrating abdominal trauma.

Where patients with splenic injury are found to be haemodynamically normal and stable, careful investigation and observation may suggest that a conservative approach is appropriate. Preservation of the spleen to reduce the long-term risks of sepsis is the aim. Patients who have had their spleen removed are prone to particular infections such as those caused by pneumococci. This is especially so in children. Grading scales exist to aid this decision-making. Patients treated conservatively must be observed very closely.

When an injured spleen is found at laparotomy it can be removed, or, in some cases, preserved. Where the latter approach is used, very careful observation should be instituted to detect any signs of complication arising in the spleen.

#### Stomach

The stomach is a flat hollow viscus in the epigastrium and left upper quadrant, which is rarely injured as a result of blunt trauma. The clinical features are usually, therefore, of a penetrating injury with peritonitis, due to leakage of gastric contents into the peritoneal cavity. Blood in the gastric aspirate or free gas on chest radiography may help to confirm the diagnosis. CT scan is insensitive. Any patient with peritonitis following trauma should undergo surgical exploration. Diagnosis may only be established at such an operation and surgical repair of the stomach is the rule.

#### Small intestine and colon

The small intestines (jejunum and ileum) are entirely within the peritoneal cavity. Injuries to them are similar to those of the intraperitoneal colon (e.g. the transverse and sigmoid colon) and may result from blunt or penetrating trauma. Rapid deceleration injuries while wearing restraints (e.g. seatbelt) may cause compression for bowel loops and consequent rupture, by a closed loop mechanism. Tears may occur to the bowel itself or to its mesentery. The latter can cause intestinal ischaemia and is a cause of late rupture. Occasionally mesenteric tears are associated with massive bleeding. The flexion/distraction nature of this mechanism of injury means that there is an association with transverse vertebral body fractures in the thoraco-lumbar spine (Chance fractures).

Intestinal perforation from either penetrating or blunt injury leads to peritonitis from leakage of intestinal content. As outlined above, such peritonitis may be easy to miss in the early stages. CT scanning even with oral contrast may not be reliable in the early stages. The treatment of intestinal injury is surgical repair at exploratory laparotomy under prophylactic antibiotic cover.

## ***2. Retroperitoneum***

Injury to structures in the retroperitoneum (kidneys, ureters, abdominal aorta, inferior vena cava (IVC), lumbar spine) may be difficult to detect. Blood loss into the retroperitoneal space can be relevant and ongoing, especially if coagulopathy is present. Bleeding into the retroperitoneum always should be ruled out, especially if no intrathoracic or intraabdominal source of bleeding can be identified, or is obvious elsewhere. Unless there is communication with the peritoneal cavity there are seldom signs of peritoneal irritation and diagnostic peritoneal lavage is negative. Haematomata will often become contained with arrest of bleeding, due to a combination of hypotension and local tamponade. Injuries may be easier to detect if there is rupture into the peritoneal cavity. The retroperitoneum is continuous with the extraperitoneal part of the pelvis, containing the rectum, bladder and pelvic vessels (Box 6.3).

### **Box 6.3: Contents of retroperitoneum/extraperitoneal pelvis**

- Pancreas
- Duodenum
- Colon – ascending and descending
- Aorta/pelvic arteries
- Inferior vena cava/pelvic veins
- Kidneys, ureter & bladder
- Rectum

### **Pancreas**

The pancreas lies horizontally across the body of the first lumbar vertebra, with its tail in contact with the splenic hilum. It may be injured by either penetrating or blunt trauma. In 60% of cases, injury results from a high-speed impact against the steering wheel. The consequences of injury include acute pancreatitis and leakage of pancreatic enzymes into the peritoneal cavity. The most common symptom is abdominal pain, which may be unexpectedly severe. If there is a clear indication for laparotomy the pancreas must always be carefully examined at the time. If there is no clear indication for laparotomy diagnosis of pancreatic injury may be very difficult. The clinical signs are often non-specific and serum amylase may be normal in the early stages. Similarly CT with dual contrast (intravenous and oral) has a high false negative rate, despite being the imaging modality of choice. Sequential measurement of serum amylase and CT scanning may be helpful. If there is any suspicion of pancreatic duct injury an ERCP should be considered. The patient's condition, and the extent of damage, will determine whether conservative or operative intervention is appropriate. Operative treatment may involve drainage, repair or excision of the pancreas.

### Duodenum

The first part of duodenum lies in continuity with the stomach in the peritoneal cavity and the remaining three parts are in the retroperitoneum. Penetrating trauma causes about three-quarters of duodenal injuries. Impact from a steering wheel or bicycle handle bar is the commonest form of blunt injury. The diagnosis may be difficult and is helped by free air under diaphragm or blood in the gastric tube. A double contrast CT (oral and intravenous) is usually confirmatory. As with many injuries within the abdomen, confirmation may only be possible at laparotomy done for non-specific reasons. The duodenum should be carefully inspected in all cases and surgical repair undertaken.

### Colon and rectum

Injuries to the intraperitoneal segments of the colon are considered above. Injuries of the retroperitoneal colon and the rectum usually result from penetrating trauma. Unless there is faecal contamination of the peritoneal cavity and consequent peritonitis, the diagnosis may be very difficult. The diagnosis may, thus, be made late as a result of abscess development. If suspected a soluble contrast enema or CT with triple contrast (oral, rectal and intravenous) may help. Surgical repair of the colonic injury is the rule. Such repair often necessitates the formation of a temporary colostomy/ileostomy, proximal to the injury.

### Aorta and vena cava

These vessels lie on the vertebral column as they pass through the abdomen. They are entirely retroperitoneal and are continuous with the pelvic vessels in the extraperitoneal parts of the pelvis. Major injury to these vessels occurs most commonly after gunshot wounds (up to 25%) and more rarely after blunt trauma to the abdomen. Injuries may present as haemorrhage or secondary thrombosis. Injuries to the aorta or vena cava may result in massive haemorrhage, but presentation may also be late due to bleeding from the formation of a false aneurysm. The presenting symptoms or signs are those of abdominal pain, shock from bleeding or ischaemia of the lower limbs or kidneys. The diagnosis may only be established at operation. Pre-operatively either ultrasonography or contrast CT scanning can aid diagnosis. Contrast CT is preferable to intravenous urography for assessment of renal involvement but great care must be exercised in such patients to ensure that transfer is only done when the patient is haemodynamically normal and stable. Many patients are best managed by immediate transfer to the operating theatre for direct repair.

### Kidneys

The kidneys lie on the posterior abdominal wall at the level of twelfth thoracic and upper three lumbar vertebrae. They are surrounded by renal fascia and are well protected by

muscle and bone behind and the peritoneal cavity in front. Blunt injuries are commoner and are often associated with injuries to adjacent organs, in particular the spleen and liver. Isolated renal damage may result from penetrating injury to the flank. Haematuria is the most consistent feature of renal trauma, but its degree may not correlate well with the severity of injury. Sometimes this sign is misleading if myoglobinuria is responsible for colouring the urine. Although a perinephric haematoma is common, haemorrhage is rarely life threatening, except where a renal pedicle has been avulsed. In a stable haemodynamically normal patient, a contrast CT is 98% accurate for detailed imaging of renal trauma, including vascular and parenchymal injury. An intravenous pyelogram (IVP) is an acceptable but less comprehensive alternative. Perinephric haematoma may also be visualised by ultrasonography. Most renal trauma is managed conservatively, even in the presence of gross haematuria. Surgical repair is reserved for persistent haemorrhage, urinary extravasation or vascular pedicle injury.

### Ureters

The ureters lie behind the peritoneum on the psoas major muscle as they run along the transverse process of the vertebrae. Unlike the kidneys, ureteric damage from blunt or penetrating trauma is uncommon. Surgical repair or reconstruction is the rule for damaged ureters. As with injuries to the retroperitoneal colon late diagnosis may occur due to secondary abscess formation.

### Bladder

The urinary bladder is a pelvic organ that may stretch to the umbilicus when full. It lies in the extraperitoneal part of the pelvis but indents the peritoneal cavity as it distends. Rupture may occur into the peritoneum or outside it. Compression injuries will often lead to intraperitoneal rupture and, thus, the clinical features of peritonitis. Blunt trauma may lead to a penetrating injury as a result of perforation by fragments of fractured pelvic bones. This accounts for 80% of bladder injuries. This sort of injury usually results in extraperitoneal extravasation of urine, which is difficult to diagnose. Secondary pelvic sepsis may develop when these injuries are missed. Bladder rupture is suspected in all cases of gross haematuria and pelvic fracture. Urinary extravasation may be palpable as a boggy perineal swelling. A retrograde cystogram is more accurate than excretory urography or diagnostic peritoneal lavage (see below) for the detection of bladder injury. Indwelling catheter drainage may suffice for most bladder ruptures, with surgical repair reserved for extensive lacerations. Most intraperitoneal ruptures require laparotomy for peritonitis and surgical repair.

## Urethra

The short female urethra is rarely damaged. In contrast, the male urethra may be injured above or below the urogenital diaphragm. Above the urogenital diaphragm extravasation occurs into the extraperitoneal pelvis. Injuries below lead to urine collecting in the scrotum and lower abdomen. Urethral injury occurs in 10% of pelvic fractures and therefore is often associated with injuries to other body regions. Isolated injury can occur as a result of blunt trauma to perineum, for instance a fall astride a hard object. The diagnosis is suspected from a suggestive history, inability to pass urine, blood at the urethral meatus and a high prostate on rectal examination. The site of rupture may be identified by leakage of contrast in ascending urethrography. Urinary leakage leads to local sepsis either in the pelvis or superficial tissues. If urethral injury is suspected, no attempt should be made to pass a urinary catheter until urological advice is sought. Suprapubic urinary drainage is often adequate for healing. Major disruptions require surgical repair.

### 3. *The pelvis*

The bony pelvis is composed of three strong bones – the two innominate bones connected to each other anteriorly and to the sacrum posteriorly. Very strong ligaments over the sacroiliac joints contribute significantly to the stability of the pelvis. For the joints to be disrupted these strong ligaments must be torn. This is associated with significant vascular and visceral damage.

#### Classification of pelvic fractures

- Internal rotational injuries. Often due to lateral compression forces eg after road traffic injury.
- External rotational injuries. Due to increasing anteroposterior trauma.
  - Symphysis pubis and/or rami are disrupted and anterior pelvic floor damaged
  - Sacroiliac joints open up
  - Posterior ligaments disrupted and pelvis becomes vertically unstable
- Translational injuries. Complete discontinuity of all posterior osteo-ligamentous structures as well as the pelvic floor. The pelvic ring is disrupted anteriorly and posteriorly (Fig. 6.3). Most extreme variety is traumatic hemipelvectomy

**Fig. 6.3: Severe translational injury**



With separation of the bones, pelvic vessels are torn and may bleed profusely. The bleeding most often is largely venous but may be also arterial. Bleeding after external rotational and translational injuries is often life threatening. The reported mortality of such open pelvic fractures is around 30-40%.

## **Appendix 6.2: Interpretation of the pelvic x-ray.**

Using a simple 'ABCS' system can allow the correct diagnosis to be made in up to 94% of cases

### **A = accuracy, adequacy & alignment**

Ensure it is the correct x-ray for the patient! An adequate pelvic x-ray should include the whole pelvis and the proximal 1/3 of femurs. The alignment of three rings is checked. The sacrum and the pelvic brim form the large one. The two small ones are the obturator foramina. If one of these circles is broken a search should be made for fractures or joint separation. The last check of alignment is made using a smooth curved line continuous with the obturator foramen and the inner surface of the neck of femur (Shenton's line).

### **B = bones**

All the bones should be traced along the cortical margin to detect a fracture, which may show up as a lucency, density or trabecular disruption.

### **C= cartilage**

The sacroiliac joints and the symphysis pubis should be checked for widening. The acetabular margin may also reveal fractures.

### **S= soft tissue**

A pelvic wall haematoma may be detected by bladder displacement.

## **Websites**

<http://www.trauma.org/abdo/>

Review of techniques discussed in chapter and case reports.

<http://www.east.org>

Current guidelines on abdominal and pelvic trauma.

<http://www.trauma.org/ortho/pelvis.html>

Cases and links to other websites.

<http://aoademo.adam.com/>

Online anatomy teacher.

### **Further reading**

1. Abdominal trauma, F. William Blaisdell, Donald D. Trunkey, Thieme-Stratton Inc. New York
2. Initial Management of Injuries R.F. Sing P.M. Reilly, BMJ Books 2001



## Chapter 7

# Head Trauma

### Objectives

At the end of this chapter the reader should understand:

- The terms commonly used when describing the type of head injury sustained
- How to assess the patient with a head injury
- How to safely manage a patient with a head injury
- When and how to communicate with a neurosurgeon
- When to perform investigations or carry out specific treatments

The anatomy and physiology relevant to head injury are in Appendix 7.1 and 7.2 respectively and types of head injury in Appendix 7.3 at the end of the chapter.

### INTRODUCTION

The incidence of moderate and severe head injury in Europe as estimated from major clinical studies is between 200 and 300 per 100.000 of the population, with road traffic accidents (RTAs) being responsible for 26% of cases. These occur during leisure time (35 %), at home (30%) and at work (15%). About 28% of victims are under the age of 15 and 18% above 65 years of age. The majority of patients are seen initially in a non-specialist centre and subsequently more than half will require transfer to a neurosurgical unit. Outcome is poor with a mortality of 31%, 3% in a vegetative state, 16% severely disabled and 20% moderately disabled. Only 30% have a good recovery 6 months after injury. As the majority of head injuries occur in younger male age groups, the economic and social consequences of delayed or inadequate treatment can be devastating. Alcohol is a contributory factor in 25% of cases of head injury. Clearly, staff working within the ED must become familiar with managing all forms of head injured patients, since they represent a large proportion of their workload.

### Types of brain injury

#### Primary brain injury

Damage that is sustained at the time of the impact is termed, primary brain injury. It may result in:

- Fractures, as a result of a direct blow, with or without underlying brain injury
- A spectrum of injury ranging from concussion to diffuse axonal injury as a result of inertial forces
- Contre coup injury, ie damage away from the site of impact
- Extradural, subdural or intracerebral haematomas

- Subarachnoid haemorrhage

Further details are given in Appendix 7.3.

### **Secondary brain injury**

This is damage that occurs after the primary brain injury as a result of:

- Hypoxia ( $pO_2 < 10$  kPa)
- Hypotension (systolic BP < 90 mmHg)
- Delay in diagnosis
- Delay in definitive treatment
- Seizures
- Extremes of arterial  $pCO_2$
- Raised ICP
- Suboptimal management of other injuries

Many of these are easily preventable, recognisable and treatable within the ED.

### **SIGNS OF A HEAD INJURY**

Signs of a head injury may be non-specific, and can develop in an atypical pattern.

Practitioners must therefore always have a high degree of suspicion of injury based on the presenting history or information obtained.

Consciousness is determined by a number of intra- and extracranial factors. Damage to the reticular formation (a neuronal network in the midbrain and brain stem) or either of the cerebral cortices will result in a loss of consciousness. Although an altered level of consciousness is a common indication that a patient has a head injury, it is only a reliable sign of the severity of injury, when contributory secondary factors (Box 1.4) have been corrected.

Another common sign of a head injury is the presence of a dilated pupil. However it must be remembered that in a patient with multiple injuries this may be the result of direct eye trauma. A high degree of vigilance along with continual reassessment of the patient is therefore necessary. Early manifestations of temporal lobe problems (e.g. extradural haematoma) relate to its close proximity to the tentorium, where its medial aspect compresses the oculomotor nerve (IIIrd cranial nerve) causing ipsilateral pupillary dilatation. A contralateral hemiparesis occurs due to compression of the corticospinal motor tracts crossing over at the

level of the midbrain. As intracranial pressure increases signs of a more general nature are apparent, of which Cushing's response is the most well known (hypertension and bradycardia). If untreated, the opposite pupil enlarges, the patient becomes apnoeic, cardiovascular instability ensues as a result of brain stem herniation or "coning". This is followed shortly after by death. Signs of injury within the posterior cranial fossa may be quite subtle, and often initially manifest only as changes in respiratory pattern or activity. It is important therefore to record and observe the respiratory pattern in head injured patients in addition to respiratory rate.

## **ASSESSMENT AND MANAGEMENT**

### **Preparation**

The medical team leader must:

- Ascertain the mechanism of any injuries
- Establish the neurological state of the patient at the scene
- Identify any subsequent changes either with or without treatment
- Be aware of the presence of any other injuries

If the patient was noted to have been talking at any point since the injury, the primary brain injury is unlikely to be severe, but secondary injury could still be extensive. Sometimes, younger injury victims may develop malignant brain oedema, after apparently relatively trivial injury, and over the following few hours rapidly deteriorate and die. This group of patient who "talk and die" must be distinguished from those with an extradural haematoma (EDH).

Resuscitation should be carried out on a trolley capable of head down and head up tilt, in order that once stabilised, the head injured patient can be managed in a 15° head up position to reduce intracranial pressure (ICP).

### **Primary survey and resuscitation**

It is essential that the patient be managed using the approach described in Chapter 1. The life-threatening injury may be extracranial even if the head injury is thought to be significant.

## Airway and cervical spine control

The roles of the airway doctor and nurse are to:

- Maintain continuous verbal communication with the patient wherever possible
- Monitor and report to the team leader any changes in the ability to communicate with the patient
- Clear and secure the airway and maintain cervical spine control.
- Ensure adequate oxygenation at all times

Indications for tracheal intubation and mechanical control of ventilation following a head injury are listed in Box 7.1.

Attempting tracheal intubation may cause or worsen secondary brain injury if not performed in a controlled manner as a result of hypoxia, hypertension and an increase in ICP. Furthermore, the cervical spinal cord may also be injured (see below). It is therefore recommended that in a head injured patient, an individual experienced in the use of anaesthetic and neuromuscular blocking drugs performs tracheal intubation.

### **Box 7.1: Indications for intubation and ventilation following a head injury**

- Inability to maintain an adequate airway
- Risk of aspiration ie loss of laryngeal reflexes
- Inadequate ventilation:
  - Hypoxia       $\text{PaO}_2 < 9 \text{ kPa}$  breathing air  
 $\text{PaO}_2 < 13 \text{ kPa}$  breathing oxygen
    - Hypercarbia  $\text{PaCO}_2 > 6 \text{ kPa}$
    - To assist in acute reduction of ICP by hyperventilation
    - Spontaneous hyperventilation causing  $\text{PaCO}_2 < 3.5 \text{ kPa}$
    - Rapidly deteriorating GCS regardless of initial level or absolute GCS<9
    - Continuous or recurrent seizures
    - Need to transport a patient out of the Department
    - Development of complications eg. neurogenic pulmonary oedema, hyperthermia

Once intubation is achieved, it may be possible to temporarily reduce ICP by hyperventilating the patient and reducing their PaCO<sub>2</sub> to 4.5kPa. This causes vasoconstriction of cerebral arterioles, thereby reducing the volume of arterial blood in the head. However, if excessive it can severely reduce blood flow to already compromised areas, and exacerbate any injury. Close monitoring of arterial blood gases is required to prevent this occurring. Hyperventilation is generally reserved for those patients in danger of imminent coning after discussion with a neurosurgeon or with the aid of close monitoring in a neurosurgical unit. The risk of an associated cervical spine injury in an unconscious patient following a road traffic accident or fall is 5-10%. Manual inline stabilisation (MILS) is the preferred technique to maintain stability whilst intubation is being carried out, and consequently, a third person is required to maintain the neck in a neutral position whilst intubation is being performed. Leaving the semi-rigid collar in place limits mouth opening and makes tracheal intubation much more difficult (Chapter 3).

Once intubation has been completed, the airway team must ensure that the collar, tape and sandbags are reapplied. It is vital to ensure the collar fits adequately and the head is maintained in a neutral position, as constriction of the neck veins from too tight a collar or tube tie, or poor positioning can elevate the ICP from venous congestion.

### **Breathing and ventilation**

In the spontaneously breathing patient, the respiratory pattern and rate gives vital information and must be continually monitored. Because of the importance of avoiding hypoxia and hypercarbia, adequacy of ventilation is best assessed by arterial blood gas analysis. Thoracic injuries should be rapidly identified and appropriate action performed to ensure adequate oxygenation.

### **Circulation and control of haemorrhage**

A closed head injury is never a cause of shock in an adult patient. Children under 18 months (with open fontanelles) or adults with massive scalp injury may lose sufficient blood to cause shock, but other life threatening injuries must always be considered and excluded.

Assuming that the patient is *not in a shock*, then fluid administration should be confined to maintenance volumes of either normal (0.9%) saline or Hartmann's solution (compound sodium lactate). Hypertension must not be treated as this represents the body's attempt to maintain cerebral perfusion in the face of raised intracranial pressure. *In the shocked patient*, the aim should be to restore blood pressure to an appropriate level for the patient until haemorrhage is controlled and then aim for a systolic blood pressure of >110mmHg because of the known deleterious effects of hypotension on brain perfusion. An adequate volume is more important than the type of fluid used. Initially a bolus of warmed crystalloid or colloid is given, according to local protocols, with further fluid type dictated by the patient's response.

In cases of head trauma, dextrose-containing fluids (5% dextrose, 4% dextrose plus 0.18% saline) are avoided because:

- They reduce plasma sodium, thereby lowering the plasma osmolality and exacerbating cerebral oedema
- They cause hyperglycaemia, which is associated with a worse neurological outcome

It is vital that all fluids administered are recorded appropriately and early consideration should be given to early monitoring of the central venous pressure to guide fluid management.

### **Dysfunction of the CNS (neurological assessment)**

Once hypoxia and hypotension have been corrected then an assessment of the patient's conscious state should be performed and any localising signs identified using the Glasgow Coma Scale (GCS) and pupillary reactions. It is always better to record the observed response rather than the associated numerical value, as this is more useful when communicating to members outside the team e.g. neurosurgeons. This assessment must be repeated at regular intervals and deterioration in any one of the observed elements of the GCS must be reported to the team leaders.

## **Exposure and environmental control**

Hypothermia is usually more of a problem after removal of the patient's clothes, but hyperthermia can develop in response to the head injury and if detected should be treated by active cooling measures e.g. ice-bags, fans and pharmacological treatments e.g. rectal paracetamol if possible. Brain temperature may be 0.5-1°C higher than central (core) temperature and so any degree of hyperthermia may be considerably worse than predicted. There is no evidence that mild hypothermia is beneficial in the early stages of managing a head injury in the ED.

## **Team leaders**

Upon completion of the primary survey, the team leader should be satisfied that appropriate resuscitation is under way and all factors that contribute to secondary brain damage have been eliminated. In addition, the patient must be adequately monitored to ensure that any change in the patient's neurological status is detected early. Continual re-assessment of heart rate, blood pressure, respiratory rate, blood gas analysis and GCS and pupils is mandatory. An arterial line can often be helpful, allowing continual blood pressure and heart rate monitoring and frequent arterial blood sampling.

**Do not forget that extracranial injuries may be the cause of a neurological deterioration**

## **Secondary survey**

A DETAILED HEAD TO TOE EXAMINATION IS CARRIED OUT AS DESCRIBED IN CHAPTER 1. FEATURES SPECIFIC TO PATIENTS WITH HEAD TRAUMA ARE DESCRIBED BELOW. THE MEDICAL TEAM LEADER IS RESPONSIBLE FOR ENSURING THAT THE EXAMINATION IS COMPLETED AS FULLY AS POSSIBLE OR IF NOT COMPLETED THAT THIS IS ALSO RECORDED.

### **Scalp**

Examine the scalp for lacerations, bruising or swelling and digitally explore all cuts for a linear or depressed skull fracture in the base. Occasionally a haematoma in the loose areolar

layer can imitate a fracture. Any open fractures exposing brain tissue should not be explored, but covered with a clean dressing and left for expert assessment. Foreign matter protruding from the skull should also be left for removal by the neurosurgeons. Significant scalp bleeding should be controlled either by direct pressure on the edges or using haemostats to grip the aponeurosis and fold the scalp back on itself.

#### Neurological assessment

A more detailed examination needs to be performed including a repeat of the GCS, pupillary responses, and detection of any lateralising (focal) signs that may indicate intracranial injury. When utilised correctly, the GCS (Box 7.2) is a very useful tool for assessment and communication, but it does not detect focal injuries. The best responses in each section should be recorded.

#### **Box 7.2: Glasgow Coma Scale**

##### Eye:

Opens spontaneously	4
Opens to speech	3
Opens to pain	2
None	1

##### Verbal:

Orientated	5
Confused	4
Inappropriate words	3
Incomprehensible sounds	2
None	1

##### Motor:

Obeys commands	6
Localises to pain	5
Flexion (withdraws) to pain	4
Abnormal flexion to pain (decorticate)	3
Extension to pain (decerebrate)	2
None	1

#### *Common pitfalls:*

- Inability to open the eyes due to swelling does not automatically mean “no eye-opening”. Record that the assessment cannot be made.
- A response to pain is best elicited by applying pressure on the supratroclear nerve in the supraorbital ridge. A peripheral stimulus may not be sensed in the presence of a spinal cord injury.
- Localising to pain means that a hand reaches above the clavicle following the supraorbital stimulus (Fig. 7.1). Limb movements confined to below the clavicle represent “withdraws from pain”.
- Splints and painful fractures limit limb movement. This may cause differences between sides. Record the best side and indicate there is disparity.

- A verbal response cannot be assessed in an intubated patient. Record “patient intubated”

**A poor neurological response or deterioration should never be attributed solely to the presence of alcohol. The presence of intracranial pathology or secondary brain damage from hypoxia, hypotension, hypovolaemia or hypoglycaemia must always be considered.**



**Fig. 7.1: Pressure on the supratrochlear nerve to elicit response to a painful stimulus.**

As the patient's airway will be secure, regular reassessment of the GCS and pupils can be delegated to the airway nurse. The aim is to detect *any change* in neurological state that may indicate injury or worsening of the patient's condition, and so it is helpful to have the same person assessing these parameters each time.

Lateralising signs are a strong indicator of intracranial pathology. These are most often a unilateral weakness or asymmetry of motor or pupillary responses, and strongly suggest the presence of focal injury. In a conscious patient, upper-arm drift is a sensitive test of partial hemiplegia. The patient is asked to close their eyes and hold their arms out in front of them, palms facing upwards. Rotation of the arm so the palm faces downwards, is an early and sensitive sign and should be a cause for concern. Congenital unilateral pupillary dilatation may be present in 10% of the population, but the pupils on both sides should have normal light reflexes.

Fully conscious patients with apparently mild head injuries may also need assessment of their short-term memory to aid in decision-making regarding admission and discharge. *They should be able to recollect three objects shown to them 3 minutes beforehand.*

### **Base of skull**

Examine the patient to elicit any clinical signs of a basal skull fracture. The base of skull lies along a line joining the landmarks of the mastoid process, tympanic membrane and orbits, and a fracture is suggested by any of the findings in Box 7.3. In the acute situation, the later signs of fracture may not be present.

#### **Box 7.3: Signs of a fracture to the base of the skull**

*Early:*

- Haemotympanum
- Bloody CSF from the ear or nose
- Scleral haemorrhages with no posterior margin

*Late (occurring up to 12-24 hours after injury):*

- Bruising over the mastoid (Battle's sign)
- Orbital bruising ('panda' or 'raccoon' eyes)

Blood dripping from the nose or ear can be tested for CSF by dropping some of the fluid onto an absorbent sheet e.g. paper towel. If CSF is mixed with the blood, a double ring pattern will develop. The presence of CSF also delays clotting of any blood discharge, although this is not such a reliable sign.

Routine administration of antibiotics is not of proven value in a base of skull fracture, even if there is a CSF leak, indicating the presence of a compound fracture. Antibiotics are generally reserved for those patients with a depressed compound fracture to prevent meningitis and abscess formation. The antibiotics chosen will depend on local policy, and should be known by the team leaders.

A nasogastric tube should not be used if there is a fractured base of skull, as the tube may be pushed up into the skull vault. As a general rule, it is safer to use the orogastric route for gastric drainage in an unconscious head injured patient.

## **Eyes**

Penetrating injuries may occur through the orbits into the anterior cranial fossa. The eyes should always be inspected therefore for obvious trauma or haemorrhage and the pupils compared for size and reactivity.

## **Other injuries**

Cardiovascular instability in an unconscious trauma patient must always be investigated and treated prior to moving the patient for further investigation, for example CT scanning. Depending on local facilities, abdominal ultrasounds scan or diagnostic peritoneal lavage may need to be carried out in the Emergency Department to exclude occult abdominal injury.

## **Other relevant conditions**

### *Agitation*

This is common after head injury, and may indicate intracranial pathology, pain or hypoxia. Efforts should be made to detect and treat the cause as agitated patients are at risk of further injury to themselves. Sedative drugs should never be used as a first line treatment for agitation, as the cause of the agitation may be missed and secondary injury worsened. If after excluding other causes, the patient remains agitated, expert anaesthetic assistance should be sought early as the patient may need to be anaesthetised. Additional investigations, for example CT scanning may therefore be required as a result, since the ability to assess and monitor the patient is impaired.

### *Convulsions*

These may occur spontaneously in patients with epilepsy or more seriously indicate primary or secondary brain damage. Further brain damage can occur if the fits are left untreated due to the hypoxia and hypercapnia that can develop during fitting. An initial convulsion can be

treated with a slow intravenous bolus of diazepam to a maximum dose of 5-10 mg depending on age and size. If this fails or the fitting recurs, it is preferable to use a slow intravenous infusion of phenytoin at a dose of 15 mg/kg given over one hour, rather than give further diazepam. Phenytoin should never be given at a rate faster than 50mg/min as it can precipitate cardiac arrhythmias. All anticonvulsants can depress both the cardiovascular and respiratory systems so blood pressure and respiration should be closely monitored.

In cases of uncontrolled fitting unresponsive to phenytoin, an intravenous barbiturate, (commonly thiopental), will be required. An expert (often an anaesthetist) should administer this, as it will also necessitate intubation and controlled ventilation of the patient. Neuromuscular blocking drugs should never be given alone, as muscle paralysis does not terminate convulsions.

### **Completion of secondary survey**

At this point, the nursing and medical team leaders should ensure the following;

- The neurological state of the patient following injury, on arrival and any subsequent changes have been recorded
- They have identified and wherever possible, treated any factors causing secondary brain damage
- They have assessed and treated any associated injuries
- Any cervical injuries have been detected

### **Definitive care**

#### **Investigations**

This is partly determined by the stability of the patient. If for example hypotension due to a ruptured viscus is detected, it is imperative the patient undergoes a laparotomy to treat this prior to undergoing cranial CT scanning. If however the patient is stable after completion of the secondary survey, then it is appropriate to carry out a CT scan to determine the exact nature of any cranial and associated injuries. Box 7.4 lists the current UK recommendations for immediate CT scan issued by the National Institute for Health and Clinical Excellence (NICE). CT is the investigation of choice, as it allows earlier detection and possible earlier neurosurgical treatment of intracranial complications with an improved outcome.

**Box 7.4: NICE indications for immediate CT scanning after cranial trauma**

- GCS less than 13 at any time since injury
- GCS equal to 13 or 14 at 2 hours after injury
- Suspected open or depressed skull fracture
- Any sign of a basal skull fracture (haemotympanum, “panda eyes”, Battle’s sign, CSF otorrhoea)
- Post-traumatic seizure
- Focal neurological deficit
- More than one episode of vomiting (use clinical judgement if less than 12 years of age)
- Amnesia for more than 30 minutes of events before impact (not possible in very young children)

Also in patients with the following risk factors providing they have experienced some loss of consciousness or amnesia:

- Age equal to or greater than 65 years
- Coagulopathy (history of bleeding, known clotting disorder, warfarin therapy)
- Dangerous mechanism of injury (e.g. pedestrian hit by car, fall more than 1m or down 5 steps). Use a lower threshold for height of fall in young children.

If CT scan facilities are not available on an urgent basis, it may be necessary to transfer the patient to a hospital with these facilities. Performing skull x-rays prior to this may enable the team leaders to clarify the necessity for transfer (see Boxes 7.5 and 7.6).

**Box 7.5: Indications for performing skull radiographs**

*Unconscious patient or with neurological signs:*

- All patients, unless CT performed or transfer to a neurosurgical centre

*Orientated Patient:*

- History of loss of consciousness or amnesia
- Suspected penetrating injury
- CSF or blood loss from nose or ear
- Scalp laceration (to bone or >5cm long), bruising or swelling
- Persistent headache or vomiting
- Violent mechanism of injury eg fall, RTA, assault with weapon

**Box 7.6: Relative risk of intracranial haematoma in head injured patients with a skull fracture**

No skull fracture

- Orientated 1:5983
- Not orientated 1:121

Skull fracture

- Orientated 1:32
- Not orientated 1:4

### **Terminology applied to head injuries**

Head injuries are grouped into three general categories to aid further management and assessment.

#### **Minor head injuries**

These constitute the majority of those that attend the ED. This group of patients have:

- Minimal disturbance of conscious level (GCS 14-15)
- Amnesia <10 minutes duration
- No neurological signs or symptoms at the time of examination
- No skull fractures, clinically or radiologically
- A responsible adult at the place they are discharged to

They must be given appropriate written instructions e.g. head injury card.

#### **Moderate head injuries**

These patients require admission for observation and investigation. Wherever possible it is recommended they remain under the care of a local admitting team with experience of caring for head injuries. This will include patients with:

- Confusion or any depression of the level of consciousness (GCS 9-13)
- A skull fracture, on x-ray or clinically
- Difficulty in assessment eg alcohol, drug intoxication, epilepsy
- Patients with relevant co-existent medical disorders or treatment eg blood clotting disorders, anticoagulants

- Patients without a responsible adult to monitor them.

If there is any deterioration in their condition during admission their status must be discussed with a neurosurgeon.

### **Severe head injuries**

These patients need urgent neurosurgical referral. Such patients include:

- Coma (GCS<9) after full resuscitation
- Those with a skull fracture and neurological signs
- A compound or depressed skull fracture
- Basal skull fracture
- Post traumatic epilepsy
- Deteriorating consciousness or neurological state regardless of initial presentation
- Neurological disturbance lasting more than 6 hours
- Amnesia > 10 minutes
- Abnormal head CT scan

### **Referral to a neurosurgeon**

Any patient with potential or actual need for neurosurgical intervention, or where a CT scan cannot be performed within a reasonable time should be referred to a neurosurgeon. This will include all of those patients in the category “severe head injury” and those in whom a CT scan is indicated (Box 7.4) but cannot be arranged within 2-4 hours.

### **Communication with a neurosurgeon**

Not every patient discussed with a neurosurgeon will need to be admitted under their care, but advice about investigation and treatment can often be useful. Neurosurgeons require a detailed referral as indicated in Box 7.7.

**Box 7.7: Patient information needed by a neurosurgeon**

- Name, age and sex
- Time and mechanism of injury
- Neurological state at the scene (description)
- Any change in neurological status during transfer to hospital
- Initial assessment (ABCDE), other injuries
- Localizing signs of neurological injury or convulsions
- Treatment administered and any response
- Results of any investigations
- Relevant past medical history and medication

**Treatment to discuss with a neurosurgeon**

The following are recognised treatments for head injuries, but should not be used routinely and ideally after neurosurgical referral.

*Mannitol*

This is an osmotic diuretic agent that has a dual effect in reducing ICP. Its early effect is due to an improvement in cerebral blood flow by altering red cell deformability and size. It also reduces interstitial brain water by establishing an osmotic gradient and movement of water between brain tissue and blood. In areas where the blood brain barrier is damaged it can however leak into the brain tissue and increase the local water content. Initially, 0.5g/kg, of 20% mannitol (175 ml in 70 kg adult), is administered and the patient reassessed, e.g. for a reduction in pupil size. A urinary catheter is always required if not already in place. Repeated doses can cause hypovolaemia or electrolyte disturbances.

*Furosemide*

This is a potent diuretic that also reduces ICP by reducing brain water and the rate of CSF production. It can be used instead of mannitol at a dose of 0.5 mg/kg. The effect of furosemide can be extremely potent if used in conjunction with mannitol, and will cause hypovolaemia, hypotension and biochemical derangement.

### *Hyperventilation*

This has been discussed above. Its use should be confined to cases of imminent coning in conjunction with other treatments eg. mannitol.

Currently no neuroprotective drugs exist. The use of steroids should be avoided because of the risk of hyperglycaemia that is associated with a worse outcome.

### **Neurosurgery**

This is rarely required in the ED as it is preferable to transfer the patient to the neurosurgeon once stabilised. Burr holes should only be placed by a trained surgeon who has consulted a neurosurgeon.

### *Neurosurgical transfer*

Transfer to a neurosurgical unit should only occur after adequate resuscitation and stabilisation of any life threatening injuries. The escorting team should consist of a trained nurse and a doctor experienced in dealing with any patient deterioration in transit. If the patient is intubated, the doctor should be a trained anaesthetist. Preparation and equipment for transfer should be in accordance with accepted guidelines and is described elsewhere. Adequate records including vital signs and GCS must be maintained during transfer.

### **Summary**

Head injury management in the ED should be directed at identifying and treating the factors that cause secondary brain injury. Life threatening injuries to other systems need to be treated prior to transfer for further neurosurgical investigation or treatment. Co-ordination of the resuscitation team is vital in order to detect these injuries and prioritise their treatment. The majority of head injuries seen in the ED do not require admission or neurosurgical input. The team must be able to confidently detect those that do.

## **APPENDIX 7.1: ANATOMY**

A basic knowledge of neuroanatomy is important to understand some of the clinical signs that may be seen after a head injury.

The scalp is made up of five distinct layers. The subcutaneous layer is very vascular and open scalp wounds that breach this layer can cause considerable blood loss unless the scalp injury is repaired. Scalp haematomas of a considerable size can also develop in the looser areolar layer.

The white and grey matter of the brain is contained within the rigid box-like skull and bathed in cerebrospinal fluid (CSF). The interior of the skull base has many bony projections. To prevent brain injury, two perpendicular folds of dura mater prevent excessive movement; the falx cerebri separates the two cerebral hemispheres and the tentorium cerebelli separates the cerebral hemispheres superiorly, from the cerebellum inferiorly. The dura mater is one of three layers of tissue covering the brain, the others being pia and arachnoid.

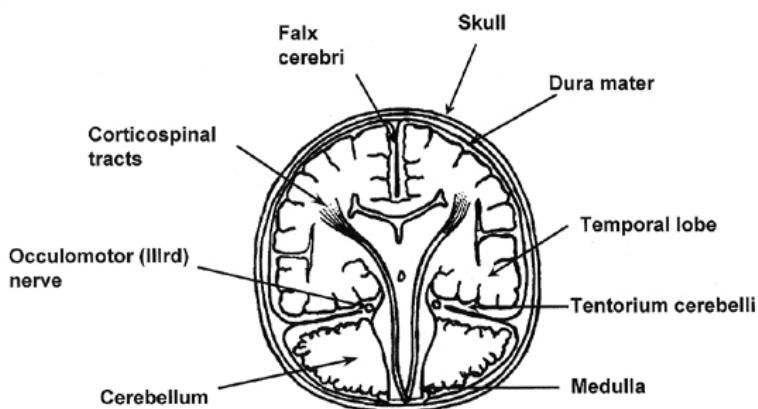
Arteries run between the dural folds and the inner surface of the skull. The most important is the middle meningeal artery, which lies beneath the temporo-parietal area of the skull. Bridging veins also run in the subarachnoid space, which is a CSF-filled space between the pia mater covering the brain and the arachnoid mater. Bridging veins carry blood from the brain to the venous sinuses that run in the dura mater.

The midbrain consists of the pons and medulla and passes through an opening in the tentorium cerebelli and continues at the level of the foramen magnum with the spinal cord. The oculomotor (III<sup>rd</sup>) nerves leave the anterior aspect of the midbrain, run forward between the free and attached edges of the tentorium cerebelli and go on to supply many of the extrinsic muscles of the eye. They also contain pre-ganglionic parasympathetic fibres that cause constriction of the ipsilateral pupil (see Fig. 7.2).

The brain is not a solid structure, within it there are a number of spaces (ventricles) containing CSF. Two lateral ventricles, one in each cerebral hemisphere, are connected to the third ventricle at the junction of the midbrain and the cerebral hemispheres. The third ventricle is in turn connected to the fourth ventricle at the level of the medulla.

The CSF is secreted by the choroid plexus in the lateral ventricles of each hemisphere, and passes through foraminae or channels in the brain before draining into the subarachnoid space at the level of the midbrain. In the healthy person, CSF communicates freely within the skull before being absorbed by folds of arachnoid villi in the walls of the venous sinuses.

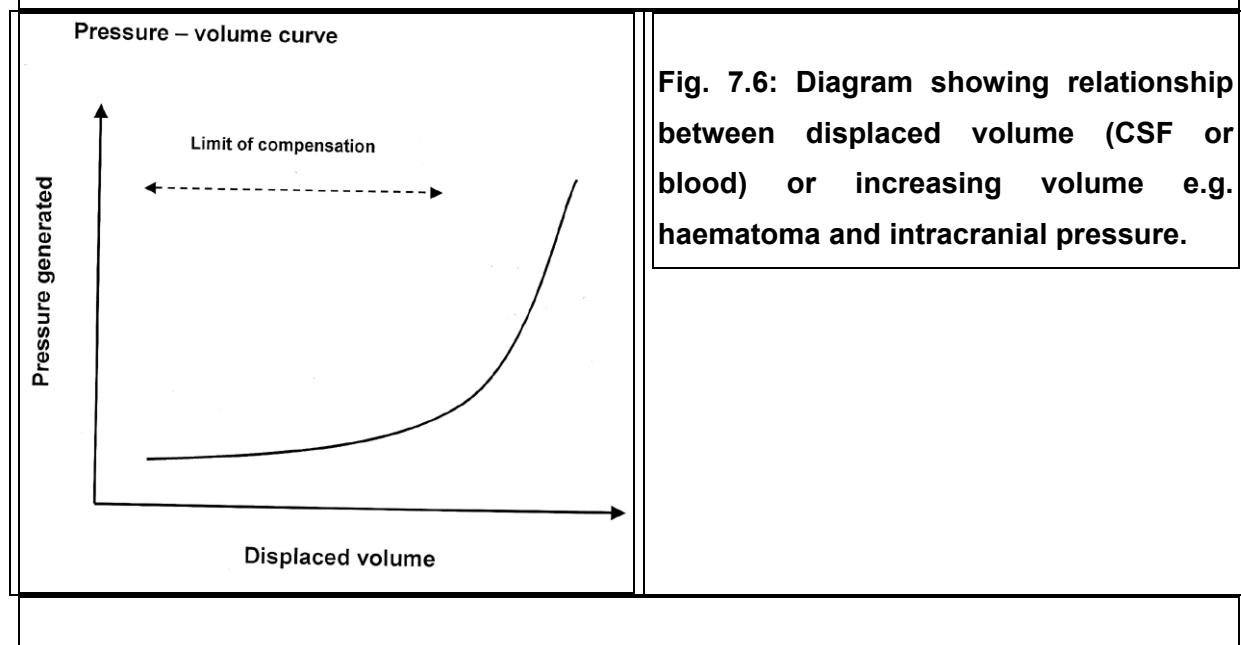
**Fig. 7.2: Coronal section of the brain and brain stem**



## APPENDIX 7.2: PHYSIOLOGY

### Intracranial pressure (ICP)

This is determined by the relationship between the skull, a rigid box of fixed volume and the volumes of the brain, CSF and blood. In health, small changes in the volume of CSF and blood occur in order to keep the intracranial pressure within the range of 5-13 mmHg. CSF can be displaced into the spinal CSF space or its absorption by the pia-arachnoid increased, and the volume of blood within the venous sinuses can change. The changes in blood and CSF volume are often referred to as indicative of the compliance,  $dV/dP$  (or more strictly elastance,  $dP/dV$ ) of the intracranial contents. Transient rises in pressure may occur e.g. due to changes in posture (bending over), sneezing, coughing etc. but these quickly return to baseline levels. Once the capacity to make these changes has become exhausted ie no further CSF or blood can be displaced, or if the volume of one of the contents within the skull rises very rapidly, e.g. an expanding intracranial haematoma, the compensatory mechanisms fail and intracranial pressure rises rapidly. This is often referred to as the Monro-Kellie principle (Fig. 7.6). The rate of rise of ICP is a direct function of the rate of increase in one of the volumes within the skull.



### Cerebral perfusion

Cerebral neurons require an almost continuous supply of oxygen and glucose. If blood flow is interrupted for as little as 4 minutes, neurons fail rapidly and die. Perfusion of the brain is dependent upon the pressure gradient across the vasculature and is termed the cerebral perfusion pressure (CPP). This is the difference between mean arterial pressure (MAP) and the mean venous pressure. The latter is difficult to measure and in health approximates to the more easily measured ICP:

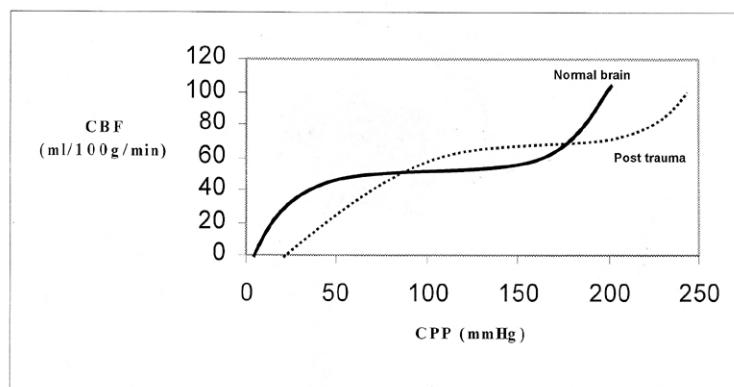
$$\text{CPP} = \text{MAP} - \text{ICP}$$

Cerebral perfusion pressure is reduced primarily by a reduction in MAP, an increase in ICP or both.

Cerebral venous pressure may also play a role in reducing CPP. When venous drainage of the brain is impaired for example by a tight tracheal tube tie or a patient coughing, venous pressure will be elevated above ICP and therefore reduce CPP.

Under normal circumstances, MAP and hence CPP varies, but blood flow to the brain must remain constant. This is achieved by a process termed autoregulation. The trigger to autoregulation is CPP; as CPP falls the cerebral arterioles dilate to maintain flow, as CPP rises they constrict to reduce flow, so that cerebral blood flow remains constant over a CPP range of 50-150 mmHg. As ICP rises, it becomes increasingly important to calculate CPP. An adequate CPP depends not only on a low ICP, but also on an adequate MAP. The threshold for developing cerebral ischaemia varies with the type of neuronal tissue, but *on average* occurs if the CPP falls to 50 mmHg or less *in a normal person*. Following a head injury the threshold for developing ischaemia is often much higher and occurs at cerebral perfusion pressures of 60-70 mmHg (Fig. 7.7). This is due to a disruption of cerebral autoregulation in the early stages after a head injury.

In the first few hours, cerebral blood flow falls although the metabolic requirements of the neurones are unchanged. Consequently, a higher cerebral perfusion pressure is needed to maintain an adequate cerebral blood flow to prevent ischaemia and neuronal death.



**Fig. 7.7: Relationship between cerebral blood flow (CBF) and cerebral perfusion pressure (CPP) in normal and injured brain**

## **Appendix 7.3: Types of head injury**

### **Direct injuries**

These result from contact with a hard object. Contact at the point of impact may deform the skull producing a linear fracture, and considerable underlying brain injury. Depressed fractures occur when bone fragments enter the cranial cavity. Compound fractures occur when there is direct communication between an open scalp laceration and the meninges or brain substance. A basal skull fracture is a special form of compound fracture whose presence requires careful treatment (see later). A contusion may develop in an area of brain lying under the impact point of a contact force. The bone is deformed inwards, and shock waves distribute out from the point of impact, producing haemorrhage, brain oedema and neuronal death. This may also occur when the brain contacts the inner aspect of the skull base. Patients usually lose consciousness at the scene of the accident, and focal signs have often developed by the time of arrival in the ED.

### **Inertial injuries**

Inertial forces can produce injuries that result in a significantly worse outcome than damage resulting from direct contact. A spectrum of injury exists ranging from concussion (the mildest) to diffuse axonal injury (the most severe).

#### *Concussion*

- May occur after minor inertial forces to the head
- The patient is always amnesic of the injuring event
- There may be amnesia for events before (anterograde amnesia) or afterwards (postgrade amnesia)
- Consciousness may have been lost for up to 5 minutes
- There may be nausea, vomiting and headaches
- The patient does not have localising signs
- Recovery is complete
- Microscopic structural brain damage occurs, and so the effect of numerous episodes can be considerable.

#### *Diffuse axonal injury (DAI)*

- Occurs after a rapid acceleration or deceleration of the head
- Is associated with a high transfer of energy eg. shaking a child, motor vehicle accident
- Causes deformation of the white and grey matter of the brain
- Leads to axonal damage, microscopic haemorrhages, tears in the brain tissue and the subsequent development of oedema

- Severe DAI can cause immediate coma at the scene of the accident and has an overall mortality of 33-50%.

### **Contre coup injuries**

**These are brain injuries that occur away from the site of impact. The head undergoes an acceleration/deceleration force, the skull and brain move in the direction of the force producing injuries opposite the point of impact by percussion effects. Greater damage develops furthest from the impact point as the brain collides with the inner skull or skull base.**

### **Haematomas**

These can occur outside the dura, (extradural) or beneath the dura (intradural).

Extradural haematoma (EDH) (Figs. 7.3 & 7.4)

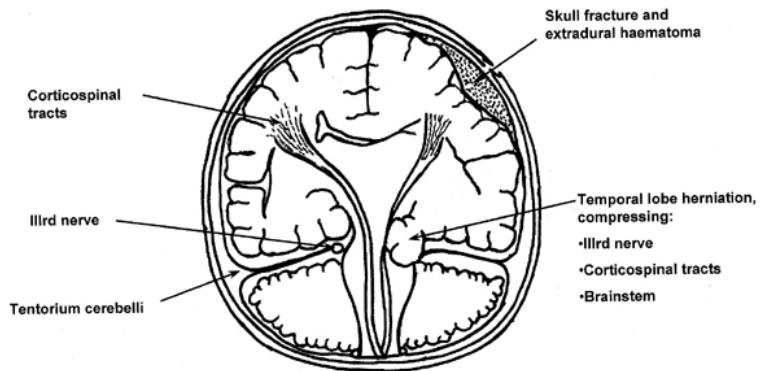
Typical features of an EDH:

- Associated with a fractured skull in 90% of cases
- Most often develops in the temporo-parietal area following a tear in the middle meningeal artery, (rarely due to a tear in a venous sinus)
- Develops quickly as the source of bleeding is arterial (middle meningeal artery)
- The classical presentation of an EDH (Box 7.8) occurs in only one third of cases
- The commonest clinical signs are a loss of consciousness and pupillary changes, although these can develop rapidly and late

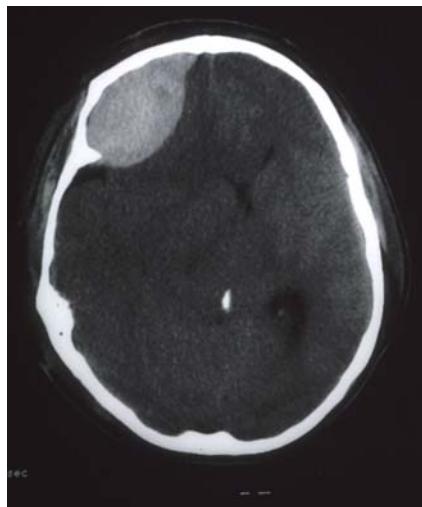
An EDH is a neurosurgical emergency, as early evacuation will result in a better patient outcome by reduction of secondary injury of the underlying brain.

#### **Box 7.8: The classic history of an extradural haematoma**

- Transient loss of consciousness at the time of the injury from a momentary disruption of the reticular formation
- Recovery of consciousness, may last several for several hours, the “lucid period”
- Gradual reduction in the level of consciousness
- Development of localising signs:
  - Pupillary dilatation
  - Contralateral hemiparesis
  - Hypertension and bradycardia due to rise in ICP



**Fig. 7.3: Coronal section of brain showing extradural haematoma causing herniation of medial temporal lobe that gives rise to the clinical signs.**

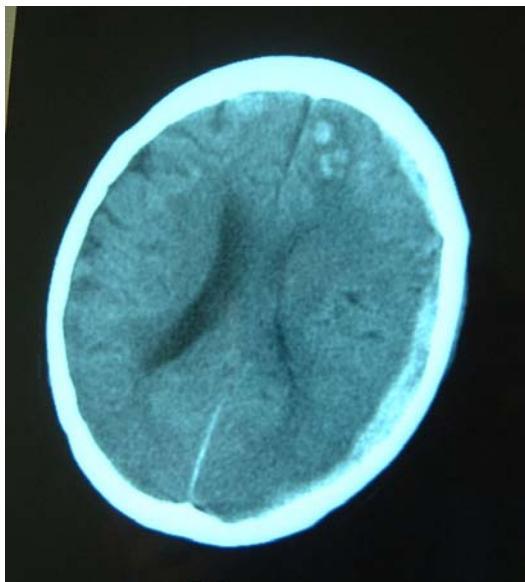


**Fig. 7.4: CT scan showing extradural haematoma. Note typical biconvex appearance.**

#### Acute intradural haematomas (IDH) (Fig. 7.5)

Typical features of an IDH:

- **Can be either subdural (SDH) or intracerebral (ICH)**
- **Both often coexist in the same patient**
- **Are three to four times more common than extradural haematomas**
- **Are produced by inertial or rotational forces, although considerably more force is needed to produce an ICH.**
- Are often associated and more severe in patients with disordered coagulation (e.g. therapeutic anticoagulation, chronic alcohol ingestion).



**Fig. 7.5: CT scan showing subdural haematoma. Note typical concave-convex appearance. Also small frontal contusions.**

Subdural haematomas develop when the bridging veins are torn and blood collects in the subdural space, commonly over the temporal lobe. Clinical deterioration of the patient can be very slow (up to several days). This type of haematoma is more common where there is pre-existing cerebral atrophy eg. the elderly, as the bridging veins are more likely to tear and a considerable blood collection can form in the space. Early evacuation within 4 hours of deterioration reduces mortality and outcome so early neurosurgical referral is vital.

Intracranial haematomas are produced by much larger forces than SDH, and are rarely found in isolation. Cerebral contusions and lacerations are often present, and as a result, mortality is considerably higher than SDH. Patients frequently lose consciousness at the time of injury, or may quickly develop seizures or focal signs.

#### *Subarachnoid haemorrhage (SAH)*

This may occur following head trauma and is associated with a worse mortality and outcome following TBI. Although often an incidental finding on CT scan following severe trauma, it is important that it is differentiated from aneurysmal arachnoid haemorrhage. The classical presentation of headaches, photophobia or other signs of meningism are far more suggestive of aneurysmal than traumatic SAH. If the former is suspected, the patient needs urgent neurosurgical referral. Management of patients with traumatic SAH should be as for any other head injured patient.

#### **Secondary brain injury**

This is damage that occurs after the primary brain injury as a result of the factors identified earlier. It is estimated that up to 30% of deaths after head injury are directly due to secondary injury.



## Chapter 8

# SPINAL TRAUMA

### Objectives

At the end of this chapter the reader should understand:

- The epidemiology of spine and spinal cord injury
- The key features of the primary and secondary survey in patients with vertebral column trauma and spinal cord injury
- The principles used to rule out injury to the cervical spine

The anatomy of the spine and the spinal cord is covered in Appendix 8.1, mechanisms of injury in Appendix 8.2 and a system for interpretation of cervical spine x-rays in Appendix 8.3. These are to be found at the end of the chapter.

### Introduction

The statistical risk of spinal trauma and spinal cord injury (SCI) after trauma is relatively low; approximately 3% of patients with multiple injuries sustain spinal trauma. However, the potential for life-long morbidity is high and the goal of prehospital and early in-hospital management of patients suffering from SCI must be to prevent further neurological damage as a result of uncontrolled movement of the injured spine or secondary injury from hypoxia or hypotension. Thus, great caution must be taken with these patients.

Despite the relatively low incidence, spinal trauma and SCI are a major cause of mortality and morbidity in young individuals and as a result have a major impact on society as a whole. Therefore, it is essential that initially all trauma patients are managed as if they have an underlying spinal or SCI until it has been excluded. The initial assessment and management of patients suffering from spinal injury and/or SCI is a challenging and difficult problem.

## Epidemiology of spine and spinal cord injury

The annual incidence of SCI including prehospital fatalities has been estimated at 43-77 per million inhabitants in the United States, which results in between 12 – 23,000 people suffering SCI every year. About 20% of these individuals die before they are admitted to the hospital. In the European Union, about 10,000 people are estimated to suffer from SCI every year. Of these patients 50-70% are between 15 and 35 years of age and 4-14% are 15 years old and younger. The male-to-female ratio is 4:1 and 15-20 per million of the population will suffer from major residual paralysis. In the USA, around 200,000 people live with a SCI with an estimated cost of therapy for these patients of over US\$ 4 billion per year. Clearly, vertebral column trauma and SCI are devastating and debilitating conditions that involve enormous financial, social, and personal costs, and affects all regions of the world.

The aetiology of spine and SCI varies worldwide, but overall, the most frequent cause in all age groups is road traffic accidents, which accounts for approximately 40-50% of all patients. In road traffic accidents, the exact nature of the injury varies according to the direction and the speed of the vehicle at impact, the victim's position, and the presence or absence of seat belts and airbags. The next most common causes are falls (21%), acts of violence (15%), and sports-related injuries (13%). In children, a higher percentage of SCI are due to sports (24%) and water recreational (13%) activities. In any given population, the prevalence of vertebral column trauma and SCI also depend upon both the recreational facilities and the popularity and activity of the various sports clubs.

### **INJURIES ASSOCIATED WITH SCI**

Up to 60% of patients suffering from spinal trauma will also have other injuries and 5-10% of patients suffering from severe traumatic brain injury (TBI) will have a SCI. Conversely, 25-50% of patients with SCI have an associated head injury. Moreover, spinal and SCI occur in 10-30% of patients with multiple injuries and SCI may occur in up to 30% of patients with abdominal and thoracic trauma causing severe haemorrhage when it is associated with a much greater fatality rate than either injury alone. Therefore, the pre-hospital and early in-hospital management of patients with severe injuries should always be conducted in accordance with the principles already described, namely the identification and treatment of immediately life-threatening injuries, but at all times bearing in mind the possibility of and need for management of spinal and SCI.

## **Assessment and management**

### **Initial reception**

There has been significant improvements in the care of patients with spinal injuries at both pre-hospital and in-hospital levels as a result of:

- Recognition of the importance of the mechanism of injury
- Triage to facilities with SCI expertise
- Advance warning to the receiving hospital of a patient with either a spinal, head or multiple injuries
- The widespread use of full spinal immobilization, including semi-rigid collars, spinal boards and vacuum mattresses.
- Advances in medical, surgical, and rehabilitative care

Injuries are however still missed, particularly in either the young or the elderly. Other causes of missing a vertebral column injury are shown in Box 8.1. It is worthwhile remembering the statistical chances of dealing with a spinal injury are approximately 3% in a multiply injured patient and 10% in patients with a head injury. Around 50% of patients with a spinal cord injury will also have another potentially life threatening injury as well.

**The team leader must ensure that all team members are aware of the potential for spinal injury.**

The goal of in-hospital management of SCI is to prevent any additional loss of neurological function while maximising the chances of recovery. This is achieved by following the principles of:

- Primary survey and resuscitation with appropriate patient immobilisation
- Detailed secondary survey including physical evaluation of the patient and radiological examination
- Definitive care (intensive care unit, surgery, and other procedures)

Information about the mechanism of an accident is required in order to identify patients with potential spinal trauma and SCI. This may come from the patient themselves and/or the pre-hospital team. The team leader must ensure that all team members are aware of the potential for vertebral column and spinal cord injury in the following circumstances:

- Conscious patient complaining of severe neck and back pain
- Conscious patient unable to move or feel either arms or legs
- Patients with severe facial injuries
- Patients involved in high-speed road traffic accidents
- Patients suffering from falls from a height
- Unconscious patients
- Intoxicated patients (drugs and/or alcohol) and trauma
- Patients with severe head, abdominal, thoracic, or multiple injuries

- Patients with distracting injuries
- Patients with neurodegenerative disorders e.g. dementia, Alzheimer's disease.

In all these groups the presence of an underlying spinal and/or SCI should be assumed until positively excluded by both clinical and radiological examination. Moreover, it is vital for the team leader to ensure that patients are not subjected to any undue movement of the spine and that they are lifted and moved in one, thereby protecting the spinal cord against any additional secondary injury.

**Box 8.1: Common causes of missing vertebral column or spinal cord injury**

- Unconsciousness
- Distraction injuries (e.g. haemorrhage, extremity fractures, burns)
- Lack of pain or deformity of the spine
- Inadequate interpretation of X-rays
- Spinal cord injury without radiological abnormalities (SCIWORA) (Appendix 8.2)
- Failure to consider the possibility of spinal injury

### **Primary survey and resuscitation**

Spinal injuries are frequently identified early, often before arrival in the ED. Whilst acknowledging this information, the team should not be distracted from the routine of the primary survey and the team leader must ensure that the basic rules of assessment and resuscitation are followed to prevent deterioration secondary to hypoxia and hypotension.

### **Transfer of the patient on arrival in hospital**

Most patients in whom spinal injury is suspected will arrive at hospital fully immobilised. However, for those patients not appropriately immobilised on a long spine board, there are two ways of transferring the patient onto the ED trolley.

- An ambulance scoop stretcher can be inserted beneath the patient, the head and neck immobilised and then the patient lifted over.
- When this device is not available a minimum of five people will be required to manually transfer the patient. It is vital that the team has been fully trained in the procedure and knows only to act on the direct instructions from the person controlling the stabilisation of the head and the cervical spine. Using his hands and forearms this person controls the neck and head of the patient whilst three other members position themselves for lifting, one for the thoracic spine, one for the lumbar spine and pelvis and one for the legs. On the controller's command all four gently lift the patient and the fifth member removes the trolley.

It is also useful to have an additional, sixth member, to hold IV infusions and monitors whilst the patient is being transferred. At no time during the course of this manoeuvre should the patient be subjected to a bending or twisting force.

### Airway and cervical spine control

The first and most urgent priority is to secure the airway as described in Chapter 3. At the same time, the neck must be stabilised in a neutral position, without any distracting force being applied. This is best achieved by applying manual in-line stabilisation (MILS), in order not to compromise airway management. If the victim is still wearing a motorcycle crash helmet, it should be removed by two skilled operators; one expands the helmet laterally and gradually “rocks” the helmet off the head until it can be rotated free, while the other person immobilises the cervical spine from below. In the conscious patient whose airway is clear, a semirigid collar, blocks and tapes can replace the nurse to immobilise the cervical spine. However some patients are likely to be anxious and at times claustrophobic from their presence and the nurse must continue to reassure, to give hope and to explain what is happening. If at any time it becomes necessary to remove the collar and blocks then it is essential that MILS must be reapplied.

Three groups of patients with spinal injuries require urgent tracheal intubation:

- Unconscious patients. This group develop a paralytic ileus rapidly and an incompetent gastroesophageal sphincter. This combination, with a potentially full stomach, puts them at a high risk of regurgitation and aspiration.
- Patients with signs of a high cervical cord injury. Complete injury above the C3 level leads to apnoeic respiratory arrest and death unless immediate ventilatory assistance is provided.
- Patients with other/associated major injuries. This will include chest head and abdominal injuries.

Intubation in these individuals is more difficult because of reduced neck movement and is best performed by an experienced anaesthetist, who may require specialized equipment for example, a fiberoptic laryngoscope.

Uncontrolled attempts to intubate the trachea in patients with an unstable cervical spine can cause or exacerbate SCI and even lead to the death of the patient. To reduce the risk of new or further neurological deficit, the patient must be intubated with great care and the benefits must be weighed against the risks. In particular, hyperflexion and hyperextension of the cervical spine must be avoided. The role of rapid sequence induction for intubation of the trachea in the trauma setting by trained staff is crucial and is the technique of choice in

patients suffering from spinal trauma in order to reduce coughing and spontaneous movements. In patients with SCI the use of the depolarising neuromuscular blocking drug succinylcholine (suxamethonium) may cause a bradycardia. Furthermore, 48 hours **after** SCI, its use may also cause cardiac arrest secondary to hyperkalemia.

Studies on cadavers have shown that the use of MILS results in significantly less antero-posterior displacement during orotracheal intubation than leaving the rigid collar in place. Furthermore, with the collar in place, mouth opening is reduced which makes intubation more difficult and increases the risk of airway compromise and aspiration. It must be remembered that although MILS helps ensure mechanical stability of the spine and reduces cervical spine movement during intubation of the trachea, it will not totally prevent it. Emergency drug assisted tracheal intubation with MILS requires a team approach (Fig. 3.16). After successful intubation of the trachea, the rigid cervical collar should be reapplied. Alternative airway devices such as a laryngeal mask, an intubating laryngeal mask and a Combitube are useful alternatives for airway management when the team members lack advanced airway skills, but they may exert greater pressure on the cervical vertebrae than conventional intubation techniques. Therefore, these devices should only be used when routine orotracheal or fiberoptic intubation is not possible. In all patients with SCI without life-threatening conditions compromise of their airway, fiberoptic tracheal intubation in combination with MILS is increasingly accepted as the optimal technique.

After securing the airway as close to 100% oxygen as possible should be given either via a non-rebreathing mask with a reservoir or mechanical ventilation. The neck should be inspected for the presence of:

- Swelling, bruising and wounds
- Subcutaneous surgical emphysema
- Deviation of the trachea
- Distended neck veins
- Laryngeal crepitus

### Breathing and ventilation

The chest is examined as already described looking for immediately life-threatening thoracic conditions. The finding of bruising from seat-belts or a fractured sternum in a road traffic accident victim should raise suspicion of thoracic spinal injury. Early considerations must be given to arterial blood gas analysis to assess the adequacy of oxygenation and ventilation. Specific problems associated with SCI are acute respiratory failure and hypoxia caused by hypoventilation due to impaired diaphragmatic function as a consequence of injuries of the upper cervical region (C3-C5). Diaphragmatic breathing may be the first clue to a significant

injury of the cervical spinal cord. When SCI spares the diaphragm but paralyzes the intercostal and abdominal muscles, there may be:

- Inadequate coughing
- Paradoxical rib movement on spontaneous ventilation
- Decrease in vital capacity (50%)
- Reduced functional residual capacity (85% of predicted values)
- Loss of active expiration

Intubation of the trachea will be required, but there is usually time to obtain expert assistance to perform this.

### Circulation and control of haemorrhage

Assessment and management are based on the principles described in Chapter 5. The presence of shock should not be assumed to be due to SCI, (ie neurogenic shock, see below). Although it may be possible to distinguish neurogenic from hypovolaemic shock, they are often combined and severe haemorrhage from co-existing injuries can complicate and exacerbate the degree of hypotension. Whatever the cause, resuscitation will be required using the principles already described. The effects of hypotension in SCI on neurological outcome are unknown, but extrapolation from the data on traumatic brain injury would suggest that hypotension is a frequent cause of secondary neurological injury. Therefore, it is essential that hypotension is avoided in these patients.

Major external haemorrhage should be controlled by direct pressure, two large intravenous cannulae are inserted and blood is taken for grouping and cross matching and any other appropriate tests. The patient's respiratory rate, pulse and blood pressure, capillary refill time, color and level of consciousness should be recorded. The type and rate of fluid infused will need to be judged according to the circulatory status of the patient and the presence of associated injuries.

In patients suffering from SCI, care is required to ensure optimal fluid resuscitation. Too little and tissue ischaemia will increase, too much may precipitate pulmonary oedema. The aim should be to maintain a mean arterial blood pressure (MAP) of 80-90 mmHg; any episode of hypotension (SBP less than 90 mmHg, MAP less than 70mmHg) should be avoided or corrected as quickly as possible. Those patients who respond inadequately to fluid resuscitation and remain bradycardic the diagnosis of neurogenic shock must be considered (see below).

For fluid resuscitation, crystalloids or colloids can be given although it is still a matter of debate as to which is preferred. For detailed discussion of the merits of different fluids, see Appendix 5.2. Whichever fluid regimen is used, fluids containing glucose should be avoided for two reasons:

- Rapid metabolism of glucose results in “free water” which supports oedema formation
- The risk of hyperglycaemia with an increase in lactate and decrease in pH

Clinical studies on the effects of elevated blood glucose levels in SCI do not exist but data from a number of settings, including stroke, cardiac arrest and critical care have shown that elevated blood glucose levels are associated with poorer outcomes. It would seem reasonable, based on an extrapolation of data obtained in these groups of critically ill patients, to aim for a target blood glucose level within the normal range (4.0 – 7.0 mmol/l). Intravenous glucose administration is only necessary in cases of acute hypoglycaemia. Glucose levels should be measured and treated as soon as possible during in-hospital management.

From the currently available clinical data it is not yet clear whether hypertonic or hyperosmotic (HHS) solutions as part of a “small volume resuscitation” technique provide a clinical benefit in the management of patients suffering from SCI. Extrapolating from the findings in patients with traumatic brain injury, the use of HHS may be justified and not harmful in patients in whom hypotension or multiple trauma is combined with SCI. However, controlled clinical trials are still lacking.

#### *Neurogenic shock*

This is defined as vascular hypotension associated with a Bradycardia as a result of spinal cord injury. It occurs following injury to the spinal cord above T6 and results in a progressive loss of sympathetic outflow with an ascending level of the lesion. The loss of vasomotor tone causes hypotension secondary to arteriolar and venous vasodilatation of the peripheral vasculature and the splanchnic vascular beds. When the lesion is above T2, there will also be a bradycardia secondary to interruption of the sympathetic innervation of the heart. These pathophysiological changes cause pooling of blood in the extremities and reduction of central venous return. The higher the lesion, the greater will be the loss of vasomotor tone and peripheral vasodilatation. Thus, neurogenic shock may be associated with a systolic blood pressure (SBP) below 70 mmHg and with severe bradycardia below 60 beats per minute. The presence of bradycardia associated with hypotension in an unconscious patient may be the only indication of a significant SCI. Moreover, such a patient cannot control and maintain body temperature or mount a normal response to any co-existing hypovolaemia caused by

other injuries. The lack of any sympathetic activity may be unmasked as profound parasympathetic reflexes such as severe bradycardia during laryngoscopy.

These patients may require the administration of vasopressors and invasive haemodynamic monitoring. A central venous line should therefore be installed early to help monitor and guide the response to fluid challenges. Although elevation of the patient's legs can be used to counteract peripheral venous pooling, it will not be possible in the presence of pelvic, lower limb or lumbar spine injuries. Expert help from spinal surgeons and intensivists will be required to ensure optimal ongoing resuscitation. Atropine should be used only in the emergency situation. It causes drying of the mucous membranes, thickens secretions and may cause or worsen a paralytic ileus.

### **Dysfunction of the CNS (neurological assessment)**

The team leader should assess the patient's level of consciousness with a full GCS assessment and a check of the pupillary light responses. During this assessment it may become apparent that there is a symmetrical weakness. This should be noted, but the full definitive neurological assessment must wait until the secondary survey.

#### ***Spinal shock***

This describes the conditions seen after SCI when there is a complete but transient loss of; sensation, muscle tone, muscle power and reflex activity (flaccid areflexia) below the level of the cord injury and rectal tone. Spinal shock may last for a variable length of time (days or weeks), but there is a potential for full recovery. The patient's cardiovascular status and ability to respond to insults is normal.

Rectal tone is classically evaluated by testing the bulbocavernosus reflex, mediated via the S2-4 region of the conus medullaris. It is frequently absent for the first 4-6 hours after cord injury, returning within 24-hours. If the reflex does not return, there is no evidence of cord function below the level of injury, including absence of sacral sparing; no determination of the completeness of the lesion can be made. Areas of the cord that are permanently damaged will eventually resolve to reveal a spastic weakness. A patient with complete transection of the spinal cord will eventually regain exaggerated reflex activity and a lack of power and sensation below the level of the lesion.

#### ***Exposure and environmental control***

The patient should be completely divested of all remaining clothes to allow them to be fully examined, while not forgetting their dignity. All patients cool rapidly once exposed, but particularly those with spinal cord injury due to the associated vasodilatation. Every effort

must be made to minimise heat loss using blankets, warm air blowers or overhead heaters. Conversely, hyperthermia must be avoided as it is associated with increased neurological injury. The overall aim should be for normothermia. At all times it is essential to ensure there is no undue movement of the spine, this is usually achieved using a co-ordinated log roll. In the conscious patient it is essential that any manoeuvres and all examination procedures are fully explained beforehand. During the course of the log-roll the opportunity can be taken to remove the spinal board and a rectal examination can be performed.

### Patient immobilization

Historically, it is estimated that up to 25% of SCI may be aggravated after the initial insult, either during transport or in the early course of prehospital and in-hospital treatment.

However, these data are more than 20-years old, and no data are available from current studies. Careful movements and the use of appropriate immobilization techniques are crucial in all trauma patients with SCI or when the mechanism of injury has the potential to cause spinal and SCI.

Immobilization of the entire spine is a management priority in the prehospital setting and in the early in-hospital course and must be performed in a systematic fashion. This consists of immobilization in a neutral spine position using a semi-rigid cervical collar and headblocks along with either a rigid backboard with head straps or a vacuum splint. Although immobilization devices are generally effective in limiting motion they can be associated with significant morbidity including patient discomfort, pressure sores and restriction of ventilation. Therefore, immobilization devices should be removed as soon as any lesion of the spine and/or the spinal cord is excluded with certainty after in-hospital radiological examinations have been performed.

Conscious patients suspected of having spinal injury but who are confused, restless, and agitated and refuse to lie down can present a problem. On no account should such patients be forcibly held down but rather reassured, allowed the freedom to move, and encouraged to keep the cervical collar on if possible. The muscle spasms associated with spinal injury do result in patients instinctively holding the head and neck still and avoiding movement. It is therefore unusual for patients to worsen spinal injury by their own voluntary movement. Causes of restlessness can be pain, fear, a full bladder, or not understanding the language. The team must try to identify and to treat the cause of the restlessness.

### Pharmacological intervention with corticosteroids

Some experimental studies have suggested that treatment with methylprednisolone (MPS) may be beneficial in SCI (e.g., cell membrane stabilization, inhibition of lipid peroxidation,

reduction of free radicals, increase blood flow, and reduction of oedema and inflammation). The clinical use of MPS was investigated in the United States in three National Acute Spinal Cord Injury Studies (NASCIS) conducted in the 1980s and 1990s. However, from a scientific point of view, even after NASCIS 1 to 3, it is still questionable as to whether treatment with MPS is beneficial in patients suffering from SCI. Moreover, MPS-treated patients have an increase in clinically important side effects e.g., severe pneumonia and wound infections (see also CRASH trial in patients with traumatic brain injury). In 2004, the National Association of Emergency Medical Services Physicians (NAEMSP) stated that the evidence in use of high-dose steroids for SCI remains inconclusive, the treatment with steroids should not be considered the standard of care, and routine use of steroids is not supported.

## **Secondary survey**

As already described, this consists of a head-to-toe examination of the patient to detect any injuries that were not immediately apparent during the primary survey. It may only be at this phase of the patient's care that a vertebral column injury or SCI becomes apparent. There are some occasions when the secondary survey needs to be delayed because of the need for surgery, for example to control haemorrhage or for transfer to the ICU. Whatever the reason, if the secondary survey is not completed in the Emergency Department, this must be clearly documented in the patient's notes. The team leader should also remind the clinicians responsible for the inpatient care on handover. All too commonly as a result of the lack of a detailed secondary survey injuries that are eminently treatable are missed and go on to produce life long problems long after the immediate life threatening conditions have been long forgotten.

The remainder of this section will concentrate on those aspects of the secondary survey that relate to the management of patients with spinal injuries. In the conscious patient, a number of signs and symptoms are associated with the presence of spinal injury:

- Pain in the spine at the level of the injury worsened with movement
- In the absence of pain tap the patient' s heels or ask them to cough ; this may reveal a painful area
- Abnormal or absent sensation
- Ignorance of other injuries, particularly fractures, in the absence of intoxicants
- Presence of weakness or inability to move a limb or limbs

The spine should be inspected with great care and kept in a neutral spine position at all times by using a log roll. Superficial lacerations, wounds, bruising, swelling and deformity are

external signs of vertebral column injury. Palpation of the entire spine from occiput to coccyx is performed to identify any tenderness, steps, deformations, and gaps between the spinous processes. Any indication of the potential presence of vertebral column injury mandates appropriate radiological investigation, according to local protocols. The incidences of findings in vertebral column injury are shown in Box 8.2.

**Box 8.2: Incidence of findings in vertebral column and spinal cord injury**

Lumbar Pain	37%
Head injury	36%
Altered mental status	31%
Cervical pain	15%
Neurological deficit	15%
Back pain	13%
Thoracic pain	11%
Spinal tenderness	8%

### Neurological function

In conscious patients, a neurological examination must also be performed on both sides to detect any abnormalities of:

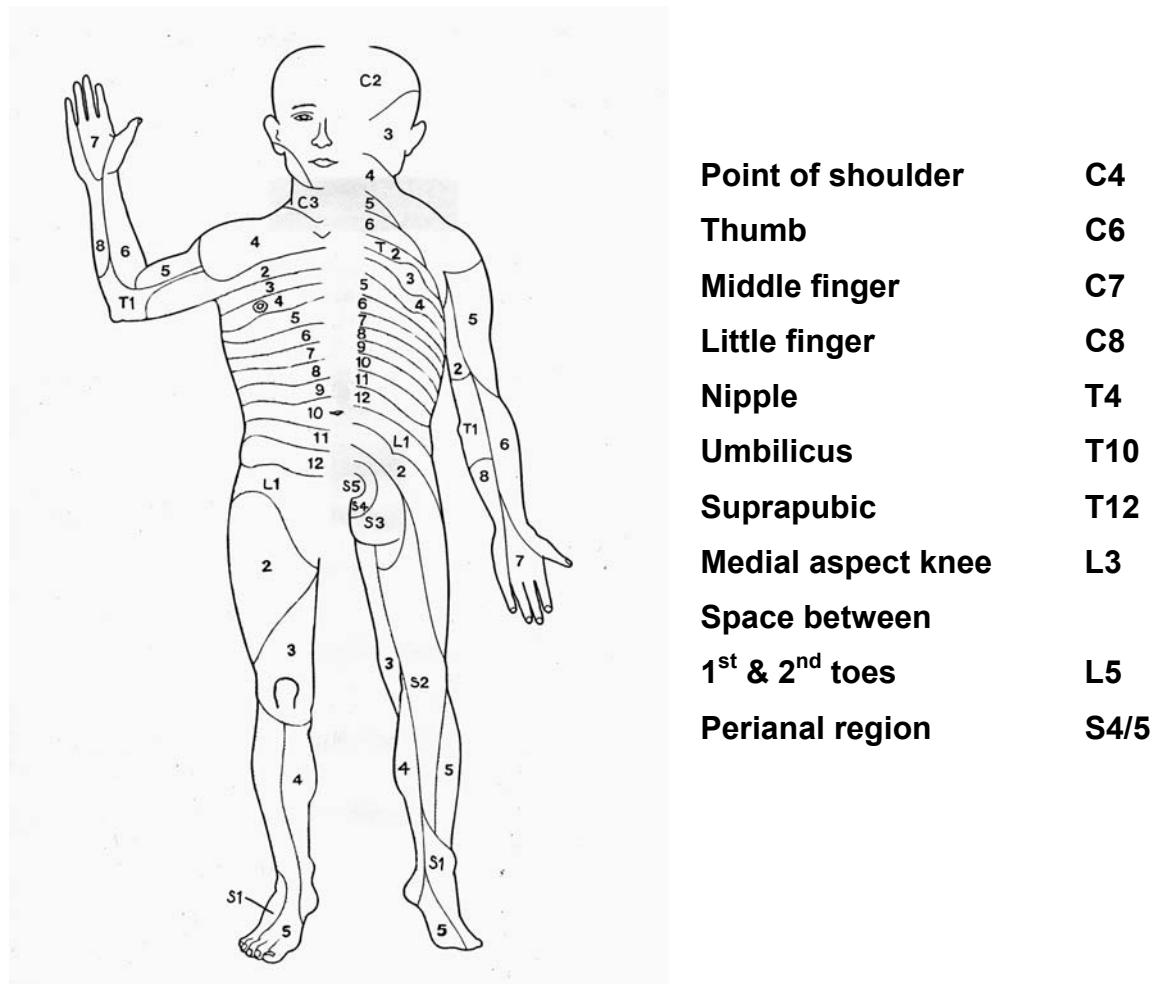
- Cranial nerves
- Sensory function in all dermatomes (light touch and pinprick)
- Muscle power (myotomes), using the MRC scale
- Reflexes

Finally, a rectal examination must be performed if not already carried out.

### Dermatomes

Sensory function is evaluated according to segmental organization in dermatomes of the human body. A dermatome is an area of the skin supplied predominantly by the sensory axons within a particular segmental nerve root (Fig. 8.1). In order to determine the level of SCI the lowest dermatome level with normal sensory function is taken as the sensory level.

**Fig. 8.1: Diagram of the dermatomes**



### *Myotomes*

Although strictly speaking most muscles are innervated by more than one nerve root, Box 8.3 shows the functions that can be regarded as being performed predominantly by muscles with one spinal root value.

### **Box 8.3: Evaluation of myotomes.**

<b>Motor function</b>	<b>Level of spinal cord segment</b>
Shoulder abduction	C5
Wrist extension	C6
Elbow extension	C7
Finger flexion	C8
Finger abduction	T1
Hip flexion	L1/L2
Knee extension	L3/L4
Ankle dorsiflexion	L4
Great toe extension	L5
Ankle plantar flexion	S1

The power of the muscle supplied by the spinal nerves in Box 8.3 is evaluated using either the Medical Research Council UK scale or the American Spinal Injury Association scale (see Box 8.4).

<b>Box 8.4: Assessment of muscle power</b>	
Medical Research Council (MRC) scale of muscle power.	American Spinal Injury Association (ASIA) scale of muscle power
<b>0</b> = total paralysis, no movement	<b>0</b> = total paralysis
<b>1</b> = a flicker of contraction, but no movement	<b>1</b> = palpable or visible contractions
<b>2</b> = movement with gravity eliminated	<b>2</b> = active movement, full range of motion, gravity eliminated
<b>3</b> = movement against gravity	<b>3</b> = active movement, full range of motion, against gravity
<b>4</b> = movement against resistance, but reduced muscle power	<b>4</b> = active movement, full range of motion, against gravity and provides some resistance
<b>5</b> = normal muscle power	<b>5</b> = active movement, full range of motion, against gravity and provides normal resistance <b>5*</b> = muscle able to exert, in examiner's judgement, sufficient resistance to be considered normal if identifiable inhibiting factors were not present

#### *Evaluation of reflexes*

The following reflexes with their approximate nerve root values should be evaluated (the order presented is simply to act as an aide memoire):

- ankle (S1,2)
- knee (L3,4)
- supinator (C5,6)
- triceps (C7,8)

#### **Grading of SCI**

A grading system for SCI has been described by the American Spinal Injury Association (see Appendix 8.2)

#### **Unconscious patients**

The key to recognizing the presence of SCI in unconscious patients is a continued high index of suspicion. The features listed in Box 8.5 increase the chance of there being a spinal injury.

If there is any spontaneous movement it is important to note it and try and identify if it was actually spontaneous or a response to pain, and any difference between limbs.

**Box 8.5: Features suggesting SCI in an unconscious patient.**

- Hypotension with a bradycardia
- Flaccid areflexia
- Diaphragmatic breathing
- Loss of response to pain below an identified dermatomal level
- Absence of reflexes below an identified level
- Priapism

As with the conscious patient, the vertebral column must be examined in its entirety. This will entail log rolling the patient with an appropriate number of staff to ensure that the vertebral column alignment is maintained and not subject to any undue forces. Finally a rectal examination is performed to assess the sphincter tone and the bulbocavernosus reflex. The latter consists of contraction of the bulbocavernosus muscle that can be detected by palpation in response to squeezing the glans penis. There will be no response if the cord is uninjured or a state of spinal shock exists. This assesses spinal roots S2-4.

**Further management of patients with SCI**

If not done so already, a long spine board must now be removed to minimize the risk of the development of pressure sores and at the same time a note must be made of the state of the pressure areas. After the primary and secondary survey, the patient's condition dictates further management (e.g. surgery, ICU).

The following interventions complete the management:

- Insert a urinary catheter if not already done
- Pass a naso/orogastric tube; ileus and aspiration pneumonitis are common complications
- Take measures to prevent hypothermia and hyperthermia
- Institute measures to prevent respiratory complications (e.g., atelectasis, muscle fatigue, increased breathing effort and ventilation-perfusion mismatch)
- Consider antiemetics and analgesics
- Repeated neurological evaluation
- Removal of immobilisation devices after clearing the spine to prevent pressure sores

## Investigation

In conscious patients without life-threatening injuries, lateral cervical spine X-rays are still a commonly used technique. In patients suffering from major trauma and/or multiple injuries and in consideration of a haemodynamically stable patient, multi-slice computed tomography (MSCT) of the whole spine is increasingly replacing lateral cervical X-rays. CT of the spine is more accurate in diagnosing vertebral column injury than plain X-rays (Fig. 8.2). Moreover, the imaging time and patient manipulation are reduced. Therefore patients with; severe brain injury, thoracic and/or abdominal trauma, are unconscious, intubated and ventilated require MSCT of the whole spine to detect or to exclude spinal or SCI

### *Magnetic resonance imaging (MRI)*

This is now the investigation of choice to identify soft tissue (non-osseous) injuries of the spinal cord, ligaments, intervertebral discs and haemorrhage.



**Fig. 8.2: Vertebral column trauma with fracture and dislocation of C5, rupture of the anterior and posterior longitudinal ligaments.**

The spinal canal is compromised by the dislocation of C4 and C5. Such an injury is associated with a high risk of spinal cord injury.

### *Plain X-rays*

A lateral cervical spine X-ray is the most common type of vertebral column X-ray. A number of errors can be made when evaluating these films that can result in injuries being missed.

These include:

- An inadequate X-ray
- Assuming that normal X-ray findings rule out spinal injury
  - a good quality lateral X-ray is only 85% sensitive
- SCI due to a vascular event with no bony injury (SCIWORA)
- Failure to appreciate the severity of the abnormality
- Failure to systematically evaluate the X-ray

The AAABCs system is helpful in interpreting a X-ray: Accuracy, Adequacy, Alignment, Bones, Cartilages and joints, Soft tissues (Appendix 8.3).

## **Who needs radiological investigation of their cervical spine?**

Many patients who have the potential for a vertebral column injury, particularly to their cervical spine, based upon the mechanism of injury, will turn out to be uninjured. A system is therefore needed to determine who needs radiological investigation of their cervical spine.

Any patient in whom the mechanism of injury suggests the potential for injury, and does not fulfill **ALL** of the following seven criteria needs investigating:

1. Alert and orientated
2. Not under the influence of drugs or alcohol
3. Neurologically normal
4. No other distracting injuries
5. Age less than 65 years

The cervical collar is now removed and replaced with MILS while the cervical spine is palpated:

6. No tenderness in the midline over the cervical spine

Finally, the patient is asked to actively move their head and neck:

7. Painfree, unrestricted rotation of the neck, 45° to the left and right

If any criteria are positive, full immobilisation is maintained and appropriate radiological investigation according to local protocol is obtained. If all these criteria are fulfilled, cervical spine immobilization is no longer required and it is safe to remove the devices immobilising the cervical spine. This is commonly referred to as “clearing the cervical spine”.

### **Summary**

The management of the patient with a spinal injury starts at the scene and continues through to rehabilitation in order to minimise the risk of secondary injury and maximise the potential for outcome. The principles of resuscitation as described throughout this book apply at all stages and great care must also be taken to ensure that the patient's condition is not worsened as a result of careless or uncoordinated handling at any point of their care. The management of vertebral column trauma and SCI requires an interdisciplinary team approach and is crucial for the long-term quality of the life of these patients.

## **Appendix 8.1: Anatomy of the vertebral column and spinal cord**

### *The vertebral column*

The human spine is made up of 33 vertebrae arranged in a column, 7 cervical, 12 thoracic, and 5 lumbar, and up to 10 fused vertebrae forming the sacrum and coccyx (Fig. 8.3). Typically, a vertebra consists of two main parts: anteriorly the body, which is a cylinder of bone, and posteriorly a vertebral arch that encloses the spinal cord. From the body of the vertebra, a pair of pedicles project backwards and from these the laminae proceed and fuse together in the midline, thereby forming the spinal canal. From each arch, a spinous process projects posteriorly and from each side a transverse process projects laterally. The superior and the inferior articular processes of the facet joints are formed from the transverse processes. Each pedicle contains a notch both superiorly and inferiorly that align with the corresponding notches on adjacent vertebrae to form the intervertebral foramina, through which pass the spinal nerves. Intervertebral discs separate the vertebral bodies. The vertebral bodies and intervertebral discs are joined by anterior and a posterior longitudinal ligaments. These ligaments, together with the facet joints, the interspinous ligaments, and the paraspinal muscles provide vertebral column stability. A series of curves, produced partly by the wedge shape of the vertebral bodies and the shapes of the intervertebral discs give rise to the characteristic cervical and lumbar lordoses. The bodies of the vertebra and the intervertebral discs provide axial support while the flexibility of the spine is facilitated partly by the natural curves, the intervertebral discs and the presence of the synovial facet joints. The greatest flexibility of the vertebral column is found in the cervical and thoracolumbar parts of the spine therefore, the risk of injury to the spinal cord is highest here.

### *Cervical vertebra*

There are seven cervical vertebra, of which the top two are structurally distinct. The 1<sup>st</sup> cervical vertebra (atlas) has no body but instead, lateral masses that articulate by synovial joints with the occipital condyles on the base of the skull (the atlanto-occipital joints). These joints allow for a considerable degree of both flexion and extension of the head. The 2<sup>nd</sup> cervical vertebra (axis) has an upward projection like a finger called odontoid process (or dens) and articulates with the back of the anterior arch of the atlas - in other words it lies within the spinal canal itself (Fig. 8.4). The joint between the atlas and the axis (the atlanto-axial joint) allows mainly for rotation of the head in a line passing through the long axis of the odontoid peg.

**Fig. 8.3: Vertebral column**

# Vertebral column and Spinal Cord

**Human vertebral column:**

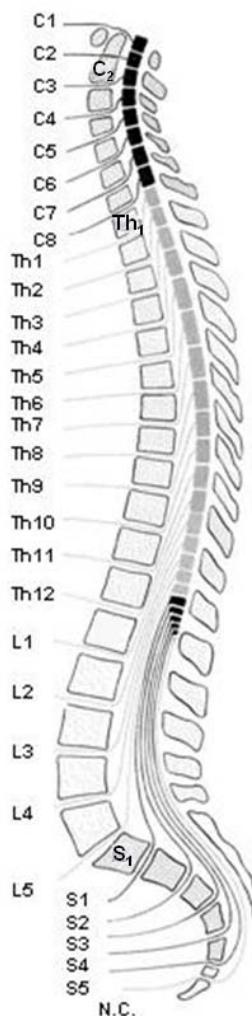
34 vertebrae

- 7 cervical
- 12 thoracic
- 5 lumbar
- 10 fused as the sacrum  
and coccyx

**Spinal cord:**

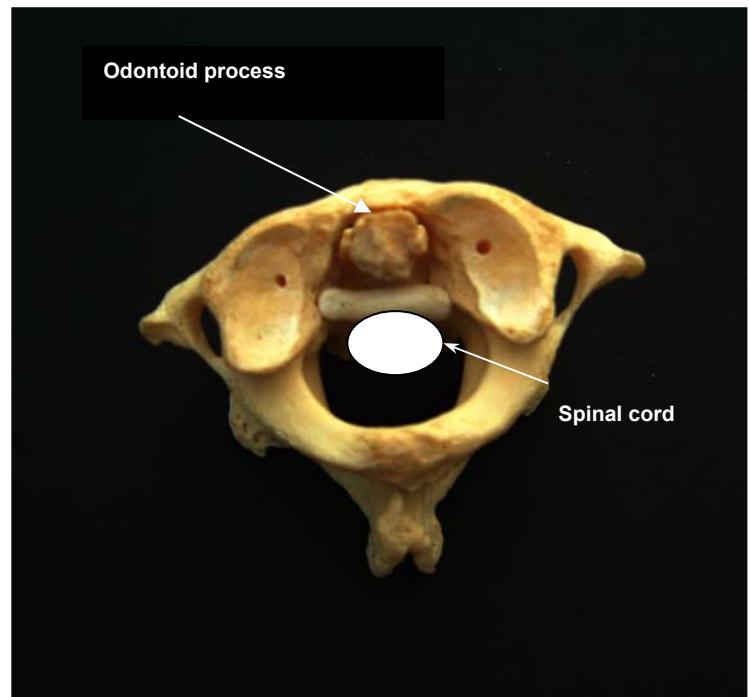
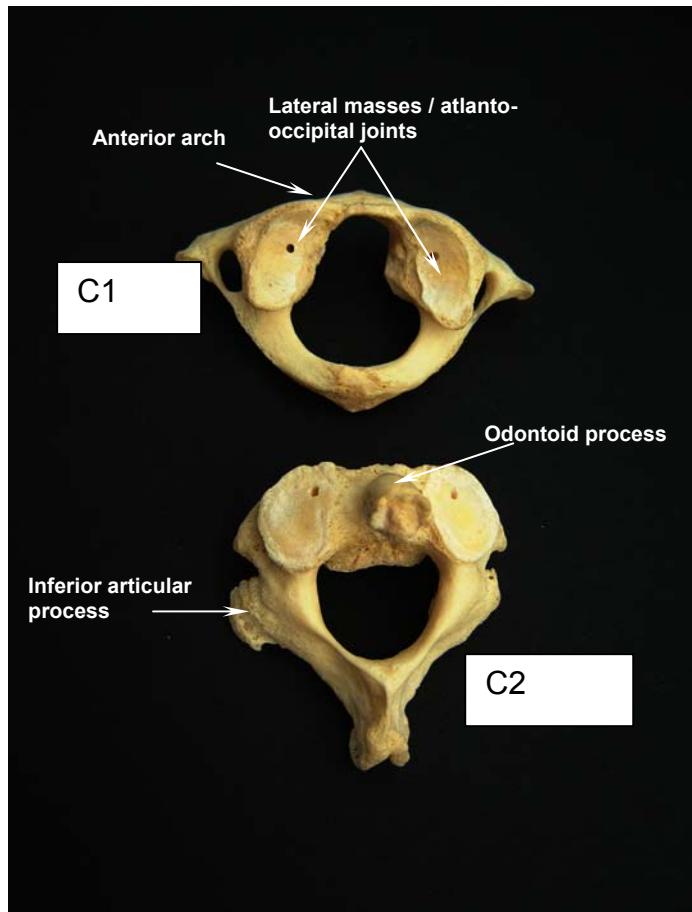
31 segments

- 8 cervical
- 12 thoracic
- 5 lumbar
- 5 saccral
- 1 coccygeal



(Modified from Bernhard et al. Anaesthetist 2005; 54:357-376. Copyright Springer Heidelberg, Germany).

**Fig. 8.4: First 2 cervical vertebrae**



Steele's rule of three: "One third of the spinal canal within C1 is occupied by the odontoid, one third by an intervening space, and one third by the spinal cord".

The lower five cervical vertebra all have backward sloping facet joints stacked somewhat akin to a series of roof tiles. These are synovial joints and allow mainly for flexion and extension and to a lesser degree rotation.

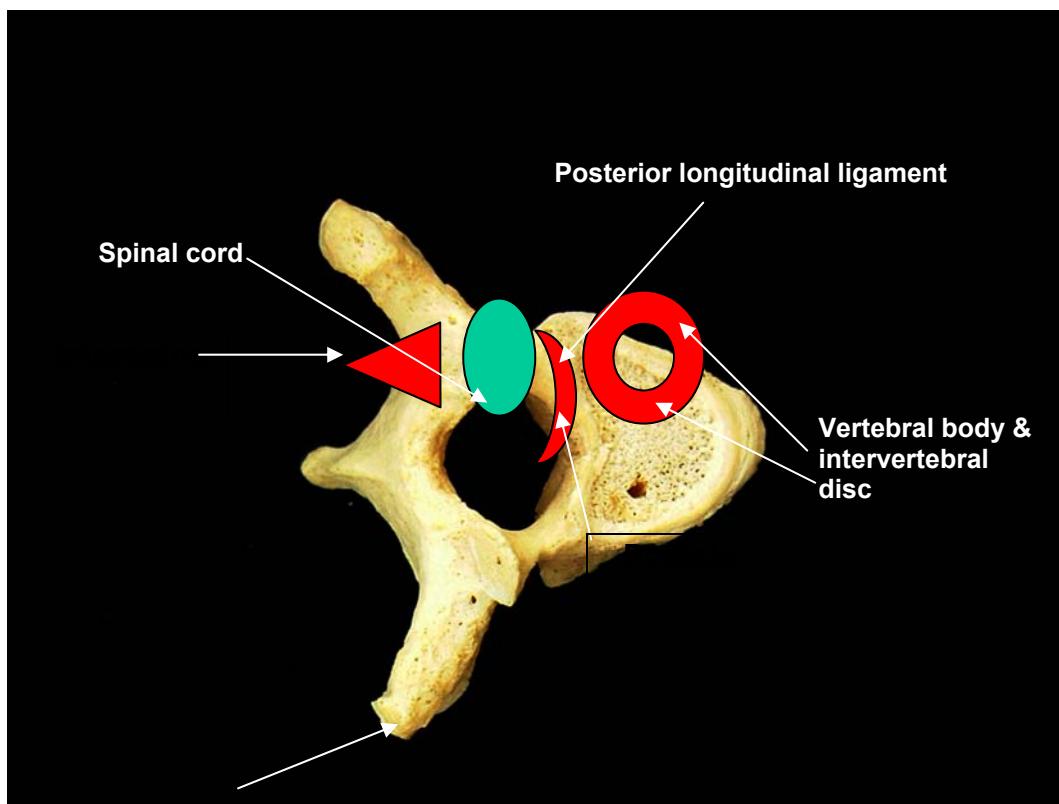
#### *Thoracic vertebra*

There are 12 vertebra, characterised by relatively long transverse process and facets joints on the sides of the vertebral bodies allowing for articulation with the ribs. The facet joints through which adjacent vertebra articulate with each other are aligned more vertically than within the cervical spine and whilst allowing flexion and extension, significantly restrict rotation.

### *Lumbar vertebra*

The five lumbar vertebrae are characterised by having the largest bodies, shorter transverse processes and retaining the vertical lie of the facet joints.

**Fig. 8.5: Boundaries of the spinal canal**



The sacrum consists of five fused vertebrae and is triangular in shape. The vertebral canal is bounded by pedicles and laminae and there are very short spinous processes. Laterally there is an articular facet for articulation with the ilium. The coccyx consists of three to five fused vertebrae articulating with the sacrum.

### *Intervertebral joints*

Movement between adjacent vertebrae is relatively slight but there is a considerable additive effect. The two regions of greatest flexibility are the junctions between the cervical and the thoracic spine and between the thoracic and lumbar spine. Combined with the fact that these are the points where the direction of curvature of the spine changes, makes them also the two commonest sites for spinal injury.

### **Spinal canal**

The spinal canal extends from the foramen magnum in the base of the skull to the sacral hiatus. It is bounded anteriorly by; the vertebral bodies, intervertebral discs and the posterior longitudinal ligament. Posterior are the laminae and the interspinous ligaments and laterally the pedicles of the vertebra and the vertebral foramina (Fig. 8.5).

## THE SPINAL CORD

The spinal cord is approximately 45 cm long. It is enclosed by the vertebral column and, in adults, terminates at the lower level of the 1<sup>st</sup> lumbar (L1) or upper level of the 2<sup>nd</sup> lumbar (L2) vertebra. The spinal cord is divided into 8 cervical, 12 thoracic, 5 lumbar, and 5 sacral segments, and 1 coccygeal segment (Fig. 8.3). The cord is surrounded by the dura mater and between this and the bony canal is the extradural space, normally filled with fat and blood vessels. The size of the space varies with the different levels of the cord, being minimal in the thoracic region and maximal at C2 where there is a large space behind the odontoid process. This affords the spinal cord at this level a degree of protection and is often referred to as "Steele's rule of three" (see below).

### *Structure of the spinal cord*

The spinal cord consists of nerve fibres that transmit impulses from the periphery to the brain (ascending tracts) and from the brain to the periphery (descending tracts). In the transverse section, the cord has a central canal surrounded by the H-shaped gray matter (Fig. 8.6). The posterior horns of the gray matter contain the nerve cells of the sensory fibres entering via the posterior nerve roots and the anterior horns contain the nerve cells of the motor nerves that give rise to the anterior nerve roots. The remainder of the cord consists of the white matter that contains the nerve fibers of the long ascending and descending tracts. The nerve fibers in some of the tracts cross (or decussate), representing an important factor in interpreting signs after trauma to the spinal cord.

## **Ascending tracts**

- 1) The anterior and lateral spinothalamic tracts convey pain and temperature. Having entered the spinal cord, they ascend and then cross to the opposite side within two segments before ascending to the thalamus and then to the sensory cortex.
- 2) The posterior columns consist of the medial and lateral tracts or fasciculus gracilis and fasciculus cuneatus respectively. They convey fine touch, vibration, and proprioception (position sense). The nerve fibres ascend uncrossed until they reach the level of the medulla.

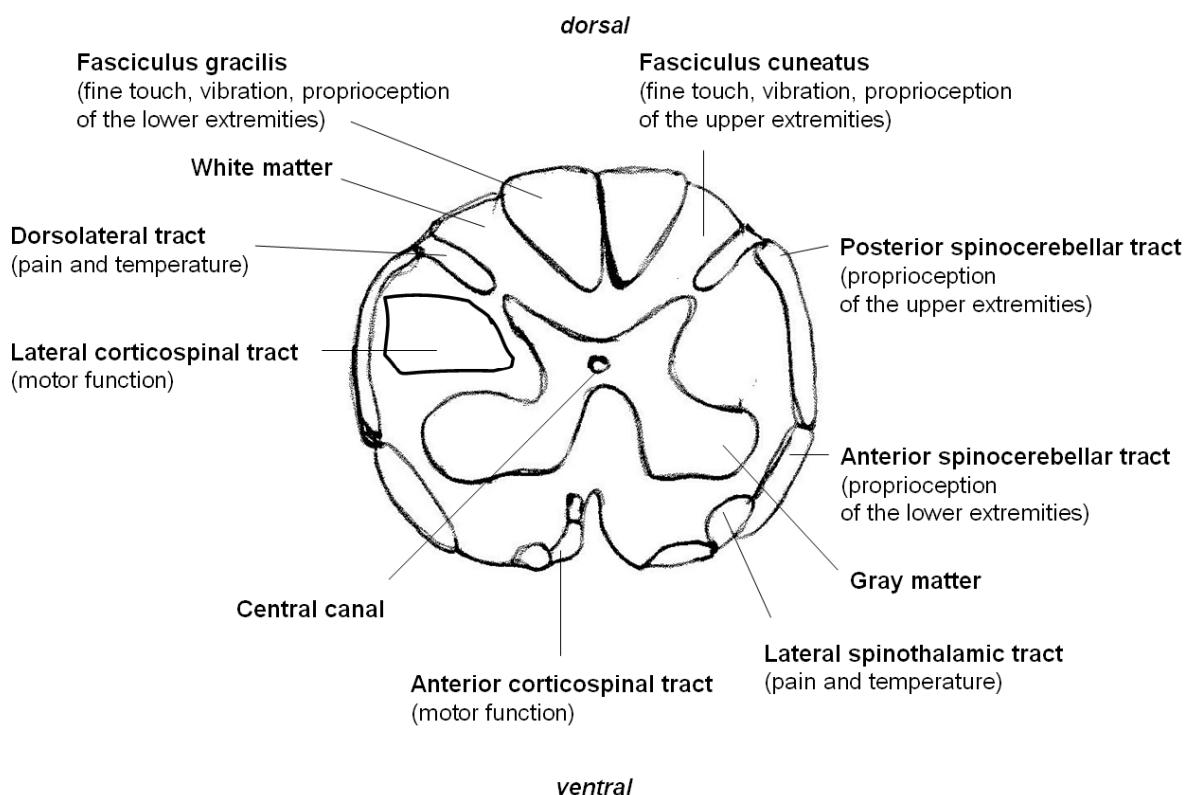
- 3) The anterior and posterior spinocerebellar tracts also convey proprioception. After they have entered the spinal cord, they ascend on the same side of the cord as they enter the cerebellum.

## **Descending tracts**

- 1) The lateral corticospinal or pyramidal tract has its origin in the motor cortex and the fibres cross to the opposite side in the medulla, that is before they reach the spinal cord. They then descend in the contralateral side of the cord.
- 2) The anterior corticospinal tract is smaller and descends uncrossed from the cortex and the fibres cross at the level at which they leave the spinal cord.

**Fig. 8.6: Cross section of the spinal cord**

## **Spinal cord**



The fibres of the sympathetic (thoracolumbar, T1-L2) and the sacral parasympathetic (S2-4) system are also located in the spinal cord. Injuries of the spine and the corresponding part of the spinal cord can cause different neurological symptoms that are related to the locations of

nerve fibres in that part of the spinal cord. Thus, neurological deficits can be related to the motor, sensory, sympathetic, and/or parasympathetic system.

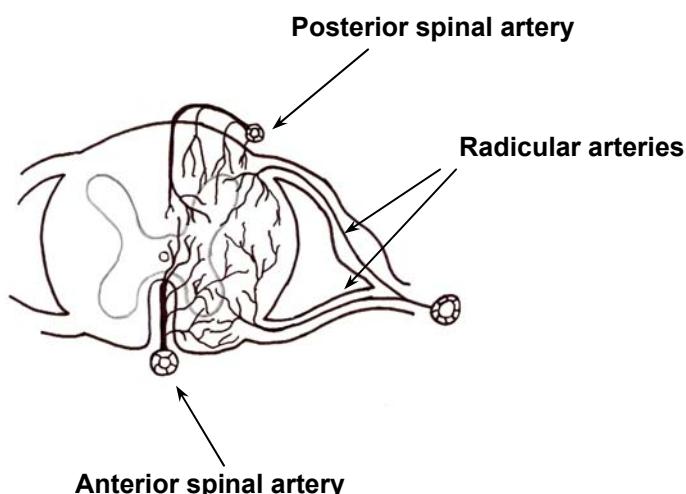
#### Spinal nerves

Each segment of the spinal cord releases a pair of anterior (motor function) and posterior (sensory) spinal nerve roots on each side. These nerve roots pass from the spinal cord to the appropriate intervertebral foramina (neuroforamina) where they unite to form a mixed sensory and motor spinal nerve. Below the level of L1, the spinal canal contains the anterior and posterior nerve roots from the lumbar, sacral, and coccygeal segments and pass almost vertically downwards to form the cauda equina.

#### *Blood supply of the spinal cord*

The main arterial supply is provided by one anterior spinal and two posterior spinal arteries that descend from the foramen magnum. The anterior spinal artery supplies the anterior two thirds of the spinal cord (anterior to the posterior columns) and the two posterior spinal arteries supply the posterior third of the spinal cord (posterior columns). There is a smaller additional supply from the radicular arteries that originate mainly from the descending aorta provide additional blood supply (Fig. 8.7). The largest artery usually arises in the lower thoracic or upper lumbar region and branches into the anterior and the two posterior spinal arteries, supplying blood to a significant part of the lower spinal cord. As there are no anastomoses between the anterior and the posterior circulation, the cord is susceptible to any reduction in blood supply and such a reduction may result in infarction of the spinal cord, particularly in the watershed areas between vessels. The spinal cord blood flow amounts to 40-60ml/100g/min and is autoregulated between 60 and 150 mmHg like cerebral blood flow.

**Fig. 8.7: Blood supply of the spinal cord**



## **Appendix 8.2: Mechanisms of injury**

### *LOCATION OF SPINE AND SCI*

Traditionally, it has been taught that the majority of spine fractures occur in the cervical spine with the remainder being fairly evenly split between the thoracic and the lumbar region. Data from the Trauma Audit Research Network, (TARN, personal communication) reveal a different distribution; 22% cervical, 28% thoracic, 41% lumbar and 8% at multiple levels. This probably reflects the varying stability of the spine. An evaluation of data from patients with SCI indicates a much higher incidence of cord injury associated with cervical vertebral column fractures compared with either thoracic or lumbar injuries. In addition there are a small number of patients who present with "Spinal Cord Injury WithOut Radiographic Abnormalities" (SCIWORA), usually young children or the elderly. In the young it occurs because there is potential for much greater mobility of the vertebral column without causing a fracture. In contrast in the elderly there are often pre-existing degenerative changes leading to significant narrowing of the spinal canal.

It is very important to know that pain from spinal and SCI is not necessarily localized in the area of injury. Studies have found that in 18% of cervical, 63% of thoracic, and 9% of lumbar injuries, the pain is located elsewhere. Thus, if patients have pain at a site that could be related to SCI, special care must be taken because the location of injury can be in another segment of the spine. Furthermore, in when a vertebral column fracture is identified at one level, a second injury at another, non-adjacent segment occurs in up to 15% of cases.

### *Spinal injury*

The spine can suffer primary injury as a result of penetrating trauma or indirectly as a result of excessive movement; flexion, extension, rotation, axial compression or distraction alone or in any combination. The spinal cord may also be injured as a result of bone or intervertebral disc fragments becoming displaced into the spinal canal. Most of the injuries to the spinal cord are the result of contusions and compression rather than complete transaction. These insults lead to local ischaemia that initiates a sequence of events at a molecular level that result in an inflammatory response and tissue oedema, extending the site of injury. Following the primary injury, secondary injury can also occur either as a result of inadequate care usually due to excessive, uncontrolled movement or as a result of hypoxia and/or hypotension that exacerbates the inflammatory response causing even more widespread injury. Therefore, correct management of these patients is essential to prevent any additional loss of function.

A spinal injury is considered stable when controlled movement will not cause any additional neurological injury and unstable when any movement can cause or exacerbate a neurological injury. A spinal injury should only be defined as unstable or stable after a CT and/or MR examination has been conducted and assessed by an expert.

#### *Hyperflexion injury*

Occurs commonly in adults between T12-L2 as a result of forced flexion (e.g., over a seatbelt following frontal impact, building collapse). In children, these injuries tend to be localized higher, at T4-T6. This type of injury often results in wedge (compression) fracture of the spine. It may be stable or unstable depending on the severity of compression. It also occurs in the cervical spine, usually at C5,6 as a result of diving accidents. The hyperflexion injury may result in a fracture of the anterior superior corner of the vertebra a "tear drop" fracture. There is often significant associated ligamentous injury and these fractures should be considered unstable.

#### *Hyperextension injury*

These are usually only found in the cervical and lumbar regions of the spine due to the stabilizing effect of the ribs. Fragments of the vertebral body can be pushed into the spinal canal as a result of such injury. If the mechanism of injury was associated with rotation, the laminae and pedicles may be fractured. The hangman's fracture is considered a special type of hyperextension injury, which combined with distraction of the cervical spine, results in a fracture through C2.

#### *Compression injuries*

Commonly compression injury affects the cervical or lumbar region of the spine, most frequently at L1. In severe cases, fragments can be pushed into the spinal canal. Such injury is also seen after diving accidents, usually at C5. A particular kind of injury occurs when axial loading compresses C1 between the occipital condyles and C2, also known as a Jefferson fracture. In addition, C1 often slides forward onto C2, but the cord remains uninjured thanks to Steele's rule of three: "One third of the spinal canal within C1 is occupied by the odontoid, one third by an intervening space, and one third by the spinal cord".

#### **Spinal cord injury (SCI)**

Injuries to the spinal cord are divided into primary and secondary injuries (see above). Spinal cord injuries can also be classified as either complete or incomplete according to the American Spine Injury Association (Box 8.6). Complete SCI should be suspected when there has not been any recovery of sensory or motor function within 48 hours. The outlook is poor as there is likely to be no further improvement. Because of this delay in recovery, an accurate prognosis for the patients cannot be given in the first few hours and statements as such should not be issued in the emergency department (ED). Incomplete SCI has a much better prognosis for some functional motor recovery. The presence or absence of sacral nerve root function may be a more stable and reliable indicator of the completeness of an injury as it represents at least partial structural integrity of the corticospinal and spinothalamic tracts. Sacral sparing can be confirmed by perianal sensation, rectal sphincter tone and flexion of the hallux.

Some special syndromes associated with incomplete SCI are described below.

**Box 8.6: Grading of SCI according to the American Spinal Injury Association (ASIA).**

ASIA-Degree	Incidence	Neurological function
A	25-50%	Complete: no motor or sensory function is preserved in the sacral segments S4-5
B	15%	Incomplete: sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-5.
C	10%	Incomplete: motor function is preserved below the neurological level and more than half the key muscles below the neurological level have a muscle power grade of less than 3.
D	30%	Incomplete: motor function is preserved below the neurological level and more than half the key muscles below the neurological level have a muscle power grade of 3 or more.
E		Motor and sensory function are normal

*Anterior cord syndrome*

- Due to the loss of function of the anterior two-thirds of the spinal cord
- Usually the result of a flexion injury or an axial loading leading to a burst fracture and damage to the anterior spinal artery
- May also be seen after a period of profound hypotension

- On examination there is loss of motor function (flaccid paralysis), sharp pain and temperature sensation below the lesion
- Proprioception, vibration and deep pressure sensation are all retained in the trunk and lower extremity because they are transmitted in the intact posterior columns in the cord.
- Carries a poor prognosis, only a 10% chance of functional motor recovery.

*Central cord syndrome*

- Most common pattern of incomplete SCI pattern.
- Often follows hyperextension to the neck, such as from a fall on to the face. Typically seen in older patients who have degenerative changes in their spine and narrowing the spinal canal
- Usually results in a vascular event, compromising blood flow to the centre of the cord
- Results in damage to the corticospinal and spinothalamic tracts, with preservation of the sacral spinothalamic and peripheral corticospinal tracts
- On examination there is a flaccid paralysis of the arms, worse distally and spastic paralysis of the legs, intact perianal sensation and an early return of bowel and bladder function. There may be disturbance of sensation with hyperesthesia, in the arms more than the legs.
- Return of motor function usually begins with the sacral elements (toe flexors, then extensors), followed by the lumbar elements of the ankle, knee and hip. Upper limb functional return is usually minimal.
- The chance of some functional motor recovery has been reported to be about 75%.

*Brown-Séquard syndrome*

- A rare injury resulting from a hemi-transection of the spinal cord and associated unilateral spinal tracts
- The neurological findings are of loss of power and proprioception, vibration and deep pressure sensation on the side of the injury at the level of the lesion
- On the opposite side of the body there is a loss of pain and temperature sensation below the level of the lesion
- The mechanism of injury is most commonly the result of a penetrating wound from either a gunshot or stabbing
- Almost all of these patients show a partial recovery, and most regain bowel and bladder function and the ability to ambulate.

*Posterior Cord Syndrome*

- Very rare syndrome.
- Loss of deep pressure, deep pain and proprioception.
- Otherwise normal cord function.
- The patient ambulates with a foot-slapping gait similar to that of someone suffering with tabes dorsalis.

*Root Injury*

- Spinal nerve root may be injured with the cord at that level or in isolation.
- Prognosis is favourable for motor recovery, with about 75% of those with complete SCI showing no root deficit at the level of injury or having a functional return.
- Those with higher cervical injuries have a 30% chance of recovery of one nerve root level, those with midcervical injuries have a 60% chance, and almost all patients with low cervical fractures have recovery of at least one nerve root level.

### Appendix 8.3: AAABCs system for interpreting of lateral cervical X-ray.

Key point	Interpretation
Accuracy	Is this the correct film for the correct patient?
Adequacy	<p>Are all 7 cervical vertebrae, the occipitio-cervical junction and the C7-T1 junction visible?</p> <p>If not: Repeat the film with the patient's arm pulled down to remove the shoulder from the field of view or take a "swimmer's view". If these fail, then MSCT</p>
Alignment	<p>Check the contours of the four longitudinal curves (Fig. 8.8).</p> <p>Anterior – along the anterior aspect of the vertebral bodies from the skull base to Th<sub>1</sub></p> <p>Posterior – along the posterior aspect of the vertebral bodies from the skull base to Th<sub>1</sub></p> <p>The spinolaminar line should be smooth except at C<sub>2</sub> where there can be slight posterior displacement (2 mm)</p> <p>The tips of the spinous processes – a tighter curve. The tips should also converge to a point behind the neck</p> <p>A break in any of these lines indicates a fractured vertebra or facet dislocation until proved otherwise. Divergence of the spinous processes is also abnormal. In some patients there is a pronounced loss of the normal curve of the cervical spine (lordosis). This may be due to muscle spasm, age, previous injury, radiographic positioning or the presence of a hard collar. If identified it therefore only indicates that the patient may have sustained a cervical spinal injury.</p>
Bones	<p>Check the cortical surfaces of all vertebrae for steps, breaks, or angulation.</p> <p>C<sub>1</sub> check the laminae and pedicles, think about a Jefferson fracture.</p> <p>C<sub>2</sub> check the outline of the odontoid and pars interarticularis, think about a hangman's fracture.</p> <p>C<sub>3</sub>-Th<sub>1</sub> start at the anterior inferior corner of the vertebral body and proceed clockwise, checking pedicles, laminae and spinous processes. The height of the anterior and posterior bodies should be the same. More than 2 mm difference suggests a compression fracture.</p> <p>Check the spinal canal – this extends from the back of the vertebral body to the spinolaminar line and is more than 13 mm wide. It may be narrowed by; dislocations, bony fragments pushed posteriorly, or pre-</p>

	existing degenerative disease.
Cartilages and joints	<p><i>Check the disc spaces, facet joints and interspinous gaps.</i> Disc spaces should be of uniform height and similar in size to those between adjacent vertebrae. Facet joints have parallel articular surfaces, with a gap less than 2 mm. Widening of the gap and visibility of both facets suggests unifacetal dislocation. There will also be anterior displacement of less than half the width of the vertebral body and associated soft tissue swelling (see below). If there is displacement greater than 50%, both facets are dislocated. There will also be narrowing of the disc space, widening (fanning) of the spinous processes and soft tissue swelling.</p> <p><i>Check the gap between C<sub>1</sub> and the front of the odontoid peg.</i> The distance between the posterior surface of the anterior arch of C<sub>1</sub> and the anterior surface of the odontoid should be less than 3 mm in adults, greater than this suggests rupture of the transverse ligament. This may occur without there being bony injury or cord damage (Steele's rule of three: "One third of the spinal canal within C<sub>1</sub> is occupied by the odontoid, one third by an intervening space and one third by the spinal cord").</p>
Soft tissues	<p><i>Check the soft tissue shadow anterior to the cervical vertebrae</i> Fractures of the cervical vertebrae or ligamentous injury will result in a hematoma as in any other area of the body. This will be seen as an increase in the width of the soft tissue shadow adjacent to the injury. In some subtle injuries this may be the only evidence. As a "rule of thumb" the soft tissue shadow between the anterior border of C<sub>1-3</sub> and the air in the oro- and nasopharynx should be less than 7 mm wide. At the level of C<sub>5</sub> this increases to about 21 mm, or the width of the vertebral body. Occasionally, this may be seen as anterior displacement of an endotracheal tube.</p> <p>It must be remembered that the stability of the cervical spine is dependent on the ligaments that are not revealed on a plain X-ray. Therefore, the lateral cervical film must be examined not only for signs of bony injury but also clues of ligamentous injury as this may indicate the presence of an unstable injury (facet joint widening, facet joint overriding, widening of the spinous processes, &gt;25% compression of a vertebral body, &gt;10° angulation between vertebral bodies, &gt;3.5 mm vertebral body</p>

overriding with fracture, Jefferson's fracture, Hangman's fracture, Tear drop fracture).

In most patients who are suspected of having a significant injury to their cervical spine, further X-rays will be required, for example anteroposterior, open mouth views and in addition, thoracic and lumbar views may also be required. For this the patient must be transferred to the X-ray department. This should only be undertaken when it is safe to do so. For further details on interpretation of X-rays the interested reader should consult the references.



**Fig. 8.8: Lateral cervical spine film showing the four longitudinal curves**

## **Further reading**

1. Ackery A, Tator C, Krassioukov A (2004) A global perspective on spinal cord injury epidemiology. *J Neurotrauma* 21:1355-1370.
2. Bernhard M, Gries A, Kremer P, Böttiger BW (2005) Spinal cord injury (SCI) – Prehospital management. *Resuscitation* 66:127-139.
3. CRASH Trial Collaborators (2004) Effects of intravenous corticosteroids on death within 14 days in 10008 adults with clinically significant head injury (MRC CRASH trial): randomised placebo controlled trial. *Lancet* 364:1321-1328.
4. Hoffman JR, Wolfson AB, Todd K, Mower WR. (1998) Selective cervical spine radiography in blunt trauma: methodology of the National Emergency X-Radiography Utilization Study (NEXUS). *Ann Emerg Med* 32:461.
5. Stiell IG, Wells GA, Vandemheen KL, et al. (2001) The Canadian C-spine rule for radiography in alert and stable trauma patients. *JAMA* 286:1841-1848.
6. Raw DA, Beattie JK, Hunter JM (2003) Anaesthesia for spinal surgery in adults. *Br J Anaesth* 91:886-904.
7. Sekhon LHS, Fehlings MG (2001) Epidemiology, demographics, and pathophysiology of the acute spinal cord injury. *Spine* 26(suppl):S2-12.
8. Stevens RD, Bhardway A, Kirsch JR, Mieski MA (2003) Critical care and perioperative management in traumatic spinal cord injury. *J Neurosurg Anesthesiol* 15:215-229.

## **Websites**

[www.trauma.org/spine](http://www.trauma.org/spine)

Good website for detailed information.

[www.spinalcord.uab.edu](http://www.spinalcord.uab.edu)

Website of the “Spinal Cord Injury Network” with numerous links to other sites.

## Chapter 9

# EXTREMITY AND SOFT TISSUE TRAUMA

## Objectives

At the end of this chapter the reader should understand:

- The significance of identifying musculoskeletal injuries
- The principles of how to assess and manage soft tissue injuries in the emergency department
- The principles of how to assess and manage common fractures in the emergency department

Details of the pathophysiology of soft tissue and bony injuries are given in Appendix 9.1.

## Introduction

The early management of the trauma victim involves a primary survey to identify and correct immediately life threatening problems. This is followed at an appropriate time by a full secondary survey. Extremity trauma may be both life-threatening and limb-threatening and may be identified during either the primary or secondary surveys respectively. Proper initial management is essential not only to prevent early mortality but also the risk of increased morbidity at a later stage. Examples of when musculoskeletal injuries may be identified during the primary and secondary survey are given in Box 9.1.

### Box 9.1: Detection of musculoskeletal injuries in the primary and secondary surveys

Primary survey	Airway Breathing Circulation  Disability Exposure	<ul style="list-style-type: none"><li>• Complaints of pain</li><li>• Hypovolaemia associated with closed pelvic fractures</li><li>• Blood loss associated with open fractures</li><li>• Penetrating injuries involving major limb vessels</li><li>• Multiple limb fractures</li><li>• Large soft tissue injuries</li><li>• Traumatic amputation</li><li>• Reduced limb movement</li><li>• Fractures and dislocations (deformity, angulation, wounds)</li></ul>
Secondary survey		<ul style="list-style-type: none"><li>• Lesser fractures and dislocations</li><li>• Lacerations, contusions and abrasions</li></ul>

Limb-threatening trauma must be recognised and dealt with in the secondary survey and typically may consist of:

- Vascular limb injuries
- Acute compartment syndrome
- Major soft tissue injuries
- Compound fractures (delayed infection)
- Neurological injuries

The majority of lacerations and fractures are less serious but are important because:

- They are very common
- Relatively minor injuries of this nature compose a significant part of the workload of Emergency Departments

Musculoskeletal injuries are a frequent cause of prolonged and sometimes significant morbidity. The scenario of a patient dissatisfied with the long term results of a relatively minor musculoskeletal injury despite exemplary management of major injuries is fairly common and may result in litigation. Many of these frequently missed injuries will be detected by carrying out a "tertiary survey" (see below).

Soft tissue injuries and extremity injuries may lead to important functional and cosmetic problems. The cosmetic problems may be depressingly obvious, and lead to significant psychiatric problems. The functional consequences include persistent pain, numbness, joint stiffness, weakness and deformity; such problems may compromise, for example hand functions or walking, and therefore produce difficulties with mobility, work and recreation.

### **Clinical assessment of limb soft tissue and bony injuries**

THE MAJORITY OF SOFT TISSUE WOUNDS WILL BE ASSESSED AND MANAGED DURING THE SECONDARY SURVEY, THE EXCEPTION BEING WOUNDS THAT ARE BLEEDING HEAVILY AND COMPOUND FRACTURES WHICH REQUIRE COVERING WITH A STERILE DRESSING TO REDUCE THE RISK OF FURTHER CONTAMINATION AND INFECTION.

### **History**

Details of the mechanism of injury must be obtained, including the time since injury. For all patients vital information can be obtained from the pre-hospital personnel who may also provide the emergency department with a photograph or digital image of the scene. Vehicle

deformation also predicts injury patterns – the so called “reading the wreckage”. Examples where the history is important include:

- Limbs run over by the wheel of a vehicle: risk of degloving injuries; the skin is sheared off the underlying tissues even though superficially it may be relatively intact
- The posture of the patient at the time of injury: this may give clues as to where to look for injuries, particularly after penetrating trauma.
- The cause of the injury: lacerations with associated contamination, for example wounds sustained with a garden digger, paint gun injection injury.

Symptoms suggestive of serious deep injury include:

- Severe pain
- Loss of function (for example inability to weight bear)
- Numbness

Additional information which may influence the choice of treatment, the early involvement of specialists for advice because of the risk of complications, infection or wound failure include:

- Steroid usage
- Diabetes mellitus
- Smoking
- Peripheral vascular disease
- Malnutrition
- Immunocompromised patients

To facilitate ongoing medical and nursing care, Box 9.2 contains important factors to be considered in patients following injury.

#### **Box 9.2: Factors to be considered in patients following injury**

<ul style="list-style-type: none"><li>• History of present condition.</li><li>• Mechanical or medical condition precipitating injury</li><li>• Last intake of fluid and diet.</li><li>• General health status</li><li>• Medications, current and past, (including use of drugs socially)</li><li>• Allergies</li><li>• Smoking</li><li>• Antitetanus status</li><li>• Family history</li></ul>	<p>In the elderly also consider:</p> <ul style="list-style-type: none"><li>• Patient profile and nutritional status</li><li>• Tissue viability score and prevention</li><li>• Mental and emotional state</li><li>• Osteoporosis and loss of function</li><li>• Medical causes for falls</li><li>• Social history in preparation for discharge planning</li><li>• MRSA status in Elderly Peoples Homes/Nursing Homes</li><li>• Spiritual considerations</li></ul>
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## **Clinical examination**

It is easy to miss both soft tissue and bony injuries. This risk can be minimised by ensuring appropriate exposure to facilitate a full clinical examination in a systematic fashion. Patients with significant injuries should routinely undergo a “tertiary survey”, the aim being to detect injuries not apparent during the initial primary and secondary surveys. It should be completed within 24-hours of first admission. This is particularly important in those patients on the Intensive Care Unit who may be sedated, ventilated and unable to indicate the presence of minor injuries. In other patients, it may take place when stable or after surgery.

**All patients with significant injuries must have a tertiary survey**

From a musculoskeletal perspective, the examination should follow a structured format.

- **Look** - compare with the other side looking for swelling, deformity and wounds
- **Feel** - for tenderness, swelling, skin temperature, peripheral pulses, crepitus and sensation and compare with the uninjured side
- **Move** – both actively and passively and compare sides

Many patients with limb injuries will require x-rays. These and other investigations are NEVER a substitute for adequate clinical examination. Care should be taken however to reduce to a minimum any pain caused by clinical examination.

Soft tissue injuries can be limb threatening (Box 9.3). They may occur in isolation. It is important not to miss these injuries in patients with multiple injuries involving the torso or head (Fig. 9.1).

### **Box 9.3: Limb threatening injuries**

- Vascular injury at or proximal to the elbow or knee
- Major joint dislocation, especially the knee
- Crush injury
- Compartment syndrome
- Open fracture
- Fracture with neurovascular injury



**Fig. 9.1: Limb threatening injury with tissue loss, crushed and non-viable muscle**

An important feature of limb threatening injuries is the risk of vascular compromise. Muscle is particularly vulnerable to relatively short periods of ischemia. Vascular damage is possible even if pulses are normal, for example with an intimal tear, or acute compartment syndrome (see below).

In assessing soft tissue injuries, it is essential to have an adequate understanding of the anatomy of the injured area, for example a deep laceration on the dorsum of the forearm has different implications to one on the palmar surface of the wrist where the latter may typically affect arteries, nerves and tendons. In all cases of penetrating trauma, clinical assessment must be made of all structures underlying the area of the wound including muscles, tendons, arteries and veins, nerves and bones. When assessing the damage due to penetrating trauma, consider the patient's posture at the time of impact.

**If it is not possible to exclude underlying damage the patient should be referred for specialist opinion and a formal exploration**

Details of the wound should be quantified and documented. This includes the length and breadth of the wound and its depth if this is apparent. The state of the wound edges should be recorded, for example ragged and any obvious contamination documented. Factors such as wound edge contusion, devitalised tissue and contamination are all likely to lead to subsequent wound infection and should be considered when determining how the wound is to be managed.

Lacerations on glass require a cautious approach. Most glass is radio-opaque; therefore an x-ray is mandatory to exclude the presence of retained fragments of glass. As glass is sharp, late injuries can occur if retained as well as there being an increased risk of infection.

Substantial injuries including nerve injuries and tendon division have been described. If there is any doubt about completeness of removal, a second x-ray should be undertaken.

### **Nerves**

Assessment of abnormal sensation early after injury may be very difficult. Patients may report the presence of sensation even when subsequent exploration shows the relevant nerve has been divided. In general, ask whether the sensation is normal and compare it to the opposite side, rather than ask if sensation is present. Assess simple touch and two-point discrimination using an appropriate blunt device, for example a contoured paperclip. The area of abnormal sensation should be mapped out and recorded.

Motor function is more accurate to assess; however again requires anatomical knowledge. Motor power is recorded using the Medical Research Council (MRC) scale outlined in an appropriate scale (Box 8.4).

If the history, clinical symptoms and clinical findings suggest the presence of a nerve injury, the patient must be referred for exploration and if necessary nerve repair. This should be undertaken in the operating theatre, not the ED.

### **Vessels**

Assessment of vascular damage should include examination of skin pallor, capillary refill time, skin temperature and skin turgor.

Examination of peripheral pulses is essential. This may be undertaken by palpation; however if there is any doubt a Doppler device can be used to locate the pulse and pressures compared to the normal side. It must be recalled that vascular injuries may be present even in the presence of distal pulse. If an injury to a major limb artery is suspected, urgent vascular opinion is necessary. The need for referral is usually obvious, with direct vascular trauma in penetrating injuries; however following blunt trauma the clinician must maintain a high index of suspicion and seek expert opinion if there is any doubt. The aim is to minimise warm ischaemic time to reduce tissue damage, ideally less than 5 hours. These patients are likely to require angiography, pre or intraoperatively.

### **Tendons**

Tendon injuries are frequent in lacerations to the limbs, in particular the wrist and hand. An understanding of the anatomy of likely tendon injuries is vital, including examination of joint

movement powered by the tendon is required. Examinations of wounds are often misleading, as the tendon ends may retract out of sight. If there is any suspicion of tendon injury, referral for a formal exploration is required. This must be carried out in a well lit operating theatre using a tourniquet and by an experienced operator.

It is important to understand the principles of assessment of the hand and wrist after a laceration, as these are particularly common. Details are provided in Appendix 9.2.

## **ANALGESIA AFTER EXTREMITY TRAUMA**

Analgesia should be given to a patient at the earliest opportunity following an injury and may already have been administered by prehospital personnel in attendance.

Non-pharmacological interventions include elevation of the injured limb, immobilisation (without compression) support in a sling, or the application of ice-packs. Entonox™ (50% oxygen and 50% nitrous oxide) is a good analgesic and used appropriately is equivalent to about 10mg of morphine. It can be used for short-term pain relief, for example whilst splinting an injured limb or gaining vascular access prior to giving intravenous analgesia. Morphine is regarded as the gold standard for pain relief. It is best given as a 1mg/ml solution intravenously (10mg of morphine diluted to 10ml with sterile saline) and titrated to effect. Young, fit patients with fractures will often require relatively large doses (20-30mg), the frail or elderly smaller amounts. If given in this way, respiratory depression is uncommon, but all practitioners giving morphine must have the ability to maintain and support the patient's airway and ventilation and have access to naloxone.

Nerve blocks, in particular the femoral nerve block, may be considered as an alternative or an adjunct to morphine in a patient with a fracture of the neck or shaft of femur. For further details in Chapter 16.

### **Management of wounds**

No matter what the cause of extensive wounds, for example road traffic accident, fall from a height, or a gun shot wound, the principles of management remain the same and are summarised in Box 9.4.

#### **Box 9.4: Principles of wound management**

- Arrest of external haemorrhage

- Adequate analgesia
- Photograph
- Removal of gross contamination
- Reduction of any extruding bone, particularly if risk of skin necrosis
- Application of a sterile “Betadine” soaked dressing, covered with transparent, self-adhesive dressing (eg Opsite)
- Realignment of the limb (angulation, rotation and length)
- Splintage of the limb, without compression if possible
- Antibiotic therapy
- Anti-tetanus protection
- Early referral for specialist opinion

External haemorrhage should be controlled following a step wise process:

- Direct pressure
- Elevation
- Wound packing, direct pressure and elevation
- Tourniquet

In the presence of life threatening haemorrhage associated with limb trauma apply a tourniquet early. The patient should then be referred urgently for surgical repair. The time of tourniquet application must be noted.

**Consider the early use of a tourniquet when there is life-threatening haemorrhage from a limb.**

### **Simple wound closure**

Simple lacerations, particularly if superficial (affecting only the skin and subcutaneous fat), may be treated under local anaesthesia in the emergency department. Prior to closure the wound must be thoroughly cleaned, washed out and inspected to ensure there is no deeper extension. Materials such as grease, oil, coal dust or paint must be removed to maximise a chance of wound healing without complications and to produce the best cosmetic result. Particulate matter for example grit and soil, not removed may result in unpleasant tattooing of the skin.

Principles of adequate wound cleaning are:

- The provision of adequate analgesia
- Irrigation with clear fluid, preferably using pulsed lavage

- Physical removal of material from the wound with forceps
- Scrubbing with a brush

Wounds requiring more than the above, where tissues are severely contused or there has been significant contamination should be left open at the initial exploration and be reviewed at 48-72 hours. If clean at that time the wound can be closed, a technique called “delayed primary closure”) and this gives results very similar to those of primary closure. There is also a dramatic decrease in the risk of wound sepsis as a result of the thorough debridement, drainage and reduction in tissue swelling. Alternatively, the wound may be left open and closed at around 2 weeks when the tissues are healthy and the swelling has resolved (secondary closure). Extensive lacerations should be referred early for assessment by an appropriate surgical specialty. The key points with regard to skin wounds are summarised in Box 9.5.

It is essential to maintain adequate records. If wounds or lacerations are present, a diagram and description or photograph should be included in the documentation.

### **Extensive wounds**

If a wound cannot be closed and is not suitable to be left to heal by second intention (limited wound area with little cosmetic importance and no risk of contracture) the patient should be referred to the Plastic or Reconstructive Surgery service at the time of initial presentation. Management options include split skin grafts, full thickness skin grafts, rotation flaps (skin or myofascial) or free flaps.

#### **Box 9.5: Key points – skin wounds**

- Soft tissue injuries must be assessed in the overall context of the patient – is the patient hypovolaemic due to an associated injury?
- Vital signs must be recorded and monitored other than in trivial injuries.
- Assess which structures may have been injured – is formal exploration required?
- Assess degree of tissue damage and contamination – is primary closure indicated?
- If yes - cleaning and primary closure under appropriate anaesthesia
- consider prophylactic antibiotics
- If no - cleaning / debridement and delayed primary closure?
- cleaning / debridement and healing by secondary intention
- referral for flap or skin graft closure

## **Specialised wounds**

### *Gun shot wounds*

Gun shot wounds require special consideration because of the associated pathophysiology, the features of which are summarised in Box 9.6. Tissue destruction occurs as a result of the direct path of the bullet producing a permanent cavity. In injury due to high-energy transfer, temporary cavitation also occurs in which a cavity, 30-40 times the volume of the permanent cavity is created. This results in extensive soft tissue damage over a wide area and as it collapses, sucks debris into the wound. The principles of treatment are as illustrated above, taking into consideration the specific features of this type of injury.

### **Box 9.6: Specific characteristics of gun shot wounds**

- Kinetic energy of the missile
- Presenting area of the missile
- The missile's tendency to deform and fragment
- The tissue density
- Tissue mechanical characteristics
- Cavitation (permanent and temporary)
- Wound contamination

## **Blast injuries**

Extremities may be injured by blast injuries. Such injuries may be associated with penetrating injury due to shrapnel, which may act as high or low velocity projectiles. As well as penetrating injury, the blast may lead to a closed injury arising from the shock wave. This leads, *per se*, to injury to vascular structures in soft tissues causing gross soft tissue swelling and ischaemia. Consequently, exploration may be required to decompress fascial compartments (fasciotomies) and assess the viability of tissue. Primary closure in such injuries is contraindicated.

## **Fractures**

### *Assessment*

### **History**

The degree of violence sufficient to cause a fracture varies between patients. Older patients with osteoporosis may suffer fractures with minimal trauma, while younger patients may suffer high-energy injuries with no fractures. Such violence may be direct (assault with a blunt weapon) or indirect (twisting injury to planted foot causing a tibial fracture). Generally, a fall over a distance greater than body height is described as a high-energy injury.

A further factor determining the degree of damage is the direction of the force. A fall to the tip of the shoulder is likely to be associated with a clavicle fracture, while a fall to the outstretched hand may lead to fractures more distally in the upper limb. Application of force to the front of the pelvis (during a RTA) may lead to an “open book” type of fracture, whereas longitudinal force along the femur (from a head on impact during a RTA) may cause a shear fracture.

**During the secondary survey, all patients must be assessed for the presence of other possible injuries on the basis of the history and mechanism of injury.**

**Fractures of the extremities (especially open fractures) frequently look impressive but are rarely immediately life-threatening.**

## **Examination**

The classical signs and symptoms of a fracture are:

- Pain
- Deformity
- Swelling
- Tenderness
- Crepitus
- Loss of function

Fractures are invariably very painful. In the upper limb they result in restricted use and in the lower limb it is not usually possible to weight bear. Some fractures, for example femoral shaft fractures and pelvic fractures may be associated with significant blood loss, requiring appropriate resuscitation and stabilisation of the fracture during the primary survey.

Gross fracture displacement may be associated with potential skin compromise or neurovascular compromise. For this reason, following a prompt primary survey and provision of analgesia, the fracture should be realigned by manual in-line traction to restore congruity with the uninjured limb without awaiting x-rays of the injury. Only when the diagnosis is uncertain, for example in fractures near joints (which can be difficult to differentiate from fracture dislocations), should x-rays be undertaken prior to limb realignment.

Before obtaining x-rays, temporary splintage or support should be applied. In the upper limb the use of a sling may be sufficient or it may be necessary to use a plaster back slab, box or vacuum splint. In the lower limb a back slab, box splint, vacuum splint or traction splint may be appropriate depending on the site of injury. Such simple measures will help minimise pain from the fracture and during any movement while the x-ray is taken.

Fracture realignment achieves:

- Reduction in pain
- Reduced blood loss
- Reduced pressure on the skin
- Reduction or relief of any neurovascular compromise
- Reduction in the volume of fat embolism
- Reduction in the risk of deep vein thrombosis

### *Imaging*

Plain x-rays are the first line of imaging in musculoskeletal trauma. To be acceptable and adequate these should:

- Be clearly labelled and identified
- Indicate the side (left or right)
- Be bi-planar (for example, anteroposterior and lateral)
- Demonstrate the joint above and the joint below any bony injury

The exception to seeing the joint above and below an injury is a fracture at the extreme end of a bone, for example a distal radial or malleolar fracture where it is acceptable to obtain x-rays of the affected part only. However it is essential that an adequate examination of the whole of the affected bone is undertaken to determine any tenderness or other features that suggest a fracture.

The clinician should be mindful of fractures such as:

- injury of the medial malleolus and/or diastasis of the distal tibio-fibular joint that may be associated with fracture of the proximal fibula (Maisonneuve fracture)
- fracture of the mid-radius that may be associated with a dislocation of the distal radio-ulna joint (Galeazzi fracture)

### ***Interpretation of x-rays***

X-rays should always be examined on a light box. Fractures (Fig. 9.2) are identified by:

- A break in the cortex of the bone on one or more of the views.

- Angulation of bone, especially in children.
- A radiolucent line (in the case of a distracted fracture) or a radiodense line (in an impacted fracture) across part or all of the bone at the injury site
- Soft tissue swelling adjacent to the suspected site of the fracture
- Soft tissue evidence of intra-articular fractures. ( for example the fat pad sign in radial head fractures or a lipohaemarthrosis in intra-articular knee Injuries)



**Fig. 9.2: X-ray of the lower leg showing all the key features of a fracture.**

The principal aspects of the **fracture pattern** that the x-rays should define include:

- Is the fracture in the diaphysis, metaphysis or epiphysis? This predicts the healing potential and is important for planning what sort of fixation to use, if any
- The fracture pattern – is the fracture transverse, oblique or spiral? This indicates the stability of the fracture to axial loading after splinting/reduction and may determine whether operative treatment is required.
- Does the fracture involve a joint surface; if so, is there displacement of the subchondral bone (and hence articular cartilage)? Is the fracture actually a fracture-dislocation or fracture-subluxation? These are associated with risks of secondary osteoarthritis and may indicate the need for open reduction and internal fixation.
- Does the fracture compromise intra-articular blood supply leading to a risk of avascular necrosis?
- Does the fracture involve a growth plate (in children)? If the fracture line actually crosses it, this is associated with risk of growth disturbance.

Further information may be required to determine fracture configuration, particularly in complex tibial plateau, tibial plafond, acetabular and pelvic fractures. This is usually obtained

following specialist opinion. Although additional plain x-rays may be helpful, CT scanning is often required to elucidate more detail and allow planning of operative treatment.

Sometimes it is not possible to identify a fracture on an initial x-ray. If careful clinical examination indicates the presence of signs of fracture (bony tenderness and swelling in the case of undisplaced fractures), the management options are:

- Decide that it doesn't matter anyway (undisplaced fracture in an unimportant site, for example a lesser toe).
- Splint the limb and repeat the x-rays usually after a week or two. If the fracture is not visible on the original x-ray, it may become visible due to bone resorption at the fracture line. This approach is frequently used in scaphoid fractures, where a repeat x-ray (with repeat clinical examination) at two weeks is typically obtained.
- MRI scan. This is currently the most sensitive test for the presence of a fracture, as perifracture oedema is readily detected. Use of this investigation depends on availability. It is increasingly used in the diagnosis of proximal femoral fractures and scaphoid fractures.

#### *Initial management of fractures*

The principles of initial fracture management are as for any wound and are summarised in Box 9.7. It is important to resist the temptation to repeatedly inspect the wound, this should not occur again until the patient is in the operating theatre for wound exploration, debridement and lavage.

The **principles** of fracture treatment include:

- *Reduction* (i.e. reduce the deformity and replace the bone fragments in their anatomical position). Principles used in the initial management of some common fractures are shown in Box 9.7.
- *Maintenance of reduction* for as long as it takes for the fracture to unite
- Provision of *optimum conditions for healing*, both for the fracture and other damaged structures such as ligaments and joint capsules
- Maintenance of a healthy soft-tissue cover
- *Early mobilisation* of adjacent joints to prevent stiffness. This first phase of *rehabilitation* aims at achieving sufficient function to promote early return of the patient to activities of daily living and to work
- *Minimisation of complications* including infection

There are a wide variety of **methods** of fracture treatment, and none of them fulfils all of these conditions perfectly. All fracture treatment involves compromise, in order to minimise the risk of complications and the impact of any disadvantages of the particular method selected, while maximising its benefits. Broadly, there are six methods of fracture treatment:

- Immediate or early mobilisation: essentially ignoring the fracture, providing minimal support (e.g. a sling for a clavicle fracture)
- Support / immobilisation in a plaster cast or plastic brace (e.g. humeral fractures)
- Traction: immobilisation of the fracture by applying a longitudinal force along the limb (e.g. femoral fracture in a child)
- External fixation: a fixation device is applied across the fracture site with screws to the bone (e.g. open tibial fractures)
- Flexible internal fixation: an intramedullary nail or internal fixator (e.g. femoral shaft fractures)
- Rigid internal fixation: a stainless steel plate (e.g. forearm shaft fractures).

The essential logical step in planning treatment is to balance the risks of a given line of treatment against what is required to achieve a good functional result. It is vital that early management of fractures aids this, and does not compromise possible outcomes.

Two of the most common early techniques required in the safe and effective management of fractures are:

- Application of a back slab (Appendix 9.3)
- Application of a Thomas splint (Appendix 9.4)

#### Box 9.7: Treatment of skeletal injuries

<b>Site of fracture:</b>	<b>Preliminary stabilisation:</b>
• Clavicle, humeral neck	• Sling
• Humeral shaft	• U slab and collar and cuff
• Forearm	• Full arm back slab
• Distal radius, metacarpal	• Short arm back slab
• Femoral shaft	• Traction splint
• Around the knee, tibia	• Full leg back slab
• Ankle, foot	• Short leg back slab

#### Early complications after a fracture

#### *Impairment of circulation to the limb beyond the fracture*

It is vital to check for the presence of pulses below the fracture, while keeping in mind the possibility that, even with palpable pulses, arterial damage may have occurred. Pulses may be present initially and then disappear, for instance with intimal flap tears of the arterial wall. The only adequate guard against ischaemia due to arterial injuries is repeated examination of temperature, sensation and pulses of limbs. If a vascular injury has occurred, an emergency vascular surgical assessment is needed. The features of an acute vascular injury are detailed in Box 9.8.

#### **Box 9.8: Signs of vascular impairment**

- Pain
- Pallor
- Perishing cold
- Pulselessness
- Paraesthesia
- Paralysis

#### **Compartmental syndrome**

Compartment syndrome most commonly follows blunt trauma, although can occur in open fractures. Although commonly associated with tibial fractures, it also occurs after injuries to the forearm, foot and buttock. Progressive swelling within the fascial compartments results in muscle ischaemia. The classical features are:

- Progressive pain
- Pain of inappropriate severity to the background injury
- Extreme tenderness over the affected muscle group
- Pain with passive movements/stretching of the affected muscle group

If unrecognised, the late features may include paraesthesiae and pulselessness. The diagnosis may not be apparent in unconscious patients where based on the history and clinical findings (for example marked limb swelling) the clinician should have a low incidence for suspecting an acute compartment syndrome and seek urgent orthopaedic opinion for

compartment pressure studies and onward surgical management. The common sites and causes of compartment syndrome are summarised in Box 9.9.

#### **Box 9.9: Compartment syndrome**

Common sites for Compartment Syndrome:

- Lower leg
- Forearm
- Hand
- Foot
- Thigh
- Buttock

Causes of Compartment Syndrome:

- Fractures
- Crush injury
- Reperfusion injury (post correction of displacement causing vascular impairment)
- Pharmacological, e.g. anticoagulants

**Remember: severe pain after a fracture, persisting after immobilisation is due to compartment syndrome until proved otherwise**

#### *Timing of surgery*

Fractures with open wounds require emergency treatment (fracture to theatre time of 6 hours or less). Patients with acute compartment syndrome require immediate special decompression.

#### Dislocations

Dislocations are joint injuries where the two joint surfaces are no longer in contact. Partial dislocations also occur (subluxation). These are often difficult to distinguish from periarticular fractures and it is important to obtain adequate imaging. However, if there is gross deformity, neurovascular compromise or a problem with overlying skin, it is appropriate to reduce the deformity prior to obtaining x-rays. Experience will help, as there are a limited number of characteristic deformities with dislocations.

A typical dislocation is the anterior dislocation of the shoulder. It usually follows a fall on the outstretched hand. This is frequently recurrent, occurs in young patients and is associated with a typical deformity of the shoulder (empty glenoid). Neurovascular injuries occasionally occur (brachial plexus injury) but are uncommon. X-ray appearances are as shown. Reduction may be achieved by gravitational traction or Kocher's manoeuvre. For further details see Appendix 9.5.

## **Soft tissue injuries: potential pitfalls**

Soft tissue injuries around joints are common and there are several potential pitfalls. These include:

- Wrist sprains. Be sure to examine the anatomical snuffbox and exclude a scaphoid fracture. Longitudinal compression along the thumb metacarpal will typically cause pain if there is a fracture in the scaphoid, and there will be pain dorsally or over the scaphoid tubercle in most fractures.
- Beware of the possibility of an injury to the scapholunate ligament. Check for dorsal tenderness over the wrist. In such patients consider obtaining a clenched fist PA view of the wrist as well as the standard scaphoid views. An increase in the gap between scaphoid and lunate, denotes scapholunate instability and the patient must be referred to the appropriate specialist.
- Haemarthroses of the knee (early onset of severe swelling after injury) should be referred for assessment. This may require an MRI scan, examination under anaesthetic and sometimes arthroscopy. Adequate examination of the knee ligaments after severe injury is rarely possible in an awake patient. Examination of the knee must include the medial and lateral collaterals and the cruciate ligaments.
- The best sign of meniscal injury is joint line tenderness. Remember to look for a block to full knee extension by comparing the knee with the other side. Many meniscal injuries are now repairable, so it is important to try to establish the diagnosis. Assessment may require MRI or arthroscopy.
- Ankle ligament injuries can usually be accurately diagnosed by clinical examination. Tenderness is present over the ligament, and minor or absent over the malleoli. The usual pattern is that the anterior talo-fibular ligament is injured, giving tenderness just anterior to the lateral malleolus. Under this circumstance, diagnosis is provided by accurate clinical examination including a positive anterior drawer test at the ankle; the Ottawa rules indicate that x-ray is not necessary if there is no bony tenderness. Ankle ligament injuries are best treated by early physiotherapy and mobilisation, often with a supportive splint, rather than a cast.

## **Summary**

Soft tissue injuries and fractures are frequently challenging, potentially disabling and occasionally life-threatening. Careful assessment of the anatomical extent of these injuries, and appropriate treatment can make an enormous difference to the initial symptoms and the degree of long-term disability experienced by patients after trauma.

## **Appendix 9.1: The pathophysiology of soft tissue and bony injuries**

Injuries to the skin can be classified as:

- Contusions
- Abrasions (partial thickness injury)
- Lacerations
- Incised wounds

Closed injuries can involve the full thickness of the skin where the blood supply from vessels penetrating the fascia is damaged, as in a degloving injury. Skin wounds heal most commonly by primary or secondary intention.

### **Primary Intention**

Most glass or knife wounds to the skin will heal by primary intention and are suitable, where there are no other associated injuries, for suturing whereby the skin edges are brought together in close apposition. The distance to be traversed by the epidermis (on the surface) and granulation tissue beneath is minimal. Alternative methods of skin closure in this situation include steri-strips and skin glue in certain anatomical positions. The wound is sealed to bacteria after one or two days, and has a functional level of wound strength (for example is unlikely to burst in any normal activity) within three to four weeks.

### **Secondary intention**

Wound repair by secondary intention occurs in wounds that are left open, typically these include ragged or contaminated wounds. After wound debridement, wound closure is not possible and the wound fills with blood clot. This forms a protective scab over which underlying repair takes place. The defect fills with granulation tissue which in time will be covered by migrating epidermis from the edges of the wound.

Wound contracture (cicatrisation) occurs, reducing the size of the wound, which enhances wound healing. In certain situations, for example across the flexor aspect of a joint, this may result in joint contracture. Healing by second intention may be the method of choice in certain wounds seen in the emergency department, for example in wounds over the distal shin where the blood supply is poor.

### *Peripheral nerve injuries*

Nerves are injured most commonly by lacerations sustained by glass or knife wounds. They may also occur due to traction, where the nerve is stretched, giving rise to internal disruption.

An example includes injury to the brachial plexus, whereby the motorcyclist falls at speed onto their shoulder with a distracting force between the shoulder and the root of the neck.

*Nerve injuries can be divided into 3 types:*

- Neurapraxia occurs due to crushing of the nerve. This is a recoverable condition where the nerve fibres remain viable. Regeneration does not need to take place and recovery is relatively rapid.
- Axonotmesis follows a more severe crush or traction injury. The nerve sheath is not physically divided but the peripheral part of the nerve dies and regeneration is required. As the nerve sheath has not been divided, regeneration will eventually lead to reasonable recovery of function.
- Neurotmesis is the complete division of a nerve. Even after optimal repair, recovery is slow, and typically proceeds at best, at 1 mm per day. An injury distal in the limb may therefore take over a year to reach maximum recovery.

Healing in the central nerve system occurs only by the formation of scar (glial) tissue with no attempt at nerve regeneration. By contrast nerve repair does occur in peripheral nerves. Suturing techniques can be used to repair laceration of peripheral nerves but traction injuries are not usually amenable to repair. The prognosis of nerve injuries, whether repaired or not, is relatively poor in adults.

## **Vascular injuries**

Vascular injuries may occur following penetrating (glass or knives) or blunt trauma (knee dislocation). In the case of the former, the diagnosis is fairly obvious; however popliteal vessel injury may initially produce damage to the intima and later occlusion of the arterial lumen. The recognition of the potential for such an injury is important.

Severe vascular trauma often requires repair to avoid amputation. Alternatively if not recognised, gangrene may occur. Less severe injuries, or injuries in fit patients, may resolve by establishing an adequate collateral circulation – this may permit survival of the part, but not permit normal function, for example cause intermittent claudication. Some vascular injuries causing occlusion will recanalise and resolve over time.

## *Tendon injuries*

Tendon injuries occur most commonly due to lacerations, for example on glass or knives. Rarely they can occur due to acute rupture of a degenerate tendon, for example, the achilles tendon, patellar tendon, quadriceps insertion and the biceps tendon.

Surgical repairs permit healing by scarring but this may also lead to tethering of the tendon to the surrounding tissues. This is a particular problem in the hand where lacerations of the flexor tendons of the fingers may result in scarring between the tendon and its tendon sheath, producing functional impairment

## **Muscles**

The most common muscle injury is a simple contusion following a direct injury. Resisted movement, for example kicking a fixed solid object whilst extending the knee may result in a closed quadriceps muscle rupture. Similarly during sport a closed rupture may occur to the medial head of the gastrocnemius muscle.

Penetrating injuries commonly result in muscle trauma, the extent and significance of which depends on the site, penetration and posture of the patient.

## **Ligaments**

Ligaments are strong, relatively unforgiving structures that help maintain joint stability. They are therefore prone to injury and as a consequence, if significant produce joint instability. Prompt recognition and early repair affords the best chance of a good functional outcome in most injuries. Certain ligaments, e.g. cruciates, require reconstruction with grafts. Common ligament injuries include lateral ligament sprains of the ankle, medial collateral ligament injuries to the knee and gamekeeper's thumb, the latter common in skiers.

The injuries can be graded based on severity.

Grade 1 – Contusional injury, no ligamentous laxity

Grade 2 – Partial ligament rupture, some laxity but a firm end point

Grade 3 – Complete rupture, no end point on testing

Contusional and partial ruptures are invariably very painful. Complete ruptures may be relatively pain free.

Patient symptoms include:

Pain

Instability (e.g. ankle or knee giving way)

Functional impairment (decreased range of movement)

### *Fractures*

A fracture is an interruption to the cortex of a bone and may be complete or incomplete. Fracture patterns reflect the magnitude and direction of the force applied. For example, a direct kick to the shin may produce an undisplaced or minimally displaced transverse fracture. Angulatory forces produce oblique fractures and rotatory forces spiral fractures with or without butterfly components. High energy impacts commonly produce comminuted fractures which may be open or closed and may be associated with neurovascular deficit.

The severity of an open fracture can be graded as follows (Gustillo and Anderson, 1976):

- Wound caused by protrusion of bone (inside to out), < 1 cm, minimal contamination
- Wound < 5 cm, minimal contamination, would be closable by suture
- Wound > 5 cm, minimal contamination, and probably closable by suture (all after debridement of damaged tissue)
- Wound > 5 cm, severe contamination, or probably not closable by suture (all after debridement of damaged tissue)
- Open wound with significant neurological or vascular injury

The elderly population at risk from osteoporotic fractures which occur in cancellous bone commonly affecting the wrist (Colles' type fracture), the neck of the humerus and fractures of the neck of the femur. Children have their own patterns of injury which include greenstick fractures and epiphyseal injuries. Fractures may occur with minimal or no trauma if pathological, for example due to bony metastases.

Falls commonly involving angular, rotatory and loading forces adjacent to joints may result in dislocations (complete displacement of normal adjacent articular surfaces) or joint subluxations (incomplete displacement of normal adjacent articular surfaces).

### *Fracture healing*

The process of fracture healing is different when comparing cancellous and cortical bone. Fractures in cancellous bone, for example Colles' type fractures and humeral neck fractures, heal by a process of "creeping substitution" whereby the fracture ends join directly by establishing a bridge of woven bone.

Cortical fractures, for example the shafts of long bones, normally unite by a callus response.

This includes the phases of:

- Haematoma
- Inflammation and granulation tissue

- Formation of woven bone matrix
- Remodelling to lamellar bone

These stages are the same as those seen in soft tissue wound repair, except that the matrix elaborated by the proliferating cells is bone (and not fibrous tissue). The matrix formed is first woven bone which becomes sufficiently strong to afford mechanical stability to the fracture. Eventually remodelling into lamellar (cortical bone) occurs. The stability is much stronger if the callus is circumferential (the so called external or periosteal callus) Fig. 9.3).



**Fig. 9.3: Fracture healed by external callus formation.**

In certain circumstances, particularly high energy fractures, the periosteum may be damaged. Providing the fracture is adequately splinted, fracture union may be achieved by the formation of endosteal or intramedullary callus. This type of healing is seen routinely in undisplaced fractures or metaphyseal fractures where very little periosteal callus may be seen.

The external callus response relies on movement. If movement is completely abolished the callus response is also abolished. In these situations following, for example, rigid internal plate fixation, the fracture unites by a process of bone remodelling across the fracture gap – known as primary cortical healing. Rigid fixation is associated with slower fracture union and a significant risk of sepsis, and therefore has been replaced by other concepts, including intramedullary fixation and internal fixators which permits some movement and a callus response. The latter are like external fixators but applied internally – using a minimally invasive technique and bridging the fracture, which permits movement and a callus response.

The majority of fractures progress to fracture union. Fractures resulting from high energy forces and associated with significant periosteal damage may be slow to unite – delayed union. In this situation whilst the external periosteal callus may not form, the fracture unites by the formation of intramedullary or endosteal callus, providing the fracture is adequately splinted either internally or externally. Ultimately if these modes of fracture healing fail to occur a state of established non-union occurs.

## **Appendix 9.2: Examination of the tendons of the hand.**

1. Inspection of the hand will reveal a normal arcade of finger positions, which will move in a characteristic fashion when the wrist is flexed and extended. If this does not occur after a laceration, it is likely that a tendon injury has occurred.
2. The flexor digitorum profundus (FDP) powers the proximal and distal interphalangeal (DIP) joint of the fingers; while the flexor digitorum superficialis (FDS) powers the proximal interphalangeal (PIP) joint.
3. Lacerations over the middle phalanx can only injure the FDP, which will cause paralysis of the DIP joint.
4. Lacerations over the proximal phalanx or in the palm can injure both FDS and FDP. If both tendons are cut, then the finger will be immobile.
5. If only the FDS is injured, it may nonetheless be possible to move both PIP and DIP joints with the FDP.

To resolve whether the FDS is injured, assess the flexion of the PIP joint with the examiner holding the other fingers fully extended. This immobilises FDP, as this is a mass action muscle where all of the tendon slips must act together. If the FDS is acting, with the FDP immobilised, the DIP joint will be flaccid despite the PIP joint movement

### **Appendix 9.3: How to apply a back slab**

These are often applied poorly; too long (this immobilises other joints unnecessarily), too short, joint in the wrong position, inadequate mobilisation.

Wearing a disposable apron and gloves:

1. Get the patient in a comfortable position.
  - In an upper limb injury, have the patient with the forearm vertically in the air, with the elbow resting on a couch and the wrist in a neutral position
  - For a lower limb injury, have the knee supported so it is flexed. It is important to have the ankle roughly in neutral; it may be necessary to push the ankle upwards from a plantar flexed position.
2. Prepare the slab.
  - For an upper limb slab measure from a couple of inches distal to the elbow to the metacarpal heads. The upper limb slab will typically use eight layers of 6 inch plaster bandage
  - For a short leg slab, measure from a couple of inches distal to the popliteal fossa to the tips of the toes; the lower limb slab will use eight thicknesses of 8 inch bandage.
  - Apply a stockinette from above the proximal joint to the end of the limb.
  - Apply a plaster wool bandage evenly over the intended extent of the slab. Start with two turns proximally and end with two turns distally.
  - Wet the bandage thoroughly in lukewarm water.
3. Apply the slab.
  - In the upper limb, to the dorsum of the wrist, starting just (1 cm) proximal to the metacarpophalangeal joints, and finishing distal to the elbow. Apply a cling bandage.
  - In the lower limb, apply one slab to each side of the ankle, and one slab along the sole of the foot and dorsum of the calf. This gives a slab that has a U shape in section, and is sufficiently strong to resist ankle plantar flexion. Start by applying the slabs distally, so the slab on the sole of the foot reaches the toes. Apply a cling bandage and press the ankle up into a neutral position on your chest. Make sure that all of the toes are visible so that the circulation can be checked

#### **Appendix 9.4: How to apply a Thomas traction splint**

Two people are required to do this. Before commencing the patient should have received adequate analgesia, for example intravenous morphine, femoral nerve block or both.

- Use a splint of the appropriate size – at least 2.5 cm greater in diameter than the thigh
- Fit the splint with fabric to support the leg (calico or stockinette).
- Apply the skin traction device to the leg – this is a rubber device with a footpiece to take the traction string and held in place with a bandage. One person applies longitudinal traction to the foot while the other applies the device. It should NOT be used in patients with poor skin, for example patients with steroid damaged skin or rheumatoid arthritis.
- While the assistant controls the leg with the traction, the foot is passed through the splint ring. Lift and support the fracture, advancing the ring until it reaches the groin. The fracture should now be supported by the fabric.
- Support the fracture site with more fabric (usually gamgee)
- Attach the strings from the foot part of the traction device to the end of the splint, and construct a “Spanish windlass” with a pair of tongue depressors to apply traction.
- Traction has now been applied to the fracture; counter traction is supplied by pressure between the ring and the groin. If possible the ring should be pulled out of the groin by immediate application of a weight (usually 2.5 kg) applied to the foot of the splint via a pulley over the end of the bed. In any event, a splint without counter traction should not be left for more than 24 hours due to the risk of perineal pressure sores.

## **Appendix 9.5: Reducing dislocations**

### **Anterior shoulder dislocation**

#### *Gravitational method*

Under adequate pain control and with the patient sitting with his affected arm over the back of a chair, gentle longitudinal traction is applied. A soft pad is placed in the armpit.

Once the shoulder clunks into place, a check radiograph is taken to confirm the position.

### **Kocher's manoeuvre**

As before, adequate pain control is required. With the patient supine or semi recumbent, externally rotate the arm – this may not be easy. Without traction, adduction the arm across the patient's chest and internally rotate it. The manoeuvre is associated with a small risk of humeral neck/head fracture if there is a substantial impression so substantial force should not be used.

**The Hippocratic method is NOT recommended.**

***Longitudinal traction is applied to the patient's arm, with counter traction applied either by placing the (unshod) foot in the armpit, or getting an assistant to pull longitudinally in the same place with a towel. Pull along the line of the limb and use the foot or towel to press the humeral head laterally. This will press the humeral head back into the glenoid with a clunk.***

### **Other dislocations**

Other dislocations are associated with a much higher risk of neurovascular injury, for example elbow and (particularly) knee dislocations. The latter has a high risk of injury to the popliteal artery, and angiography should be considered after such an injury. If the patient presents with the knee still dislocated then it should be reduced. Careful clinical examination is required to assess the presence of a vascular injury.

Hip dislocations, are typically seen after road traffic accidents and may be difficult to differentiate from the (much commoner) proximal femoral fractures. The hip is usually dislocated posterior, which will give the limb a posture of shortening, flexion and internal rotation. Dislocations usually occur in young patients and are often associated with a posterior lip fracture of the acetabulum, or a sciatic nerve injury. Careful clinical examination is required to exclude this. A major risk with this injury is of avascular necrosis of the femoral head. Urgent reduction is VITAL, as there is clear evidence that the risk of avascular

necrosis increases with the length of time that the hip is dislocated. Reduction requires general anaesthesia with muscle relaxation.

Ankle and hindfoot dislocations may tent the overlying skin dangerously, and are injuries that require treatment before x-rays are obtained. Following adequate analgesia, longitudinal force is usually sufficient to reduce the deformity, reduce the tension on the overlying skin and allow x-rays to be taken.

# **CHAPTER 10**

## **TRAUMA IN CHILDREN**

### **Objectives**

At the end of this chapter, the reader should:

- Understand the system used to assess and treat the injured child
- Understand the anatomical and physiological features specific to children relevant to the management of trauma
- Appreciate how the management of traumatic injuries in children differs to that in adults
- **Be aware of the features that may help in offering a prognosis following severe injury**

An outline of injury patterns commonly seen in children is given in Appendix 10.1 at the end of the chapter.

### **Introduction**

Trauma is the commonest cause of death in children over the age of one year and sadly the majority occur before the child arrives at hospital. The pattern of injuries in children differs to that seen in adults; mortality is related primarily to head injury. Haemorrhagic shock and severe life threatening chest injuries are uncommon. In the United Kingdom in 2003, approximately 250 children under the age of 15 years died because of injury. In 2004 1370 children sustained trauma and were entered onto the TARN UK (Trauma Audit and Research Network, personal communication) database from participating hospitals. Of these 38 died as a result of their injuries (2.7%). It has been estimated the average Emergency Department (ED) may see as few as two to four severely injured children a year, therefore exposure to children with life-threatening trauma is an uncommon event for most trauma teams. Consequently, a methodical approach to the management of the injured child is essential.

### **Preparation**

It is essential to have a methodical approach to the assessment of an injured child to avoid missing injuries. Traumatic injury and resuscitation are dynamic processes that require assessment and reassessment. The approach described is in line with those used by the Advanced Paediatric Life Support (APLS) and European Paediatric Life Support (EPLS) programmes.

Warning that a child with trauma is en route to the ED allows the necessary members of staff required to care for the child to be contacted, appropriate roles assigned and preparation of the relevant equipment. Paediatric staff can provide support to staff in the ED and may be able to offer additional support to the child's family during the resuscitation. This shared responsibility aids continuity should the child be transferred to Paediatric Intensive Care Unit (PICU) or a paediatric ward. Most parents wish to be given the opportunity to remain with their child even if invasive procedures are required so the additional role of chaperone needs to be assigned. Local policies for caring for relatives in the resuscitation room should be used or further information can be obtained from the ERC.

## **Initial Assessment and Resuscitation**

### **The “5-second round”.**

As for adults, the team leader's actions will be dictated initially by the extent of the care delivered by the pre-hospital team. Where minimal intervention and treatment has been provided pre-hospital, the medical team leader will often carry out a very quick overview of the patient to try and identify obvious life-threatening conditions. Depending on the age of the patient, this is achieved by looking at and listening to them, while at the same time feeling the patient's periphery. The aim is observation, not intervention. Quickly look and ascertain:

- Is the child alert, talking, crying, moving or unconscious?
- Is their airway clear, acceptable, obstructed needing immediate intervention?
- Is ventilation and oxygenation adequate or unacceptable?
- Are there signs of massive external haemorrhage or severe hypovolaemic shock
- On exposing the patient, feel for pulse, skin temperature. What is their skin colour, are there any major deformities of head, neck, trunk or limbs?

Depending on the findings, immediate action may be to direct the team to:

- Clearing and maintaining an airway, give oxygen and support ventilation

- Control of external haemorrhage, start CPR
- Request further help or support e.g. paediatric specialists
- No immediate life-threatening injuries, proceed with full primary survey

## **Primary survey and resuscitation**

During the Primary Survey a member of the team must be assigned to obtain details from the pre-hospital staff, witnesses (if present) and parents of the child regarding mechanism of injury, treatment administered at scene or en route, the child's past medical history, medications, allergies, immunisation status and an estimate of when food or fluid was last ingested (AMPLE). An estimate of the child's weight needs to be made as soon as possible as most drugs are given on a dose/kg basis. At birth a child weighs approximately 3 kg, this increases to about 10 kg at the age of one year. For children aged 1 to 10 years, weight can be estimated by the formula;

$$\text{Weight (kg)} = 2 \times (\text{age} + 4)$$

This allows calculation and preparation of the doses of medications likely to be required.

## **Airway and cervical spine control**

Airway obstruction from the tongue, foreign material, aspiration and apnoea are particular hazards to the injured child with a decreased level of consciousness. Assessment and management of the airway follows the same principles as for adults (Chapter 3). If there are signs of airway obstruction, the first step is to use a jaw thrust (Fig. 10.1). Obvious liquid material should be removed from the oropharynx using a soft, flexible catheter. An oropharyngeal airway of the appropriate size can be inserted over the tongue under direct vision, to help maintain a patent airway. It must **not** be inserted "upside-down" and rotated as in an adult as this may damage the soft palate and cause bleeding. Nasopharyngeal airways are not used as they tend to damage the adenoidal tissue that is prominent in children and again this can cause significant haemorrhage. While attempts are being made to clear the airway, a high concentration of oxygen must be given. Airway obstruction in the paediatric trauma patient is rarely ever complete. If ventilation is inadequate, it should be supported using a bag-mask device (see below). If the airway remains obstructed, the next manoeuvre

would be either the insertion of an LMA, or tracheal intubation. The latter will require the use of sedative and neuromuscular blocking drugs and even then intubation in injured children can be difficult and **must only be performed by those well practised in the technique**. The only exception to this is the child who has suffered a cardiac arrest.

Children are at greater risk of regurgitation and aspiration because of a shorter oesophagus, a lower pressure gradient between the larynx and stomach, lower oesophageal sphincter tone and gastric distension from swallowing air. A naso or oro-gastric tube should be used to decompress the stomach if this is excessive, although the oral route is preferred if there is craniofacial trauma. This can be left in place during bag-mask ventilation to prevent gastric distension, providing it does not cause too large a leak around the facemask.



**Fig 10.1: Jaw thrust and facemask being held on the face of a small child.**

Note the avoidance of pressure in the submandibular area that can obstruct the airway.

Oro-tracheal intubation is preferred in the resuscitation room; naso-tracheal intubation can result in neck extension and possible exacerbation of a cervical spine injury or damage to the adenoids and subsequent haemorrhage. At worse there is the risk of penetration through a fracture in the base of the skull. Cricoid pressure must be applied to reduce the risk of aspiration during induction and will decrease the volume of air forced into the stomach of small children during bag-mask ventilation. Cricoid pressure should not be released until

correct placement of the tube is confirmed by a normal end-tidal carbon dioxide waveform and bilateral breath sounds. Major complications have been reported in 25% of children who required intubation, 80% of which, were life threatening.

The appropriate sizes of a tracheal tube for a child can be calculated as follows:

**Oral Endotracheal Tube**

$$\text{Internal diameter (mm)} = (\text{age}/4) + 4$$

$$\text{Length (cm)} = (\text{age}/2) + 12$$

**Nasal Endotracheal Tube**

$$\text{Internal diameter (mm)} = (\text{age}/4) + 4$$

$$\text{Length (cm)} = (\text{age}/2) + 15$$

Potential difficulties in managing the paediatric airway can be minimised by an awareness of the anatomical differences between the adult and child airway (Box 10. 1). Indications for intubation and ventilation are outlined in Box 10.2.

**Box 10.1: Structural characteristics of the paediatric airway**

**Anatomical feature:**

- Large occiput (<3 years), short neck
  - Infants (<6 months) breath via the nose
  - Relatively large tongue, floppy epiglottis
  - Relatively short trachea
- Effect:**
- Head and neck flexes
  - Complete airway obstruction may occur if blocked by blood, oedema
  - Obscures view of glottis
  - Risk of right main bronchus intubation

**Box 10.2: Indications for intubation and ventilation**

- Inability to oxygenate and ventilate with a bag-valve-mask technique
- Obvious need for prolonged control of the airway e.g. multiple injuries

- Decrease in the level of consciousness e.g. head injury
- Inadequate ventilation e.g. flail chest, exhaustion
- Persisting hypotension despite adequate fluid resuscitation

If a surgical airway is required, needle cricothyroidotomy is the recommended technique in children under the age of 12 years. This is described in Chapter 3. In children this is a difficult, high risk procedure as paratracheal placement of the cannula and insufflation of oxygen will result in massive mediastinal emphysema that may be fatal. Furthermore, even if successful, it may cause damage to the cricoid cartilage, the only complete ring of cartilage in the airway, causing collapse of the upper airway. Healing results in tracheal stenosis and long term airway problems.

Most children can be oxygenated adequately using a good technique with a bag-mask and airway while expert help is obtained.

Even though the incidence of cervical spine injury is low in children, the child's head should be immobilised, initially by the airway person, using manual in-line stabilisation, unless the child is already immobilised. Ideally, an appropriately sized collar, lateral head supports and straps are required to immobilise the head and neck. In practice, this ideal cannot always be achieved in very young children or infants; attempts to stabilise the head and neck in this manner in agitated or uncooperative children may cause more harm than benefit if they simply struggle in an attempt to remove the collar which may aggravate pre-existing injuries. In the unconscious child, care must be taken to ensure that the application of strapping and an appropriately sized collar does not impair ventilation or obstruct the jugular veins and raise ICP. The cervical collar must the correct size for the child; too small and the head will flex, too large and the neck is not immobilised. In small children a vacuum splint can be used to immobilise the whole child, including head and neck. Any movement of the child must be performed in a controlled manner, ensuring the spine is immobilised until a spinal injury is

excluded by a normal neurological examination and radiological assessment. Prior to fitting the collar the neck should be inspected for wounds, venous distension, surgical emphysema and tracheal deviation.

### **Breathing and ventilation**

The adequacy of ventilation should be assessed once a patent airway is assured and oxygen given as appropriate. This is done by observing movement of the chest wall, counting the respiratory rate, examining the percussion note and listening for air entry. Recession of the intercostal and subcostal muscles, flaring of the nostrils and grunting is indicative of respiratory distress. A depressed level of consciousness or agitation are signs of hypoxia, cyanosis is a late sign of hypoxia; children more commonly appear pale. If breathing is ineffective, ventilation should be assisted initially using a bag-mask device with 100% oxygen before progressing to a definitive airway. A naso- or orogastric tube should be passed to minimise the risk of gastric distention. The treatment of life threatening chest injuries is similar to that in adults.

### **Circulation and control of haemorrhage**

Although the blood volume per kg body weight is higher in children than adults (100 ml/kg in a neonate, 80 ml/kg in a child, 70 ml/kg adult) the absolute circulating blood volume is small and the loss of relatively small absolute volumes can result in significant haemodynamic compromise. Children are however, extremely efficient in compensating for the loss of blood as a result of a relatively greater ability to increase systemic vascular resistance and heart rate. The signs of early haemorrhagic shock are subtle in children and the onset of decompensation is abrupt. Hypotension is therefore a late and pre-terminal sign. Isolated intracranial haemorrhage in infants may result in hypovolaemic shock but is not associated with hypovolaemic shock in older children.

### *Assessment of the circulation*

Trying to estimate blood loss inn the injured child is difficult. Heart rate is unreliable as a tachycardia is dependent on too many other factors; an anxious, frightened child in pain from a relatively minor injury will be tachycardic. Respiratory rate will also be increased. More reliable signs are capillary refill time (CRT, normal <2 seconds), skin colour and temperature, and conscious level. The team member allocated to deal with “circulation” should therefore make these their priority in assessment.

Pulse oximetry may prove difficult in the presence of shock, hypothermia, peripheral vasoconstriction or a restless child. This is followed by attaching the ECG to allow continuous monitoring of the heart rate and rhythm, using lead II. The blood pressure (BP) is then measured by indirect techniques using appropriately sized cuffs. Normal vital signs vary significantly with a child's age, but these values must either be known or readily available to allow an accurate assessment to be made (Box 10.3). In the initial stages, evaluation of the vital signs should be performed every five minutes. Any obvious blood loss needs to be controlled by direct pressure as soon as it is identified.

Loss of up to 25% blood volume will be associated with a slight increase in CRT, cool peripheries and the child may be slightly agitated. Heart rate will be more dependent on the factors described above. As blood loss increases, so does the CRT. The peripheries will become cold and mottles and the child increasingly lethargic. Once more than 40% of the estimated blood volume is lost, peripheral perfusion will be absent, often with no discernable CRT and cold, waxy-looking skin. Most children will be unconscious, with sighing, agonal respiration. The only reliable change in heart rate is an accompanying bradycardia that usually precedes cardiac arrest.

<b>Box 10.3: Normal vital signs in children</b>				
<b>Age</b>	<b>Weight (kg)</b>	<b>PR (beats/min)</b>	<b>RR (breaths/min)</b>	<b>Systolic BP (mmHg)</b>
3-6 months	5-7	100-160	30-40	70-90

<b>Age</b>	<b>Weight (kg)</b>	<b>PR (beats/min)</b>	<b>RR (breaths/min)</b>	<b>Systolic BP (mmHg)</b>
3-6 months	5-7	100-160	30-40	70-90

1 year	10	100-160	30-40	70-90
2 years	12	95-140	25-30	80-100
3-4 years	14-16	95-140	25-30	80-100
5-8 years	18-24	80-120	20-25	90-110
10 years	30	80-100	15-20	90-110
12 years	40	60-100	12-20	100-120

Venous access is a high priority in the child with severe injury and should be delegated to the most appropriate person. The optimal sites for peripheral venous access are the veins on the dorsum of the hand or foot and the saphenous vein anterior to the medial malleolus. The antecubital vein is often easy to cannulate but the catheter is readily kinked by flexion of the elbow. The elbow should be splinted if used. Two short, wide bore intravenous cannulae are the ideal, the size dictated by the size of the child.

In the presence of shock, if intravascular access is not achieved within 90 seconds via the percutaneous route, the intraosseous route should be used in children up to six years of age. The most common site used for intraosseous access is 2-3 cm below the tibial tuberosity on the flattened medial aspect of the tibia, alternatively the anterolateral surface of the femur, 3 cm above the lateral condyle, and the medial malleolus (Fig. 10.2). Access is achieved using a specially designed intraosseous needle (Fig. 10.2). There are mechanised devices available to assist in insertion of the needle (Fig. 10.3). Fractured bones should be avoided, particularly those with fractures proximal to the site of entry. The technique of insertion is described in Box 10.4.

Alternatives to the above are percutaneous cannulation of either the femoral or central veins or venous cutdown. The latter can be difficult in the shocked child, particularly when the physician is inexperienced. Ultrasound guidance may help achieve successful central venous access.



**Fig. 10.2:** Sites for insertion of an intraosseous needle; below the tibial tuberosity, anterolateral femur and medial malleolus. Close-up of intraosseous needles.



**Fig. 10.3:** Bone injection gun (left) and EZ-IO (right).

Initially, a bolus of a warmed, isotonic crystalloid (10 to 20 ml/kg) is given. Hypotonic or glucose-containing fluids must not be used. In small children the most effective and accurate method of giving fluid is via a syringe. The person tasked with dealing with the circulation should make a careful record of the volumes administered, particularly in very small children. The child should be reassessed after each bolus; improvement will be evident by a fall in heart rate, an improvement in capillary refill and an increase in blood pressure. Failure to respond to fluid should prompt a search for other causes of shock whilst further boluses of fluid are administered. Any child presenting with profound haemorrhagic shock or who fails to respond to 40ml/kg of crystalloid and/or colloid should receive warmed, packed red blood

cells (10ml/kg). If type specific or fully cross-matched blood is not available within 10 minutes then Group O Rh negative blood should be used. Rapid review by an experienced surgeon is essential in the management of the hypovolaemic child.

**Box 10.4: Technique for insertion of intraosseous needle**

- A pillow should support the knee and proximal lower leg. The skin should be cleaned.
- In a conscious child, infiltrate the area and underlying periosteum with 3-5ml 1% lignocaine.
- A 16-18g intraosseous needle is inserted 90° to the skin and advanced until a 'give' is felt as the cortex is penetrated. Making a small skin incision at the point of entry and a 'twisting and boring' motion of the needle facilitates insertion and entry through the cortex by the trocar and needle.
- Remove the trocar and attach a syringe. Infusion of saline can, if necessary clear the needle of any clot
- Correct placement is confirmed by aspiration of marrow content and easy infusion of fluid. The aspirated sample can be sent to the laboratory for routine bloods and used for bedside glucose estimation.
- Fluids need to be administered in boluses. The flow rates are high enough for volume resuscitation. Intraosseous lines need to be replaced by venous cannulation as soon as possible.
- Complications are rare but include extravasation, subperiosteal infusion, fat and bone marrow embolism, osteomyelitis, damage to the growth plate and cortex, pain and subcutaneous oedema.

FAST (Focused Assessment by Sonography in Trauma) examination in the emergency department will help clinicians rapidly identify free fluid in the abdominal cavity or pericardium, and an experienced operator can quickly confirm the presence of a pneumothorax or a haemothorax. This may aid triage to surgery. Although CT examination facilitates grading of intra-abdominal solid organ injury severity, it is current practice in many centres to use physiological rather than anatomical criteria to decide on the need for laparotomy in children. Haemodynamic instability, as defined by "the need for blood transfusion in excess of 25 ml/kg within the first two hours" has been identified as a strong

indicator of a major hepatic vascular injury. Treatment algorithms have been proposed to aid decisions regarding operative management in children with severe hepatic and splenic injury.

### **Disability of the CNS (neurological assessment)**

The initial evaluation of the central nervous system in an injured child incorporates assessment of the level of consciousness by AVPU or preferably the GCS (Box 10.5) and examination of the pupil size and reactivity. The neurological status needs to be repeated over time and interpreted in line with the child's clinical condition. This can be performed by a member of the nursing or medical team and should be recorded appropriately. The assessment of GCS is not very precise in children under the age of five years. The presence of abnormal posturing, limb movement and tone should be noted.

#### **Box 10.5: Glasgow Coma Scale - Age <4 years**

Eye opening:

Spontaneously	4
To verbal stimuli	3
To pain	2
No response	1

Verbal response:

Alert, babbles, words to usual ability	5
Less than usual words, spontaneous irritable cry	4
Cries only to pain	3
Moans to pain	2
No response to pain	1

Motor response:

Obeys verbal command	6
Localises to pain	5
Withdraws from pain	4
Abnormal flexion to pain (decorticate)	3
Abnormal extension to pain (decerebrate)	2

### **Exposure and environmental control**

On arrival, the child needs to be undressed and covered to prevent a drop in temperature. Small children have a high body surface area to weight ratio, which is at its greatest when the child is newborn. Consequently, children lose heat much more rapidly than adults do; for example, newborn children will lose 1°C every four minutes if left uncovered. Any part of the body covered by splints or collar should be examined. The back should be inspected during the log roll. Wounds need to be photographed and then covered in aqueous iodine soaked dressings e.g. Betadine (unless allergic to iodine). A member of the nursing team should have the role of monitoring the child's temperature and minimising heat loss by ensuring that all fluids are warmed, exposure is minimal and external heating devices are used appropriately.

### **Analgesia**

Analgesia should always be administered if required. An intravenous opiate is the most appropriate method in severe pain. Morphine must be diluted (usually to 1mg in 10ml) and given according to the estimated body weight (0.015-0.03mg/kg, Chapter 15) and the dose checked by a member of the nursing staff to ensure accurate and safe administration. The child's pain should be re-assessed at regular intervals by a nurse or doctor and further doses administered if necessary. An alternative is ketamine, a particularly useful drug in children with circulatory compromise. The intravenous dose is 0.25-0.5mg/kg. It can also be given intramuscular, 0.5-1.0mg/kg. Other methods such as distraction techniques, regional nerve blocks, splintage and immobilisation will be applicable to some patients.

### **Secondary survey**

If not already available, the nurse assigned to the relatives should obtain details of the child's past medical history, medications, allergies, immunisation status and an estimate of when

food or fluid was last ingested (AMPLE). Once the initial primary survey and resuscitative efforts has been completed, a secondary survey is performed. As in adults this consists of a detailed “head to toe” examination of:

- The head, face and neck, including eyes and ears
- The extremities, including all joints
- Repeat examination of the chest and abdomen
- Rectal examination and log roll

Appropriate radiological investigations are performed. The high incidence of spinal cord injury without radiological abnormality (SCIWORA) in children should reinforce the importance of a detailed neurological examination as the best method of identifying cord injury.

If not already done, a naso-gastric tube should be inserted. The stomach will need to be decompressed via the oral route if there is the possibility of a base of skull fracture. Urinary catheterisation is reserved for those children unable to pass urine spontaneously or in those where continuous monitoring of urine output is essential. A urine bag should be used to monitor the urine output of infants.

Occasionally, it is necessary for the child to be transferred urgently for surgery to control haemorrhage and it will not be possible to complete a secondary survey. If so, the surgeon transferring the child to theatre must be informed of this and the need for further examination recorded in the notes.

### **Investigations**

Routine laboratory investigations include FBC, blood group and cross match and amylase. Additional biochemistry such as urea and electrolytes (U&E) or toxicology may be required but are not routinely performed in all children. Coagulation studies should be performed on patients with severe trauma. Hypotension, a GCS <13, compound or multiple fractures and

extensive soft tissue injuries are all associated with disturbances in coagulation. Arterial blood gas analysis is mandatory in the presence of pulmonary injury and in any shocked child as the base deficit quantifies the severity of shock and predicts the development of multiple organ failure and continuing haemorrhage. The admission base deficit can be a useful marker of physiologic derangement and should alert the team to uncompensated shock or potentially lethal injuries. A bedside estimation of the blood glucose must always be performed. Urinalysis is required once the patient is catheterised or passes urine spontaneously.

Standard radiological investigations in the resuscitation room include x-rays of the chest, pelvis and cervical spine. A chest x-ray will identify any significant pneumothorax, haemothorax or pulmonary contusion. Imaging of the cervical spine consists of a cross table lateral view, antero-posterior view and an open mouth view. Imaging the high cervical spine can be difficult in the unconscious or intubated patient and the lateral cervical spine x-ray is best performed with traction on the arms. CT imaging of the cervical spine may be required to view C1/C2. However, it is important to remember the higher incidence of ligamentous injury and neurological injury without bone abnormality in children, which may not be apparent on CT. Other modalities such as MRI should therefore be considered if clinical examination suggests the possibility of cord injury despite normal plain radiography. However, the child must be fully resuscitated and haemodynamically stable before such an examination is undertaken.

Peritoneal lavage, although sensitive for identifying intraperitoneal blood, does not provide any information on the type or severity of organ injury and CT examination of the abdomen and pelvis is the method of choice when further evaluation of the abdomen is required. Children however should only be transferred to the CT scanner if they are haemodynamically stable or the same level of ongoing resuscitation and monitoring can be assured. Abdominal ultrasound within the ED is very useful in the hypotensive child considered too unstable to be transferred. In the stable child, ultrasound may have a role in prioritising imaging studies.

## Audit and review

The use of trauma scores can aid internal audit by highlighting those cases for review. The Paediatric Trauma Score (Box 10.6) is an injury specific scoring method designed for children and is used to identify those cases that should be reviewed formally. Such audit meetings or case reviews need to be multi-specialty. Membership and submission of data to a trauma registry is to be commended.

Box 10.6 The Paediatric Trauma Score			
Variables	+2	+1	-1
<b>Airway</b>	Normal	Maintainable	Not maintained
<b>CNS</b>	Awake	Obtunded	Coma
<b>Body weight (kg)</b>	>20	10-20	<10
<b>Systolic blood pressure (mm Hg)</b>	>90	50 - 90	<50
<b>Open wound</b>	None	Minor	Major
<b>Skeletal injury</b>	None	Closed fracture	Open/ multiple fractures

## Prognosis

No motor response from the GCS, the Injury Severity Score (ISS) – (International Classification of Disease, Ninth Revision-based Injury Severity Score) and “unresponsive” from AVPU score have been identified as independent predictors of inpatient mortality in paediatric patients with blunt trauma. In children sustaining severe, multiple traumatic injuries a systolic blood pressure below 60 mmHg on admission represented the single greatest predictor of fatality. Those with a GCS <8 on admission or penetrating trauma were also at greater risk of death.

## Summary

Although trauma is the commonest cause of death in children over the age of one year, it is relatively rare for the ED to be exposed to children with significant injury. It is essential that clinical staff have a systematic approach to the management of an injured child, are aware of how these patients differ from adults in their response to injury and have a selection of appropriate paediatric equipment readily available.

## **Appendix 10.1: Injury patterns in children**

### **Head injuries**

Head injury is the commonest cause of death in children over the age one year. Severe cerebral oedema (Fig. 10.4) occurs three to four times more frequently in children than adults and often occurs in the absence of contusion, ischaemic brain damage or intracranial haematoma



**Fig. 10.4: CT cerebral oedema**

### **Cervical spine injury**

The incidence of spinal cord injury amongst paediatric trauma patients is low (1.5%) and can be accounted for by the specific anatomy of the cervical spine (Box 10.7). In children, 60-80% of spinal injuries are in the cervical region compared to 30-40% in adults. The frequency of injury in the upper cervical spine injury (52% at C1-4) is nearly twice that in the lower cervical spine (28% at C5-C7) with lower cervical spine injuries predominating in those over eight years. Up to 50% of children with a neurological deficit due to a cervical spine injury may have no radiological abnormality; this condition is referred to as "Spinal Cord Injury Without Radiological Abnormality" (SCIWORA) and is due to transient vertebral displacement causing spinal cord injury but with subsequent realignment to a normal configuration with an apparently normal vertebral column appearance on x-ray examination.

Mortality rates from cervical spine injury have been shown to be higher in younger children (<10 years) than in older children (30% v 7%), but major neurological sequelae are uncommon in children who survive

**Box 10.7 : Structural characteristics of the paediatric cervical spine**

Anatomical feature	Effect
• Interspinous ligament and cartilaginous structures have greater laxity and elasticity	• Greater mobility and less stability
• Horizontal angulation of the articulating facets and uncinate processes	• Greater mobility and less stability
• Anterior surface of vertebrae wedge shaped	• Facilitates forward vertebral movement resulting in anterior dislocation
• Underdeveloped neck musculature	• More susceptible to flexion and extension injuries
• Head disproportionately large	• Causes torque and acceleration stress to occur higher in C spine and more susceptible to flexion and extension injuries

**Thoracic injury**

Chest injuries are predominantly due to blunt trauma and represent between 0.7%-4.5% of all paediatric trauma cases. . The most important factor in thoracic trauma in children is that it is a marker of significant injury, associated with extrathoracic injury in 70% of cases. Mortality is strongly related to the presence of these other injuries. The child's skeleton is incompletely calcified and is therefore more compliant. A good example of this is the rarity of rib fractures in children; when they occur there is usually serious underlying lung injury and mortality

increases in proportion to the number of ribs fractured. An isolated simple pneumothorax is relatively rare in a child but tension pneumothorax develops more readily (Fig. 10.5). Pulmonary contusion is the most common injury seen after blunt chest trauma and may occur in association with pneumothorax, haemothorax or post-traumatic serosanguinous effusion. Massive haemothorax is rare in children because blunt trauma rarely results in haemorrhage from major intrathoracic arteries.



**Fig. 10.5:** Tension pneumothorax, right lung.  
Note marked displacement of the mediastinum

### **Abdominal and pelvic injury**

Although children have proportionally larger solid organs that are more vulnerable to penetrating injury, blunt trauma occurs more frequently. The spleen is the organ most commonly affected in blunt abdominal trauma with hepatic injuries the second most common, occurring in 3% of children with blunt abdominal trauma. Non-operative management is the preferred method of treatment for solid organ injury in children, as haemorrhage is generally self-limiting and responds well to fluid or blood transfusion. Figures from one paediatric trauma centre report only 4% of blunt liver injuries and 21% of blunt splenic injuries requiring operative management.

The young child's predilection to aerophagy (air swallowing) can lead to painful abdominal distension, making examination difficult and increasing the risk of regurgitation and aspiration. Intra-abdominal injury may present with peritonism, distension and signs of circulatory shock but on many occasions the signs may be absent or subtle. Repeated examination, observation and monitoring of the vital signs are essential in the child with a possible abdominal injury.

Pelvic fractures (Fig. 10.6) are less frequent in children than in adults and are less likely to cause severe retroperitoneal haemorrhage. Nevertheless, severe injury can occur in children and may not be overlooked. The treatment principles include resuscitation, analgesia, immobilisation, and if necessary, embolisation and extraperitoneal packing.



**Fig. 10.6: Pelvic fracture in an 8 year-old child.**

Note that the articulations within the pelvic girdle are not ossified. There is an iliosacral dislocation , bilateral pubic fractures and a scrotal haematoma.

## Musculoskeletal injury

Orthopaedic trauma represents a substantial proportion of all paediatric trauma. Review of the 1997 Kids Inpatient Database in the USA showed that the most common injuries are fractured femur (21% of all orthopaedics trauma), fibula, tibia or both (21.5%), followed by humeral fracture (17%). Vertebral fracture represented 5.2% of admissions.

The paediatric skeleton contains growth plates and a thick, osteogenic periosteum whilst the bones are more porous and elastic. Fractures are consequently less likely to cross both cortices or be comminuted while bone healing is very rapid, primarily because of the osteogenic periosteum. Delayed or non-union rarely occurs in the paediatric age group. However growth plate injuries and epiphyseal injuries can lead to growth disturbance that may be significant (Fig. 10.7). Dislocations and ligamentous injuries are uncommon in children compared with adults. Children with multiple injuries can have occult axial fractures and epiphyseal injuries, which can be difficult to diagnose in the early stages of assessment so necessitating repeat and detailed examination.



**Fig. 10.7(a): Tibial fracture**

**Salter Harris Type I**

#### **Non-accidental injury (NAI)**

The possibility of NAI should always be considered when assessing a child with traumatic injuries. NAI has been shown to account for up to 10.6% of all blunt trauma in those under five years. Children injured as a result of child abuse tend to be younger, sustain greater



**Fig. 10.7(b): Distal radial fracture**

**Salter Harris Type II**

trauma, are more likely to have a pre-injury medical history and retinal haemorrhages when compared to children with unintentional injuries. One recent study examining abusive head injury as a cause of death in 42 children showed 81% had retinal haemorrhages. Children suspected of being abused need to be referred to the appropriate authorities, according to local policy. Child protection procedures should be instituted in every case of suspected child abuse.

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# **CHAPTER 11**

## **TRAUMA IN WOMEN**

### **Objectives**

At the end of this chapter the reader should understand:

- The anatomical and pathophysiological changes associated with pregnancy
- How these changes impact on management of the pregnant trauma patient
- The incidence and social toll of domestic violence
- How to screen potential victims of domestic violence

### **Introduction**

In this egalitarian age, one would be perfectly justified in asking why a chapter on "Trauma in Women" is necessary. The answer is both simple and tragic; simple because trauma in pregnancy is common (6-8%) and the most frequent cause of non-obstetric death in this group of patients. The anatomical and physiological (often referred to as "pathophysiological") changes induced by pregnancy influence the patterns of injuries seen, the response of the patient and foetus to those injuries, and any therapeutic interventions. Tragic, because violence directed against women is remarkably common, under-diagnosed, and without effective intervention, is often fatal. This chapter provides an overview of the management of these two very particular situations.

**The trauma team is dealing with two patients – the mother AND the foetus**

**Optimal resuscitation of the foetus is obtained by optimal resuscitation of the mother**

**The SEQUENCE of care is exactly the same as with all other trauma patients**

### **Anatomical implications on the mechanism of injury**

For the first 12 weeks of pregnancy, the uterus remains safely contained within the bony pelvis. The fundus is approximately at the level of the umbilicus at 20 weeks of gestation, reaching the costal margins at around 34 to 36 weeks. The descent of the foetal head into the pelvis, as the woman approaches term, may be associated with a slight decrease in fundal height. Engagement of the foetal head means that maternal pelvic fractures can be associated with serious intracranial injury to the foetus, with or without foetal skull fracture.

During the second trimester, the uterus ascends out of the pelvis, but the thick, muscular uterine walls, the amniotic fluid, and the relatively small size of the foetus, all help to diminish the direct consequences of trauma. As the third trimester progresses, the uterine wall thins affording progressively less mechanical protection to the foetus. At the same time, the bowel

is pushed into the thorax, making the uterus the most vulnerable intraabdominal organ. It should be pointed out that the uterus itself has elastic properties that are not shared by the placenta. Thus, shear forces applied to the utero-placental interface can result in cleavage. This explains the incidence of placental abruption (*abruptio placentae*) in otherwise minor trauma. As will be discussed below, this possibility must always be considered in the management of the pregnant patient.

The uterus and foetus are relatively well protected during the first and second trimesters of pregnancy.

In blunt trauma, compression, shearing, and deceleration can result in injury to the foetus. Use of seatbelts can influence the pattern of lesions seen after vehicular trauma. When only lap belts are used, the extreme forward flexion may lead to uterine compression, and raises the possibility of rupture and/or placental abruption. If the belt is located too high, it can cause direct injury to the uterus. Shoulder harnesses are associated with improved safety, presumably by decreasing the chances of the victim being ejected from the vehicle, by reducing forward flexion, and by increasing the surface over which forces are dissipated.

Pregnant women should be instructed on the proper use of three-point restraints. The lap belt should be worn under the abdomen, running relatively tightly over the iliac spines. The shoulder harness should be displaced to the side of the uterus, and continue upwards between the breasts, over the midline of the clavicle.

**In the event of a motor vehicle accident, foetal mortality is not increased if three-point harnesses are correctly used, but is almost 3 times higher if the victim is unrestrained.**

Penetrating injuries in late pregnancy can result in complex combinations of gastrointestinal involvement because of displacement of viscera. On the other hand, the uterus is quite effective in protecting the mother, both in terms of its size (increasing the probability that it will be the "target"), as well as for its capacity to absorb energy. Maternal outcome, therefore, tends to be favourable after penetrating injury. The foetus, on the other hand, does not fare as well. For example, gunshot wounds involving the uterus are associated with a 7 - 10% maternal mortality, but with a foetal death rate of around 70%.

## Pre-hospital care

This follows the procedures described in Chapter 1. Ideally, local triage protocols should ensure that pregnant trauma victims are transported to centres where obstetric and, for near-term pregnancies, neonatal facilities are available. As will be seen later, the ability to adequately monitor the foetus is crucial for decision-making during clinical management. It is also important that the pre-hospital team warn the receiving hospital so that appropriate obstetric personnel can be available when the patient arrives.

### **Primary survey & resuscitation**

Although the system described in Chapter 1 requires some adaptations to take account of the anatomical and physiological changes induced by pregnancy, the priorities remain the same.

#### *Airway and cervical spine control*

Upper airway oedema due to capillary engorgement is normal in the late stages of pregnancy along with changes in the distribution of fat around the face and enlargement of the breast tissue. Vascular fragility in the mucosa of the upper airway is almost universally present, increasing the risk of haemorrhage. Compromise of the upper airway by inhalation of gastric contents is a constant threat in pregnancy, particularly from the second trimester onwards. This is because of mechanical displacement of the stomach, with alteration of the physiological sphincter effect of the gastro-oesophageal angle. Furthermore, progesterone causes relaxation of the lower oesophageal sphincter and gastric emptying is also slowed by the hormonal changes of later pregnancy.

The features described above result in a greater risk of regurgitation and acid aspiration and early consideration should be given to prevention by securing the airway with a cuffed tracheal tube. However it is well recognised that there is an increased incidence of difficult and failed intubation in this group of patients. A short handled laryngoscope may be used to help overcome the obstruction caused by enlarged breasts along with a smaller diameter tracheal tube (7.0mm) that will be easier and less traumatic to insert in the presence of pharyngeal oedema. Alternative airway devices must also be available to allow the safe management of the airway in cases of difficult or failed intubation, for example a supraglottic device. Skilled assistance should always be sought as early as possible.

All pregnant women must be considered to have a full stomach. Cricoid pressure must be part of the routine during intubation. Occasionally, it may make tracheal intubation more difficult and the decision must be made whether to release it to try and improve the view at laryngoscopy (Chapter 3). Although the use of anti-acid and pro-gastrokinetic medication

may decrease the risk of acid aspiration, there is unlikely to be time for them to be given and be effective in those patients requiring intubation in the ED. Pregnancy reduces requirements for anaesthetic and sedative drugs by 20 to 25%. This must be remembered when these drugs are given to facilitate tracheal intubation. Where a cervical spine injury is suspected the patient's head and neck should be immobilised as described in Chapter 1.

#### Breathing and ventilation

Maternal hyperventilation is normal, as a result of an increase in tidal volume and results in a  $P_aCO_2$  value of 4.0-4.5kPa (30-34mm Hg). A compensatory fall in serum bicarbonate maintains pH in the normal range. The presence of the gravid uterus causes a number of significant changes; the functional residual capacity (FRC) is decreased by approximately 20%, in late pregnancy, airway closure occurs during tidal breathing in the supine position in 50% of patients and pulmonary compliance is reduced. The oxygen consumption is increased by up to 50% in late pregnancy. As a result of these changes, the pregnant patient will desaturate and become hypoxic significantly more rapidly than an age-matched, non-pregnant woman. Supplemental oxygen must therefore be provided to ALL pregnant trauma patients.

Because of the reduction in FRC (the reserve from which oxygen is drawn during apnoea), and the increase in oxygen consumption, careful attention must be paid to ensure adequate preoxygenation before the use of hypnotics and neuromuscular blocking drugs to facilitate tracheal intubation. This should be achieved by 3 to 6 minutes of tidal breathing of 100% oxygen via a tight-fitting mask and high flow oxygen. If expired gas composition can be monitored, the aim should be to achieve an end-tidal oxygen concentration ( $FetO_2$ ) of at least 0.85. Alternately, with a cooperative patient or when time is limited, 6 to 8 vital capacity breaths with a tight fitting mask and 100% oxygen will provide an acceptable alternative.

The reduced pulmonary compliance will necessitate the need for higher peak inspiratory pressures during mechanical ventilation. Expert help should be sought early to reduce the risk of pulmonary barotrauma. The adequacy of ventilation must be judged by analysis of an arterial blood sample, remembering what is normal in a pregnant patient. The finding of a "normal"  $P_aCO_2$  level implies hypercarbia and impaired ventilation.

#### Circulation and control of haemorrhage

Maternal cardiovascular adaptation to pregnancy is extensive, and complicates management of the victim of trauma. Teleologically, these changes all aim at allowing the mother to

provide metabolic support for her foetus and tolerate the stress of childbirth. These changes must be borne in mind when caring for a pregnant trauma victim.

Heart rate increases gradually throughout pregnancy, reaching a maximum during the third trimester, at 10 to 15 beats per minute over baseline. Blood pressure decreases by 5 to 15 mmHg during the second trimester, with a return to normal values by term. The blood volume increases by 40 to 50%, with the increase in plasma volume exceeding that of red cell mass. This causes the "physiologic anaemia of pregnancy" and in late pregnancy a haematocrit of 30 to 35% is normal. This results in a relative decrease in the loss of red cell mass as a result of haemorrhage during childbirth.

Cardiac output increases throughout pregnancy, reaching 20 to 30% over non-pregnant values soon after the end of the first trimester and up to 40% at the beginning of the third trimester, by which time 25% goes to the uterus. By the third trimester, the uterus itself may reduce venous return and cardiac output by mechanically compressing the inferior vena cava and the aorta when a pregnant woman assumes the supine position. This is known as aortocaval compression. In most women it is not accompanied by measurable changes in haemodynamic parameters. However, in 10% of women, lying flat causes the "supine hypotensive syndrome", with falls in cardiac output of sufficient magnitude to mimic a low output state with symptoms of dizziness or nausea and signs of foetal distress. Such a situation will lead to significant impairment of placental circulation and foetal distress when exacerbated by the presence of hypovolaemia ..

Normally the placental vasculature is maximally dilated and there is no significant autoregulatory reserve. This means that falls in maternal blood pressure are inevitably associated with placental vasoconstriction and dramatic falls in foetal oxygenation. Furthermore, uterine vessels are exquisitely sensitive to catecholamines, resulting in profound vasoconstriction. Consequently physiological compensation for hypovolaemia in the mother will gravely compromise the foetus.

The circulatory changes in the pregnant state allow the mother to adapt to the loss of 1000-1500 ml of blood volume with only minimal signs, but for the reasons described above will be accompanied by signs of foetal distress. Therefore, whenever possible, foetal monitoring should be instituted early, because it will help identify the presence of maternal hypovolaemia. The presence of normal foetal parameters implies minimal maternal blood loss or near-adequate resuscitation of the mother. Conversely the presence of maternal hypotension, not due to aortocaval compression is associated with 80% foetal mortality.

Aggressive volume resuscitation must be instituted early in these cases using the principles described in Chapter 5. In Rhesus negative women with obvious uterine bleeding, an early prophylactic dose of anti-D immunoglobulin (Rhogam®) should be given (see below).

As trauma patients are cared for supine, there is a high risk of aortocaval compression in women in the third trimester and efforts must be made to provide uterine displacement, usually to the left. In the pregnant trauma patient this is perhaps "easier said than done", but can be accomplished initially by tilting the whole spine board 5 to 10° to the left *en bloc*; certain trolleys also allow this type of movement. It should also be remembered that the uterus can be manually displaced in situations of extreme urgency. Wedges or a pillow under the right hip and nursing the patient in the left lateral position should be used only after spinal injury has been excluded.

In addition to the usual causes of shock, uterine rupture must be considered. Symptoms and signs suggestive of this condition are abdominal tenderness, guarding, rigidity, an abnormal foetal lie, and the easy palpation of foetal parts. On X-ray, the finding of an abnormal foetal position or of extended foetal parts is also highly suggestive of uterine rupture. Foetal survival in these cases is rare.

The indications and relative advantages of ultrasound and CT scanning are the same in the pregnant patient as in other patients (Chapter 6). If diagnostic peritoneal lavage (DPL) is indicated, an open, supraumbilical approach should be used. Problems with dependent positioning of the catheter, as well as with obtaining adequate fluid return, are common.

#### **Disability of the CNS (neurological assessment)**

It should be remembered that the trauma may have been secondary to an eclamptic seizure and that a reduced level of consciousness could be confused with the presence of a head injury. Dipstick analysis of urine should be performed as soon as a specimen can be obtained. The concomitant presence of proteinuria, hypertension and peripheral oedema usually allow the diagnosis to be made, but skilled obstetric help will be indispensable under these circumstances.

**There is no specific test for eclampsia. Its diagnosis depends on the clinician considering its possibility**

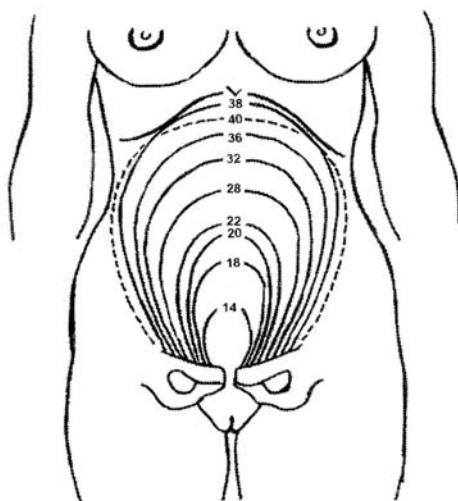
### Exposure and environmental control

As with all trauma patients, a full examination must be carried out taking care to avoid hypothermia. Bleeding from the urogenital tract may first be seen at this point.

### **SECONDARY SURVEY**

After initial evaluation and resuscitation, the systematic secondary survey is undertaken. Careful examination of the patient's abdomen is essential. Signs of peritoneal irritation may be difficult to elicit because of thinning of the abdominal wall musculature, and stretching of the peritoneal membrane itself. Areas of tenderness and guarding are important to note. Foetal movements may be palpated, as well as the presence or absence of uterine contractions. As a rough estimate the gestational age in weeks is approximately equal to the height (in cm) above the pubic symphysis (Fig. 11.1).

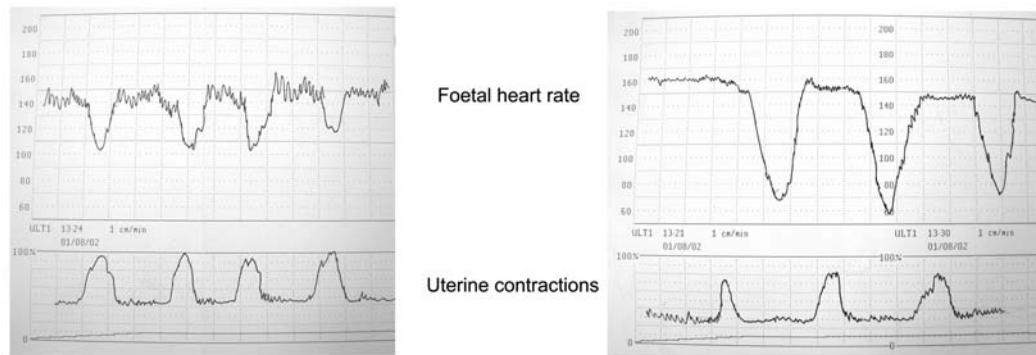
**Fig. 11.1: Diagram of fundal height with gestation in weeks.**



Obstetric consultation must also be obtained by this phase to allow assessment of the foetus and perform any vaginal examination. In addition to assessment of foetal position, the presence of vaginal bleeding, amniotic fluid, and any dilatation and/or effacement of the cervix are important elements to evaluate. Vaginal bleeding is present in up to 70% of cases of placental abruption. Additional signs are uterine tenderness, contractions, and uterine irritability (contractions induced by palpation of the uterus). It occurs in 2 to 4% of women

with minor trauma, and up to 50% with major abdominal trauma. Assessment of foetal heart sounds should be an early priority. This can be done using Doppler as early as 10 weeks of gestation. Classic cardiotocographic (CTG) monitoring can be instituted between 20 - 24 weeks gestation. In effect, the foetal heart rate gives valuable information as to foetal well-being. The normal foetal heart rate is 120 to 160 beats per minute. A tachycardia or bradycardia, recurrent decelerations, lack of accelerations, or the loss of beat-to-beat heart rate variability should all alert the trauma team to the possibility of foetal distress (Fig. 11.2).

**Fig. 11.2: Cardiotocograph showing normal response of foetus to contractions and foetal distress**



**Type 1 dips, little significance; foetal heart rate decelerations mirror contractions.  
Good beat-to-beat variation in heart rate.**

**Type 2 dips or late decelerations; foetal bradycardia well after the contraction.  
These are serious and indicate foetal hypoxia**

Foetal viability is defined as the age at which 50% survival can be expected. In all but the most specialised centres, this is currently felt to be between 25 and 26 weeks gestational age, corresponding to a foetal weight of about 750 grams. The date of the last menstrual period and the expected date of delivery are therefore very important pieces of information. In addition, a history of any complications in the current or past pregnancies is important to elicit.

All pregnant trauma victims with a viable foetus and a gestational age of  $\geq 24$  weeks should receive continuous CTG monitoring, even without evidence of direct abdominal injury. This

type of evaluation has the highest sensitivity and specificity for the detection of the onset of premature labour, placental abruption, and foetal asphyxia. There is some controversy as to the optimal duration of electronic foetal monitoring in the pregnant patient after trauma. If the initial assessment reveals more than 6 contractions per hour, abdominal or uterine tenderness, ruptured membranes, hypotension or vaginal bleeding, CTG should be continued for at least 24 hours. In the absence of any of these signs or symptoms, and with normal abdominal ultrasound and initial CTG, an otherwise normal patient can be discharged after 4 hours. On the other hand, signs of placental abruption and foetal distress in a patient with a viable foetus should prompt immediate operative delivery.

### **Foeto-maternal haemorrhage (FMH)**

This occurs when there is blood loss from the foeto-maternal unit. Apart from the risk of foetal hypovolaemia and anaemia, in 8-30% of pregnant trauma cases, foetal red cells may enter the maternal circulation. This results in the risk of a Rh-negative mother being sensitised by her Rh-positive foetus. There is no correlation between the severity of the trauma or the gestational age and the extent of FMH resulting in sensitisation. As little as 0.1 ml of foetal blood will sensitize up to 70% of Rh-negative mothers. To avoid this complication, a prophylactic dose (300 µg) of anti-D immunoglobulin (Rhogam®) should be administered to all pregnant, Rh-negative women, with the possible exception of those with trauma limited to sites far from the abdomen. This should be given within 72 hours of the injury. One dose is sufficient to neutralise 30 ml of foetal blood. If larger quantities are suspected of being lost to the maternal circulation, a Kleihauer-Betke (KB) test may be performed after 24h, to quantitatively assess the foetal haemorrhage. Repeat doses of Rhogam are rarely necessary because it is estimated that less than 10% of cases of FMH involve more than 30 ml of foetal blood.

Despite the potential of X-rays to induce foetal malformations, neoplasia, and intrauterine growth retardation, appropriate radiological studies should not be avoided in pregnant victims of trauma. For sites away from the abdomen, abdominal shielding is useful. It is felt that foetal exposures to doses of less than 5 to 10 RAD are probably not associated with adverse effects. Approximate doses of absorbed radiation with an AP chest x-ray are 0.005 RAD, a pelvic x-ray 0.4 RAD, a head CT examination 0.05 RAD, an upper abdominal CT < 3 RAD, and a lower abdominal CT 3 to 9 RAD.

### **Peri-mortem caesarean section**

The role of peri-mortem caesarian section is controversial. Data in trauma are singularly lacking. In the context of "medical" cardiac arrests, it is relatively well documented that if

cardiopulmonary resuscitation (CPR) is not effective after 5 minutes, delivery of the foetus if over 25 weeks may improve maternal haemodynamics sufficiently to allow recovery of a spontaneous circulation. In the victim of trauma suffering cardiac arrest, on the other hand, it is highly likely that the foetus has also suffered from a prolonged period of severe hypoperfusion. A decision to perform a peri-mortem caesarian section must be made in close collaboration with the obstetrician, and be made within 5 minutes of the cardiac arrest.

### **Domestic violence**

The incidence of domestic violence is enormous. A Swiss survey of 1500 women showed an incidence of 20%, and a similar study in the UK concluded that 25% of women were victims of some form of domestic violence. Other data reveal that up to one third of women are beaten, coerced into having sexual intercourse or otherwise abused in their lifetimes.

The Council of Europe has declared that domestic violence is the major cause of death and disability in women between 16 and 44 years of age, with a total greater than that of cancer and traffic accidents. In Russia, 14,000 women were killed by their partners in 1999 and 70% of female murder victims are killed by partners or ex-partners. In the UK each week, a partner or former partner kills two women. The police receive one domestic violence call each minute, but these figures are even more shocking when one considers that only about 35% of cases of this type are ever reported to the police.

**The incidence of foetal morbidity due to this violence is greater than that due to diabetes and pre-eclampsia/ eclampsia combined.**

### *Diagnosis and management*

Given the incidence of this problem, trauma team members are sure to encounter it relatively frequently in their day-to-day practice. But because of the shame and guilt involved, under-diagnosis is the rule rather than the exception. For this reason, a high clinical index of suspicion is necessary. In a manner similar to child abuse, and for the same reasons, several elements should be kept in mind when evaluation a woman with trauma. These may suggest a non-accidental origin of the injuries.

- Injuries that are inconsistent with the stated explanation
- Poor self-esteem, obvious depression, history of attempted suicide
- Frequent office or emergency department visits
- Self-blame for injuries
- A partner who insists on being present during history and physical examination

- Self-abuse
- Substance abuse
- A patient who is disproportionately upset with respect to the injuries and/or the history
- Clothing torn in a manner inconsistent with the history
- Seeking care some time after injury

Irrespective of the actual or suspected cause, the first priority to all women is to identify and treat any immediate life-threatening injuries in the sequence described in Chapter 1.

### **Screening**

One of the most effective techniques to reduce the occurrence of domestic violence is systematic screening of women patients consulting in emergency departments. The following three questions, asked in a non-judgmental manner without the partner being present will detect 65 to 70% of abused women.

- Have you been kicked, hit, punched or otherwise hurt by someone within the past year? If so, by whom?
- Do you feel safe in your current relationship?
- Is there a partner from a previous relationship who is making you feel unsafe now?

Although it is important to identify victims of domestic violence, when they present to the emergency department, this is secondary to their immediate care. Clearly after the patient's injuries have been dealt with, a member of the team should be tasked with screening for evidence of violence and ensuring referral to the appropriate local authorities according to local protocols.

### **SUMMARY**

The anatomical and physiological changes that occur in pregnancy are sufficient to require members of the trauma team to have an understanding of how to adapt the primary survey and resuscitation in this group of patients to maximise the chances for the outcome of both the mother and child after major trauma. There should be early involvement of obstetricians in all pregnant trauma patients and neonatologists or paediatricians where a viable pregnancy is involved. All trauma team members should be skilled at recognising those signs that suggest a woman's injuries are the result of domestic violence and be aware of the local policies to deal with this situation.



## **Web sites**

Newton E (2003) Trauma and Pregnancy

<http://www.emedicine.com/med/topic3268.htm#section~introduction> (accessed April 2006)

Desjardins G Management of the Injured Pregnant Patient

<http://www.trauma.org/resus/pregnancytrauma.html> (accessed April 2006)

Domestic violence against women and girls (2000) Innocenti Digest, UNICEF;

<http://www.unicef-icdc.org/publications/pdf/digest6e.pdf>

European policy action centre on violence against women

<http://www.womenlobby.org>

## **Further reading**

1. Grossman N. Blunt Trauma in Pregnancy. *American Family Physician* 2004;70(7):1303
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3. Curet M, Schermer C, Demarest G *et al.* Predictors of outcome in trauma during pregnancy: identification of patients who can be monitored for less than 6 hours. *J Trauma* 2000;49(1):18
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# **CHAPTER 12**

## **MEDICAL PROBLEMS IN TRAUMA PATIENTS**

### **Objectives**

At the end of this chapter the reader should understand:

- The incidence and significance of medical problems in trauma patients
- What potential medical problems may pre-exist
- The pathophysiological aspects of aging
- How medical problems and aging influence clinical presentation and management
- Why you should not detract from the ABCDE system of trauma management

### **Introduction**

Most trauma victims are young, but the consequences of injury are evident at all ages with at least two survivors with serious permanent disabilities for every person killed. In those over the age of 65 years the death rate is approximately double that for younger ages. The cause of this increase in morbidity and mortality is twofold. Firstly, lack of physiological reserve culminating in changes in structure and function, and secondly, the increased numbers of pre-existing medical conditions, some of which may be subclinical.

A variety of factors affect the survival of trauma patients (Box 12.1). However, host factors including age and sex have also been reported as independent risk factors that adversely influence the outcome. In addition, the presence and number of co-morbid or pre-existing medical conditions (PEMC) increase mortality and morbidity after trauma. Only recently has attention been focused on the incidence, type and effect of these conditions on the outcome of patients following major trauma. Pre-morbid conditions are found in all age groups, and as expected the frequency and number increases with increasing age. In the USA the incidence of pre-morbid problems ranges from 4.3 to 16 %, whereas in the UK such conditions occur in 39% of major trauma patients.

<b>Box 12.1: Four key determinants of survival following major trauma</b>
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- |   |
|---|
| <ul style="list-style-type: none"><li>• Pre-injury health</li></ul> |
|---|

- Magnitude of injury
- Time to definitive care
- Quality of care

Although outcome following major trauma is influenced by the factors listed in Box 12.1, mortality increases with the number and type of pre-existing condition from 5% with none to over 12% with two or more. The commonest pre-existing conditions affect the cardiovascular and respiratory systems, and psychiatric problems. Death however is more likely to occur in patients with pre-existing disease affecting the heart (3 fold), kidney (6 fold) and liver (6 fold).

Unfortunately the significance and impact of co-existing medical problems are all too frequently overlooked. It is therefore essential to have a high index of suspicion and consider the possibility of a PEMC during the primary survey if either a patient's clinical features appear inconsistent or the response to resuscitation is inappropriate. Further clues will be found in the AMPLE history, and also from the results of relevant investigations.

**Remember ABCDE, and if the patient is not responding as expected,  
think PEMC.**

### **Clinical assessment and immediate management.**

As all stages of resuscitation may be influenced by both medical problems and aging they should be considered using the ABCDE system. The first section will focus on a selection of medical problems whilst the second will examine the pathophysiology of aging with special reference to assessment and resuscitation.

### **Primary survey and resuscitation**

#### Airway with cervical spine control

A variety of conditions compromise airway integrity and increase risk of injury to the cervical spine in many different ways:

1. Increased risk of aspiration. For example, a pseudobulbar palsy is due to either a stroke or disseminated sclerosis. A cerebrovascular accident (stroke) can also cause bulbar muscle weakness as can motor neurone disease and myasthenia gravis.
2. Mechanical impairment to airway management. Both macroglossia, associated with amyloid and acromegaly and the constricted, small mouth of systemic sclerosis may cause problems with insertion of airway adjuncts and tracheal intubation.

Temperomandibular joint pathology (e.g. rheumatoid disease) may make it impossible to open the mouth wide enough to allow intubation.

3. The cervical spine is often affected in inflammatory conditions, such as rheumatoid disease and ankylosing spondylitis. The changes in patients with rheumatoid disease include pain, stiffness, subluxation and hence the potential for instability and cervical cord injury. Ankylosing spondylitis is another inflammatory polyarthropathy that classically produces a rigid spine that is very susceptible to injury. Thus only a trivial amount of force is required to damage the cervical spine and cord.
4. Patients with Down's syndrome are another group of patients who have an increased risk of c-spine injury, especially atlanto-axial subluxation.

#### Breathing and ventilation

Respiratory diseases are common, thus it is not surprising that 21% of trauma victims have co-existent pulmonary pathology. Many respiratory conditions may not be apparent on initial assessment, especially when the patient has sustained a chest injury. This can cause diagnostic difficulties but clues may be gained from the initial assessment, past medical history, treatment response, arterial blood gases and chest X-ray. The common respiratory problems that may be encountered and cause diagnostic difficulty are:

1. Chronic Obstructive Pulmonary Disease (COPD). This causes pulmonary dysfunction and subsequent respiratory failure leading to diagnostic confusion, especially in patients who are cyanosed. Another problem is that some of these patients will have developed a hypoxemic rather than hypercapnoeic ventilatory drive. Hypoxaemia, especially in the major trauma situation, is a major cause of morbidity and mortality and correction of this takes precedence over concerns of hypercapnia. Therefore these patients require a high inspired oxygen concentration (at least 85%) initially. It can subsequently be titrated according to arterial blood gas results, and the need for assisted ventilation

**The presence of chronic pulmonary disease should be regarded as an early indication for ventilation.**

2. Bronchospasm has many causes including inhalation of noxious compounds, asthma, COPD and pulmonary oedema. It is important to remember that severe bronchospasm can cause hypotension (impaired venous return), and that an acute asthmatic has an increased risk of developing a pneumothorax. High flow oxygen, nebulised/intravenous bronchodilators along with intravenous steroids should be given according to the clinical situation. An AMPEL history will help in making the correct diagnosis

3. Pulmonary oedema is a non-specific feature associated with inhalation of noxious substances, pulmonary parenchymal injury, acute respiratory distress syndrome, neurological injury and myocardial trauma. In the western world, however, it is also a common sequel to ischaemic heart disease (left ventricular dysfunction) and to a lesser extent mitral or aortic valve pathology. If pulmonary oedema is present, either clinically or radiologically, when the patient arrives in the Emergency Department (providing there is neither inhalation injury nor delayed transfer) then it is likely to be due to pre-existing cardiac disease. Such a patient will benefit from early ventilatory support, either using non-invasive techniques or tracheal intubation with fluids and inotropes guided by invasive haemodynamic monitoring. Do not treat these patients with diuretics or vasodilators without invasive monitoring as these drugs can both cause and exacerbate tissue hypoxaemia. Early expert help from those with training in critical care is essential in the management of these patients.
4. A pleural effusion is a manifestation of many medical conditions, including left ventricular failure, pleuro-pulmonary malignancy, and connective tissue diseases. Irrespective of the cause, a chest drain is still the correct management of choice. This will improve ventilation and perfusion, and will also help to exclude coexistent trauma.
5. Pulmonary embolism. After skeletal injury, especially multiple fractures, marrow fat can enter the venous circulation and cause pulmonary "fat" emboli. Clinical features include tachypnoea, tachycardia, a petechial rash, and also a spectrum of neurological signs. The latter are due to hypoxaemia, and humoral and cellular factors that are released from bone as well as the fat globules that traverse the pulmonary capillaries by moulding their shape and hence gain access to the systemic circulation

In contrast, pulmonary embolism (PE) due to the presence of deep vein thrombosis occurs later, usually after a period of immobility during recovery. However, it may be seen in those with a predisposition (e.g. antithrombin III deficiency) after minor injury or in vehicle drivers following a long journey. In the context of trauma, it is likely that a massive PE will present as pulseless electrical activity (PEA). As thrombolysis is usually precluded due to multiple injuries, the management of these patients follows the principles described in Chapter 1.

A list of multisystem diseases, in which there is co-existent pulmonary problems, is given in Box 12.2.

### Circulation and haemorrhage control

Hypotension in major trauma patients is due to blood loss until proved otherwise (Chapter 5). If the patient fails to respond to fluid resuscitation then continued blood loss possibly into a body cavity or soft tissues is the likely cause. However, if this is not evident then it is important to consider other medical conditions (Box 12.3). This will require, in addition to a full reassessment, a 12 lead electrocardiogram (ECG), full blood count, electrolytes, arterial blood gases and a chest X-ray.

#### **Box 12.3: Medical causes of hypotension**

- Blood loss
- Cardiac tamponade
- Left/right ventricular failure
- Arrhythmias
- Pulmonary embolism
- Tension pneumothorax
- Septicaemia
- Anaphylaxis
- Vasodilators
- Adrenal insufficiency
- Autonomic dysfunction
- Spinal cord injury
- Uraemia
- Toxins

Occult blood loss in the gastro-intestinal tract occurs secondary to trauma. Blood loss may also occur as a result of pre-existing pathology such as peptic ulcer disease and gastroduodenitis or the development of stress ulceration, for example Curling's ulcers after extensive burns or Cushing's ulcers after central nervous system trauma (Box 12.4). However, such "stress" induced gastro-intestinal bleeding is unlikely to influence the initial resuscitation as it usually takes hours or days to develop and is frequently occult.

#### **Box 12.4: Risk factors for stress induced gastroduodenal damage**

- Shock
- Sepsis

- Severe central neurological injury
- Steroids
- Ventilation for >24 hours
- Coagulopathy

The presence of distended internal jugular veins is a non-specific sign that can be difficult to elicit in the trauma patient. Although obstruction to venous return is the immediate concern, other causes include right ventricular failure (from whatever cause), cardiac tamponade and acute severe bronchospasm. The presence of pulseless electrical activity (PEA) will help in this differential diagnosis as will echocardiography.

During initial assessment it is unusual to diagnose septic shock in the trauma patient unless there has been a prolonged delay in either extrication or transfer. Although these patients initially have hypotension they have warm, well-perfused peripheries. This is a useful physical sign in the differential diagnosis of shock.

Hypotension is an important manifestation of adrenal failure and to a lesser extent pituitary insufficiency. Primary adrenocortical failure is uncommon. In contrast, many patients use chronic steroid treatment and hence the release of endogenous release corticosteroids is suppressed. Even if the steroids have been withdrawn gradually, adrenal function may still be suboptimal, and a stress response blunted. Thus any acute stress, for example trauma, can precipitate an Addisonian crisis. The resultant hypotension may be either transiently responsive or unresponsive to treatment, and either confusion or coma can co-exist mimicking the features of hypovolaemic shock. The classic electrolyte changes (hyponatraemia, hyperkalaemia, uraemia) are not always present. Intravenous hydrocortisone (100mg) should be given immediately once the diagnosis is suspected, followed by 50mg twice daily until it is convenient to do a short synacthen test. It is important to remember that the dose of steroids should be increased (often doubled) in acutely ill patient on chronic steroid treatment to help with the stress response

Ischaemic heart disease is one of the most prevalent diseases in the western world and it can be exacerbated by blood loss, hypoxaemia, hypovolaemia or hypotension. If the patient is unconscious, a myocardial infarction may not be apparent. The presence of an arrhythmia may be the only clue to myocardial damage that may have either precipitated or exacerbated the traumatic insult. Thus continuous ECG monitoring is essential, but it must be remembered that using a single lead as is often the case in the resuscitation room, is not guaranteed to show acute changes associated with myocardial infarction. Whilst a series of

12 lead ECGs will be useful in the diagnosis of an arrhythmia they will not facilitate differentiation of myocardial infarction and myocardial trauma, especially contusion. Under these circumstances clarification can be sought by the patient's age, mechanism of injury and previous medical history. Markers of myocardial damage, the cardio-specific CPK-MB isoenzyme and troponin I /T, are useful and an echocardiogram is essential.

Arrhythmias may occur for a number of reasons including myocardial damage, hypoxaemia, electrolyte disturbance, increased intracranial pressure and coexistent drug therapy. Arrhythmias can also cause hypotension, cardiac failure, hypoxaemia and loss of consciousness. Tachyarrhythmias should only be assumed to be medical in origin when all possible trauma related causes have been ruled out. Drug treatment may be required in these cases, when there is no evidence of hypotension, cardiac failure and impaired consciousness. It is important to remember that drugs used to treat arrhythmias can cause arrhythmias and depress myocardial function. Correction of hypovolaemia can restore sinus rhythm, in particular when the arrhythmia is atrial fibrillation. In the presence of cardiac failure, hypotension, and impaired consciousness, a tachyarrhythmia should be treated by cardioversion in accordance with the current ERC guidelines.

Bradyarrhythmias are rare in the trauma patient. The causes include the "physiological" response in the young fit patient/athlete, and "pathological" in the trauma patient with marked hypovolaemic shock, neurogenic shock, raised intracranial pressure, myocardial injury, medications and pre-existing conditions. Correcting the underlying condition is often sufficient, but the patient may need atropine, chronotropes and pacing according to ERC guidelines.

A particularly difficult problem is the trauma patient who has ischaemic heart disease and hypotension. Following major trauma the commonest cause of hypotension is blood loss, but this not always the entire answer. It may be the product of blood loss and cardiac dysfunction including an arrhythmia and cardiac failure. Under these circumstances it is essential that fluid replacement is titrated accurately, guided by invasive monitoring. Furthermore, loss of consciousness may be independent of the arrhythmia. Therefore, if the patient fails to respond to a fluid challenge then cardioversion is recommended as the arrhythmia can only exacerbate the clinical situation.

Valvular heart disease, particularly calcific aortic stenosis, is on the increase in the elderly population. A narrow pulse pressure may be misdiagnosed as a sign of hypovolaemia, but

other features are usually absent. In this situation excessive fluid resuscitation can result in left ventricular failure. Cardiac decompensation may also occur because of ischaemic heart disease and myocardial trauma. An early echocardiogram is essential providing details of valvular pathology and left ventricular function. Early referral to a cardiologist is recommended.

Patients with valvular disease should be given prophylactic antibiotics according to local protocols, as soon as is convenient providing there are no contra-indications. Intravenous amoxycillin 1 gm and gentamicin 120 mg followed by a further dose of amoxycillin after 12 hours is widely used. Alternatively, local protocols should be followed. Patients with a metal prosthetic valve are likely to be anticoagulated with warfarin. Where there is active haemorrhage that cannot be controlled by direct measures, an assessment of the patient's clotting should be made (using the INR) and fresh frozen plasma (FFP) will be required urgently. Vitamin K should be reserved for immediately life-threatening haemorrhage or for an INR >6. Subsequently, the patient can be started on heparin to ensure an APTT (activated partial thromboplastin time) test to control ratio of two.

Cardiac pacemakers can profoundly influence the clinical picture following major trauma. Potential problems with pacemakers are listed in Box 12.5.

**Box 12.5: Potential pacemaker problems**

Damage to	pacemaker wire
Pacemaker failure	
Inappropriate function	
Mask myocardial damage/bradycardia/tachycardia	
Inappropriate response to	shock fluid resuscitation (especially if fixed rate function)

It is always important to remember that patients may have either inhaled or ingested substances that can have a profound effect on their physiological state (see below). A full medical history and an account from witnesses can be invaluable.

Hypertension is always a concern. The history and physical examination are important and will help differentiate between acute and chronic hypertension. Trauma patients presenting with acute hypertension should be assessed for treatable causes; pain, raised intracranial

pressure, pre-eclampsia and for signs of drug overdose (see below). Only when these causes have been either excluded or adequately treated should rare conditions be considered. Persistent or variable hypertension can indicate the presence of phaeochromocytoma and although rare, it does present problems for the unwary. Acute hypertension needs careful controlled reduction and the patient will require invasive monitoring and intravenous alpha and beta-blockers.

Chronic anaemia is a common condition (Box 12.6). Information as to the presence and type of anaemia may not be available from the initial haemoglobin estimation and a blood film should always be requested to provide further clues. Therefore patients should be transfused according to their clinical features rather than their haemoglobin concentration/packed cell volume. Although the immediate management takes priority, the patient's chronic problem should not be ignored. Transfusion may prevent further investigation initially so the patient should be referred to an appropriate specialist.

**Box 12.6: Chronic Anaemia**

Menstruation	
Diet	
Chronic renal failure	
Chronic liver disease	
Chronic intestinal	Inflammatory bowel disease
Multi-system disease	Rheumatoid disease Ankylosing spondylitis Systemic lupus erythematosus
Haemolytic anaemia	Intrinsic red cell disorders - membrane disorder - hereditary spherocytosis/elliptocytosis - enzyme deficiency - G 6 PD/Pyruvate kinase - haemoglobinopathy - Sickle cell/Thalassaemia Extrinsic red cell disorders - auto-immune - non-immune
Surgical	Post gastrectomy Ileal resection

Haemoglobinopathies, in particular sickle-cell disease cause acute pain that may reflect a sickle crisis. This can cause and complicate trauma, and in particular, mimic intraperitoneal

injury. Tenderness is common and abdominal imaging (ultrasound or CT scan) is recommended. Mortality from sickle cell disease is high, therefore liaise early with a specialist.

Coagulopathy, either a bleeding diathesis or a thrombophilia, can profoundly influence the patient's response to both trauma and resuscitation. If a history is not available and clinical features, in particular chronic liver disease, are not obvious, then this problem will only come to light during resuscitation. The potential causes are listed in Box 12.7. If there is any suspicion of coagulopathy check both the activated partial thromboplastin and prothrombin times. These should be corrected appropriately, according to the clinical context, after discussion with a haematologist.

**Box 12.7: Coagulation disorders**

Clotting deficiencies	Congenital Haemophilia Von Willebrand's disease Anticoagulant therapy -Warfarin Disseminated Intravascular Coagulation -sepsis -severe trauma -fat emboli
Platelet defects	Thrombocytopenia (especially if $<20 \times 10^9/l$ )
Platelet dysfunction	Von Willebrand's disease drugs eg:- aspirin, dipyridamole uraemia diabetes mellitus myeloproliferative disease paraproteinaemias

Pericardial effusion. There are many causes of a pericardial effusion including left ventricular failure, uraemia, and rheumatoid disease.. If there is clinical evidence of a pericardial effusion then pericardiocentesis will improve cardiac function and exclude associated pericardial/myocardial trauma. This procedure should be guided by echocardiography.

Cardiac transplants are becoming increasingly common. The type of transplant will govern whether the myocardium is able to respond either directly to neural, sympathetic stimulation or indirectly to circulating catecholamines.

*Disability of the CNS (neurological assessment)*

Approximately 19% of major trauma patients will have a pre-existing neurological condition, and 30% underlying psychiatric problems. Between 55-72% of patients have positive toxicology screens, comprising alcohol and drugs (35%), drugs (45%) and alcohol alone (20%). With the increasing numbers of aged patients there is a coexisting increase in cerebrovascular disease. In contrast, there is one easily reversible cause of neurological symptoms and signs – hypoglycaemia. Therefore, it is essential to check the plasma glucose, as hypoglycaemia is easy to treat, but potentially lethal and easy to miss. Clues to many neurological problems will be found when assessing airway, breathing and circulation. Fits or coma are usually ascribed to intracerebral injury, however in the presence of a normal CT scan other conditions need to be considered (Box 12.8). Hyponatraemia and hypocalcaemia can follow rapid volume expansion, particularly if large volumes of 5% dextrose have been given in error. Hypernatraemia is often due to dehydration or diabetes insipidus and usually occurs after several days.

Many of the chronic neurological diseases especially stroke and demyelination have lateralising signs. These may mimic or mask occult intracerebral trauma. If no history is available then the presence of these physical signs necessitates a CT scan. It is best to exclude pathology rather than to ignore it.

Autonomic dysfunction not only masks the patient's response to trauma and fluid resuscitation but also mimics spinal cord injury. With the exception of diabetes mellitus, medical causes of autonomic dysfunction are rare. Therefore, treat the patient as though they have an acute spinal injury. Invasive monitoring is essential to guide cautious fluid resuscitation and use of inotropes.

**Box 12.8: Causes of fits/coma**

Drugs and chemicals	alcohol
	lead poisoning
	cocaine
Infections	meningitis

	encephalitis
	intracerebral abscess
	malaria
Vascular	thromboembolic disease
	hypertension
	dysrhythmia
Metabolic	hypO/hypERglycaemia
	uraemia
	electrolyte imbalance
	hypocalcaemia
	anoxia
	inappropriate anti diuretic hormone secretion(SIADH)
	hepatic encephalopathy
Intracerebral	tumours
	haematomas

Drugs are included under "D" because their major effect is to influence the conscious level - especially following an overdose. In the United States a significant proportion of major trauma victims use drugs, but currently this is less of a problem in Europe, a situation likely to change with increasing use of recreational drugs.

Irrespective of the drug used the clinical manifestations will be related, in particular, to the dose and route of administration. Airway and cervical spine integrity, oxygenation and breathing are usually compromised because of a reduced conscious level and respiratory drive. Circulatory effects not only ensure that monitoring is essential but are a trap for the unwary. Drugs can influence assessment of the circulation and can masquerade as shock or myocardial injury. The spectrum of neurological changes is extensive and reflects either stimulation or depression of central nervous system activity possibly mimicking hypoxaemia and intracerebral injury. Therefore do not ascribe any clinical features directly to a drug until underlying trauma has been either treated or excluded.

Unfortunately drugs can affect all aspects of initial assessment and resuscitation. A comprehensive review of this topic is beyond the scope of this manual, however, in the context of major trauma, after alcohol, the following are some of the most commonly used.

**Cocaine** has a stimulant effect on the central nervous system mediated predominantly by delayed inactivation of adrenaline and noradrenaline. Relevant clinical manifestations are

listed in Box 12.9. Acute overdosage produces ventricular dysrhythmias, anxiety, paranoia and tonic/clonic seizures. High concentrations of cocaine can depress medullary centre activity causing unconsciousness and eventually cardiorespiratory arrest. Thus, cocaine may induce or mask the clinical features of hypoxaemia, shock, myocardial damage and head injury.

**Opioids** are a homogeneous group of natural and synthetic compounds. Their effects include hypoxaemia and carbon dioxide retention due to depression of the respiratory drive. Furthermore hypotension secondary to venodilatation is a common feature. Unreactive pupils and raised intracranial pressure from retained carbon dioxide mediated cerebral vasodilatation, can mimic or exacerbate head injury.

**Amphetamines** cause a variety of clinical effects primarily attributed to stimulation of the central nervous system and peripheral adrenoreceptors. Major manifestations include severe agitation, psychosis, hypertension and an increased risk of intracranial haemorrhage. Rarely acidosis, rhabdomyolysis and acute renal failure occur. Thus amphetamine overdose can manifest as hypoxaemia, head injury and skeletal muscle trauma.

**Ecstasy** is a synthetic amphetamine derivative (3,4-methylenedioxymetamphetamine) that induces a mild stimulant effect combined with euphoria, benevolence and enhanced perception. Its effects appear to be compounded by physical activity and dehydration, typically dancing at "rave" parties. Acute, severe complications include hypoglycaemia, convulsions, collapse, hyperpyrexia (including neuroleptic malignant syndrome), intracerebral haemorrhage, disseminated intravascular coagulation, rhabdomyolysis, hyponatraemia and acute renal failure.

**Box 12.9: Clinical features of cocaine use - relevant to trauma resuscitation.**

Respiratory	Pulmonary oedema (cardiac/non cardiac) Bronchospasm Alveolar haemorrhage "Crack" lung Bronchiolitis obliterans
Cardiovascular	Dysrhythmia Myocardial ischaemia/infarction Biventricular failure Hypertension

	Cardiomyopathy
	Myocarditis
	Endocarditis (I.V. use)
	Aortic dissection
	Deep vein thrombosis/thrombophlebitis
Neurological	Intracerebral haemorrhage
	Cerebral infarction
	Coma
	Convulsions
	Headaches
	C.S.F. rhinorrhoea
Locomotor	Rhabdomyolysis
	Vasculitis
Obstetric	Abruption placenta
	Spontaneous abortion

**Tricyclic antidepressants** act primarily by inhibiting the re-uptake of noradrenaline and 5-hydroxytryptamine, and blocking central and peripheral muscarinic receptors. Most of the side-effects are anticholinergic. However, acute overdose causes respiratory depression, dysrhythmia, hypotension, hypothermia, convulsion, and coma. Therefore these features can mimic shock, and injuries to the myocardium, head and spinal cord.

**Solvent inhalation** is associated with a host of cardiovascular manifestations that are attributed to volatile hydrocarbons sensitising the myocardium to the arrhythmogenic effects of endogenous catecholamines. In contrast, however, habitual solvent use is associated with rhabdomyolysis, renal and hepatic damage, marrow depression, cerebellar damage, neuropsychiatric features and metabolic disturbances. Clues to potential use include age and peri-oral/nasal erythema.

Even when it is known or suspected that the victim is under the influence of drugs, do not deviate from the initial assessment to identify and treat life-threatening injuries. Suspected pathology has to be treated. Do not assume physical signs are due to drugs. Injuries and side effects have to be prioritised for treatment. Relevant antagonists, sedatives and anaesthetics should be used only after liaison with appropriate colleagues.

### Exposure and environmental control

Hypothermia can cause or follow either major trauma or resuscitation. Potential causes should be sought in the secondary survey. Prevention of hypothermia in the resuscitation room is important. Appropriate means to maintain the body temperature include warmed intravenous fluids and overhead heating. The treatment of hypothermia has been described earlier in this book. Remember that hypothermia not attributed to exposure should alert you the presence of occult pathology, in particular sepsis, pancreatitis, hypothyroidism, alcohol and phenothiazine overdose.

### **Investigations**

Arterial blood gas results provide invaluable information in the resuscitation room, but must be interpreted in light of the clinical situation. For example the presence of respiratory alkalosis, if the patient is ventilated, is likely to reflect inappropriate excessive ventilation. In contrast, if the patient is not ventilated then compensatory hyperventilation may occur as a consequence of hypoxaemia, sepsis, pulmonary emboli or anxiety.

Metabolic acidosis, however, especially with an increased anion gap, may reflect hypoperfusion and lactic acid accumulation due to inadequate perfusion. If the patient fails to respond to appropriate treatment then it is important to exclude other potential causes (Box 12.3). Many of these problems may not be apparent on initial assessment. Clues to their presence will be found in the initial assessment, AMPLE history, the response to treatment, arterial blood gases and x-rays.

An electrolyte imbalance is a frequent finding especially in patients taking diuretics. Chronic hypokalaemia is common, but only requires urgent treatment if there is an associated arrhythmia. In contrast, hyperkalaemia is likely to be the most important electrolyte problem in the acute situation. This should not be attributed to a haemolysed sample because patients can have coexisting renal dysfunction, metabolic acidosis in association with shock, diabetic ketoacidosis or even tissue (especially muscular) necrosis. Pre-existing drug treatment is a common cause. This is important particularly in patients who have acute and/or chronic renal failure where under perfusion may exacerbate the situation and warrant early dialysis and intervention by a nephrologist. The relevant clinical features associated with a disturbance of sodium and calcium homeostasis have been described earlier in this chapter.

Always retain samples of blood and urine for toxicology if a drug related problem is suspected.

## **Secondary survey**

### ***AMPLE History***

Many medical problems will be identified in trauma patients by the AMPLE history from the patient, police and paramedical staff. Further information may be sought from hospital records, the patient's relatives and general practitioner, as well as any witnesses. With regards to medical problems relevant information from an AMPLE history should include:

A: An acute allergy is unlikely to be present in a trauma patient unless the reaction either caused the injury or resulted from medical therapy. Many of the consequent clinical features require minimal medical intervention. In contrast, however, anaphylaxis may mimic, mask or co-

exist with shock. Adrenaline (epinephrine) is the essential treatment and given in accordance with the current ERC guidelines.

M: Many patients are taking prescribed drugs and the type and dose may not be known at the time of resuscitation. Beta blockers, calcium channel antagonists, angiotensin converting enzyme (ACE) inhibitors and, to a lesser extent, nitrates are important because they modify the cardiovascular response to both trauma and resuscitation. The use of oral steroid preparations, as described earlier, is common and an Addisonian crisis can be precipitated especially if this drug is omitted. As previously discussed, the use of recreational drugs may also cause and modify the patient's response to trauma.

The link between alcohol consumption and major trauma, in particular road traffic accidents, is well established. Alcohol can influence the presentation, treatment and outcome following trauma. It is important to realise that alcohol can produce a variety of clinical manifestations as can withdrawal from the same. In the presence of chronic alcohol consumption give adequate thiamine intravenously and control withdrawal symptoms with chlordiazepoxide. It is **important to remember that thiamine must be given before intravenous dextrose**. Thus, the possibility of Wernicke's encephalopathy will be avoided in susceptible individuals.

P: Past medical history is extremely important, as this will alert the physician to potential factors that may influence the clinical presentation and response to treatment.

L: Last meal. In the context of medical problems this may explain the cause of hypoglycaemia. Furthermore, the time of last medication is important irrespective of whether the drugs were prescribed or taken as over dosage.

E: Events/Environment. The environment in which the trauma victim was found provides important information as to the potential cause/s of hypothermia. Always consider occult pathology.

#### Physical examination

During the "head to toe" assessment of the patient other medical problems may be identified. Their treatment will depend on the patient's injuries and history, and prioritised accordingly. Regular re-assessment is important as physical signs change, especially if new clinical information is forthcoming. The presence and effect of most medical problems will not become

apparent until the secondary survey. Specific information regarding drugs should be available, and if appropriate use antagonists; but do not forget the potential problems that can result from poly-pharmacy. Many medical conditions affect multiple systems and it is advisable to review the patient as a "whole" - for example the significance of episcleritis may not be immediately apparent until other systems have been assessed. Furthermore it is important to remember that treatment can cause deterioration in the patient's condition, as in the case of enthusiastic fluid resuscitation precipitating biventricular failure.

### **The elderly patient**

The definition of "the elderly" is very variable, but for the purposes of this section, those aged 65 years or more will be used. The elderly population is increasing with falls being the major cause of trauma. They are seriously injured less frequently than the young, and mortality is similar between these two groups but increases markedly with pre-existing medical problems, which of course increase with age. Many important anatomical and physiological changes occur with increasing age. These influence the presentation and management of elderly trauma victims. Some of these changes are summarised in Box 12.11 and described in the next section using the ABCDE system.

### **Airway and cervical spine**

Airway management can be difficult because the frequent lack of teeth prevents a tight seal with a face-mask. Leave well-fitting dentures in place and inform the trauma team leader. In contrast, the lack of teeth combined with mandibular resorption will make tracheal intubation easier. There is also an increased risk of aspiration due to impaired swallowing, poor crico-pharyngeal tone, and delayed gastric emptying. It is essential to consider an alternative site (ear, nose) for pulse oximetry in the cold, shivering, vasoconstricted elderly patient.

Degenerative disease affects both joints and ligaments of the cervical spine and is exacerbated by osteoporosis. As these changes predisposes to instability flexion should be avoided. In contrast, extension can compromise vertebrobasilar blood flow that is often critical in the elderly who have bilateral carotid artery disease and thus rely almost totally on posterior cerebral perfusion of the circle of Willis. Anterior osteophytes predispose to canal stenosis and increase the risk of cord syndromes. If in doubt, always immobilise the c-spine until it can be cleared both clinically and radiologically.

### **Breathing and ventilation**

There are many reasons why the elderly are less likely to tolerate chest wall injury especially with associated contusion, or pneumothorax, or haemothorax. With increasing age the drive to ventilation is impaired with blunting of the normal response to hypoxaemia and hypercarbia. In addition, the chest wall becomes more rigid due to costochondral calcification, spinal shortening, and progressive kyphosis. This reduces rib excursion and increases the risk of injury with minimal force. Hence the elderly are more at risk of pulmonary contusions. Respiratory effort is also impaired because of poor compliance, respiratory muscle fatigue and an inability to sustain ventilatory effort.

### **Box 12.11: Summary of anatomical and physiological changes in the elderly**

#### **GENERAL**

- Limited physiological reserve
- Multiple pathology
- Atypical presentation
- Reduced visceral perception of pain
- Co-morbidities
- Polypharmacy
- Abuse of the elderly patient
- End of life decisions

#### **AIRWAY**

- Edentureless
- Mandibular resorption
- Poor muscle tone
- Temperomandibular pathology

#### **CERVICAL SPINE**

- Degenerative disease, instability, impaired vertebrobasilar blood flow

#### **BREATHING AND VENTILATION**

- Reduced compliance of chest wall and lungs
- Reduced area for gas exchange
- Increased collapse of small airways
- Impaired mucociliary escalator

#### **CIRCULATION**

- Reduced compliance of ventricles
- Rigid aorta (systolic hypertension)
- Reduced sympathetic drive
  - limited compensatory tachycardia
  - poor vasomotor tone

#### **DISABILITY**

- Confusion has multiple causes and does not indicate dementia

## **Exposure**

- Reduced ability to generate heat
- Impaired perception of temperature changes

## **Renal**

- Reduced blood flow
- Reduced number of nephrons
- Reduced sensitivity to ADH
- Impaired excretion of sodium and water

## **Metabolic**

- Reduced total body water
- Increased body fat
- Reduced thirst and nutritional intake

## **Hepatic**

- Impaired clearance of many drugs

## **Immune**

- Relatively immunosuppressed

Respiratory function is further compromised by collapse of the small airways due to an increase in closing capacity, culminating in either partial or no ventilation of the lungs. This in turn causes air trapping, V/Q mismatch and hypoxaemia. In addition, trapped air will be absorbed leading to areas of atelectasis which combined with impaired mucociliary clearance predisposes to infection. Immediate assessment is vital with early use of mechanical ventilation to overcome the tendency to hypoxaemia because of reduced compliance, increased closing capacity and V/Q mismatch. Be wary of barotrauma and keep inspiratory pressures as low as possible because the elderly are susceptible to pneumothoraces especially with co-existing lung disease.

## **Circulation and haemorrhage control**

With increasing age the ventricles become rigid due to reduced myocardial mass and fibrosis which causes diastolic dysfunction. The arterial walls also become more rigid producing an increase in afterload that is superimposed on an already impaired left ventricle. The atria therefore assume greater importance in maintaining ventricular filling to ensure adequate cardiac output. As a consequence the elderly are less tolerant of a tachycardia that shortens diastole and further impairs ventricular filling, an effect exacerbated if the tachycardia is not sinus. The increased rigidity of the arterial system increases the systolic blood pressure with minimal change in the diastolic component, resulting in a wide pulse pressure.

The elderly have limited sympathetic drive and maintain cardiac output by increasing stroke volume, rather than by a tachycardia. Thus the elderly are less likely to present with the classic signs of hypovolaemic shock. A tachycardia is often absent, systolic blood pressure falsely reassuringly high, and no narrowed pulse pressure. In addition, the poorly compliant ventricles are intolerant of even modest circulating volume depletion or expansion. These facts

combined with changes in metabolism and renal function, require early invasive monitoring to guide fluid replacement.

As the elderly have such a reduced cardiac reserve, early, rapid and complete assessment for all sources of haemorrhage is necessary. Remember that the elderly are at risk of life threatening haemorrhage even from minor fractures of either the pelvis or hips.

#### *Disability of the CND (neurological assessment)*

There are many functional and structural changes in the brain that occur with aging. The decline in short term memory, visual and auditory reaction times, and rapid information processing place the elderly at greater risk of injury; and can also influence the accuracy of the AMPLE history. The space left by the atrophied brain is replaced by cerebrospinal fluid. This stretches the parasagittal veins that in turn are more prone to rupture on impact. The loss of brain volume also allows for more movement following acceleration or deceleration. As a consequence, there is a greater risk of haematoma that can be exacerbated by co-existent drug therapy (eg warfarin, aspirin, clopidogrel). Early head CT is essential.

#### *Exposure and Environmental control*

A reduction in lean body mass and impaired heat generation predispose to hypothermia. This is exacerbated by impaired detection of temperature change, and failing control of vasoconstriction. If hypothermia develops a series of events will further compromise functional integrity, and lessen the chance of survival. Thus be proactive and remember prevention is better than cure.

### **Summary**

Medical problems occur frequently in trauma patients. They may either precipitate the trauma or arise as a consequence of the injury or its subsequent treatment. They can also have a tremendous impact on resuscitation. Despite the numerous problems discussed we advocate that the approach and treatment of the major trauma patient follows the ABCDE system. If the patient fails to respond to treatment in the expected way then consider an underlying medical problem. An AMPLE history is invaluable, especially when supplemented by information by the General Practitioner (if known). The physician has an important role to play in the management of major trauma patients and therefore should be included in the trauma team.

# **CHAPTER 13**

## **MAXILLOFACIAL TRAUMA**

### **Objectives**

At the end of this chapter, the reader should understand:

- The importance of airway management in maxillofacial trauma.
- The relationship between facial injuries and injuries to the cervical spine.
- The management of severe bleeding in the head and neck region.

The applied anatomy relevant to maxillofacial injuries and the clinical features of facial and laryngeal injuries are contained in appendices 13.1 and 13.2 respectively at the end of the chapter.

### **Introduction**

Following the introduction of seat belt legislation, interpersonal violence has overtaken road traffic accidents as the most common cause of facial injuries in the United Kingdom. Home Office data demonstrates that interpersonal violence more than doubled between 1974 and 1990, and continues to increase. Where facial injuries result from violent crime, 50% of the victims have raised blood alcohol levels – an association likely to complicate the pre-hospital and early hospital care. One study has demonstrated that in assault cases resulting in fractures, 83% involved the facial skeleton. Isolated fractures of the mandible, nose or zygoma are most common in this situation. More extensive fractures of the midface and nasoethmoid regions are typically due to road traffic accidents or substantial falls. These are more likely to be life threatening, and also more likely to be associated with other injuries, particularly of the chest and abdomen.

### **Assessment and management**

This section emphasises the assessment and management of a maxillofacial injuries in the first two hours from the time the patient arrives in the accident unit, until their care is taken over by the maxillofacial team. It is not the intention to deal with the definitive surgical care of hard and soft tissue facial trauma. The initial management of the facial injury follows the procedure described in Chapter 1.

## **Primary survey and resuscitation**

**The primary survey is designed to detect and treat immediate life-threatening injuries.**

During the primary survey it is not necessary to make an accurate diagnosis of the facial injuries. Instead the team should focus on any potential life-threatening conditions such as airway obstruction. Those aspects of the primary survey, which are of particular importance in injuries to the head and neck are emphasised here.

### ***Airway and cervical spine control***

Airway obstruction is the most common cause of death in facial injury. The patency of the airway must be assessed immediately by speaking to the patient and assessing their response. This will yield immediate information, not only on the patency of the airway, but also the level of consciousness. Although a large proportion of casualties with facial injuries may be under the influence of alcohol or drugs, it should not be assumed that confusion is due to intoxication. Such behaviour may well be due to hypoxia, and improve once the airway is established, and other causes of hypoxia corrected. At the same time listen for the characteristic noises of airway obstruction such as stridor, snoring or gurgling. If the patient is hoarse, consider an injury to the larynx, or a foreign body such as a tooth or denture impacted on the vocal cords.

The conscious patient with bleeding from facial injuries is usually more comfortable sitting up with the head held forward. This allows blood and secretions to drain forwards out of the mouth, otherwise blood will gravitate into the hypopharynx causing coughing. If the patient is unable to sit up, then a prone or semi-prone position is preferable. While the airway is being assessed, movement of the cervical spine must be minimized, particularly in the unconscious patient, as described in Chapter 1. Even in severe facial injuries it is usually possible to establish an airway with simple procedures, although intubation may be required to protect the airway when it is proving difficult to control bleeding within the mouth and pharynx. It is unusual in civilian practice to have to resort to a surgical airway except where there has been a failed intubation, a foreign body has impacted in the vocal cords, or direct damage to the larynx. The stages to secure the airway are:

- Clear debris (broken teeth/dentures) from the mouth with Magill's forceps and suction. Keep any retrieved fragments to help the maxillofacial team account for missing or broken teeth.
- Try a jaw thrust or chin lift.
- If clearing the mouth and a jaw thrust have been unsuccessful, try pulling the tongue forward. In the unconscious patient this best achieved with a towel clip, or suture passed

through the dorsum of the tongue as far posteriorly as possible. Other instruments tend to crush the tongue, and increase the pain and swelling.

- If the anterior part of the mandible is comminuted, or there is a bilateral fracture, the tongue may have lost its anterior support allowing it to fall back against the posterior wall of the pharynx. In this situation, pulling the front of the mandible forward may clear the airway.
- If the maxilla has been pushed backwards down the inclined plane of the skull base, then pulling it forwards to disimpact it may also clear the airway. Backwards displacement of the maxilla may be suggested by finding the lower front teeth in front of the upper teeth, with an open bite.

In the majority of cases these manoeuvres will have established an airway, but it must be maintained. In most cases this is achieved with a nasopharyngeal or oropharyngeal airway of the correct size, although a tongue suture may sometimes be indicated to hold the tongue forward. Note that an oropharyngeal airway is easily dislodged, and poorly tolerated in a responsive patient. A nasopharyngeal airway is much better tolerated and less likely to be dislodged, but neither will prevent the aspiration of blood or vomit. They require frequent suction to prevent them becoming blocked. Also remember that care is needed when passing a nasopharyngeal tube in a patient with fractures of the middle third of the facial skeleton, as these may be associated with fractures of the base of the skull. Nasopharyngeal tubes should be passed horizontally through the nostril, and not upwards towards the skull base. Whichever method has been used to maintain the airway, it must be checked regularly. In practice, the casualty is usually intubated with a cuffed tube, both to maintain the airway and to reduce the chances of aspiration.

When these initial attempts to establish an airway fail, the most common cause is bleeding in the pharynx or nasopharynx, which has not been controlled (see below under circulation). There may also have been direct trauma to the larynx, from, for example, a karate blow, or a foreign body impacted in the vocal cords. An attempt is made to intubate the casualty, but if the degree of bleeding is too great to see the vocal cords, do not persist. Proceed instead to a surgical airway. If there is a foreign body impacted in the hypopharynx, this usually becomes apparent during attempted intubation and may be removed. If, however, it cannot readily be removed, do not persist but proceed quickly to a surgical airway.

### The surgical airway

A cricothyroidotomy is the preferred technique to establish a surgical airway in an acute emergency. It affords rapid and relatively safe access to the airway. Tracheostomy should be

regarded as a semi-elective procedure to be carried out by an experienced surgeon in a controlled environment. It is usually possible to establish a surgical airway with a cricothyroidotomy within two minutes. An alternative is needle cricothyroidotomy with insufflation of oxygen from a high-pressure source as described in Chapter 3. The only exception is a fracture of the larynx when a tracheostomy rather than cricothyroidotomy will be indicated.

#### Surgical airways in children

Establishing and maintaining the airway in a child in the presence of severe facial injuries may be challenging, but obstruction is rarely complete. If a surgical airway is needed, cricothyroidotomy should be avoided in children under the age of 12 as the cricoid cartilage is the only circumferential support for the upper trachea in this age group and accidental damage to it during cricothyroidotomy may have serious consequences. If an experienced surgeon is available, and time permits, a formal tracheostomy may be carried out. An alternative when all else has failed is needle cricothyroidotomy as described in Chapter 3. However, this is a technique with considerable risk of complications in small children.

#### **Breathing and ventilation**

The following is an account of those aspects of breathing assessment of particular relevance in the presence of facial injuries. A full account of the chest examination is given in Chapter 4.

If a semi-rigid collar has not been fitted, before doing so, examine the neck. This will yield important information not only about direct injury to the neck, but also to abnormalities of the chest. There may be laryngeal crepitus or surgical emphysema associated with a fracture of the larynx. Take particular care when examining penetrating wounds of the neck, resulting from ballistic injuries or knife wounds. If examination suggests that the wound extends deep to the platysma muscle, do not insert a gloved finger as torrential bleeding may result if a major underlying blood vessel has been damaged. Even perforations of the internal jugular vein may have tamponaded themselves by the time the casualty arrives. Such wounds should be formally explored in theatre with the appropriate vascular instruments readily to hand.

It is always safer to assume that a penetrating wound of the lower neck or supraclavicular fossa has involved the apex of the lung until proved otherwise. A haemopneumothorax may occur even when there are no apparent injuries below the clavicle. An assessment of the breathing is therefore important even when there does not seem to have been an injury to the chest (Figs. 13.1a, 13.1b).



**Fig. 13.1a (far left):**  
**Stab wound to neck, no**  
**injuries below the clavicle.**

**Fig. 13.1b (near left):**  
**CXR of same patient as in**  
**13.1a showing**  
**haemothorax.**

Facial injuries sustained in road traffic accidents are frequently associated with abdominal injuries, and may result in a ruptured diaphragm leading to abnormal signs in the chest if the viscera have been forced upwards into the thoracic cavity. Damage to the phrenic nerve in the neck following penetrating injuries will also paralyse the diaphragm on that side.

### **Circulation and haemorrhage control**

The major problems relating to maxillofacial injuries in the first two hours nearly always relate to the airway or bleeding. It is the A and C of the primary survey that are the most important. It is not the intention to cover the assessment of the circulation in this section, but to emphasise the control of bleeding in the head and neck region. A common mistake is to attribute hypovolaemic shock to maxillofacial injury, when covert bleeding in the abdomen, chest or pelvis is the more likely cause.

The tissues of the head and neck have an excellent blood supply, but this does mean that facial injuries bleed profusely. Nonetheless, in the absence of a severe middle third facial fracture or damage to a major blood vessel in the neck, the degree of bleeding is usually insufficient to cause clinical hypovolaemic shock. An exception to this is scalp injuries in children or severe fractures of the middle third of the face in adults. Scalp lacerations alone are unlikely to cause hypovolaemia in an adult, but significant scalp injuries in children may be associated with significant blood loss.

### ***Control of bleeding in the orofacial region***

It is important to control bleeding in the mouth and oropharynx as quickly as possible, not only to preserve blood but also to maintain an airway. The primary survey is normally carried out with the casualty in the supine position with in-line immobilisation of the neck. In this position any blood in the mouth will gravitate to the hypopharynx and obstruct the airway. Most bleeding in the oral cavity is accessible and can be controlled with local pressure with a swab. The tongue is very vascular and bleeds easily. Nonetheless, bleeding from tongue lacerations is readily controlled with deep sutures to include the underlying muscle. Infiltration with local anaesthetic containing a vasoconstrictor may also help reduce bleeding from intraoral lacerations.

Mandibular fractures are often open into the mouth, and bleeding from the bone ends may be difficult to control because of restricted access. This is due to damage to the inferior alveolar vessels and will usually stop once the bone ends have been approximated and temporarily immobilised. It sometimes helps to loop a stainless steel wire or suture around the teeth on either side of the fracture site to pull the bone ends together as a temporary measure. A particularly difficult area is severe post-nasal bleeding into the oropharynx in association with a fracture of the maxilla. The bleeding appears to be coming down from behind the soft palate and is not controlled by simple nasal packs. In this situation the bleeding is often from an associated skull base fracture. Bleeding of this type may be life threatening and many units of blood may be lost.

The following should be considered:

- Secure the airway first. Intubation with a cuffed tube is often possible if the blood pooled in the oropharynx is sucked out. If unsuccessful, proceed quickly to a surgical airway. It may also help to raise the head of the trolley to reduce the venous pressure in the head.
- Pass a Foley catheter back through each nostril until they can be seen behind the soft palate. Inflate the balloons and then pull them forwards to exert local pressure to the mucosa in the area. It may then be necessary to insert anterior nasal packs. The "Epistat™" device with anterior and posterior balloons may be used to the same end. Once the balloons are inflated, a finger can be inserted into the back of the mouth to push the back of the mobile maxilla up against the inflated bulb.
- Consider hypovolaemic resuscitation maintaining the systolic pressure at 80 mmHg until control has been achieved.

External bleeding from the scalp and soft tissues of the face is generally easy to bring under control with direct pressure or sutures. Resist the temptation to use electrocautery blindly, or to

try and apply artery clips deep in wounds without adequate vision, as it is easy to damage exposed branches of the facial nerve (VII cranial nerve) leading to paralysis of some of the facial muscles. This is particularly the case in the region of the parotid salivary gland.

The management of penetrating wounds of the neck has been discussed above. Probing wounds in the neck may precipitate induce bleeding. Do not probe neck wounds breaching the platysma until the casualty is in an operating theatre where unexpected severe bleeding may be controlled surgically.

### **Dysfunction of the CNS (neurological assessment)**

The assessment of the level of consciousness in the primary survey by the AVPU method is carried out in the normal way. There are some specific considerations in the presence of head and neck injuries.

Maxillofacial injuries are often associated with head injuries and eye injuries. Although alcohol or drugs will complicate the neurological assessment it is essential to establish a baseline level and reassess at intervals so that any deterioration is detected promptly and acted on. Speech may be slurred as a result of mouth and jaw injuries, and this will alter the patient's response to questions.

Eye injuries associated with maxillofacial injuries are easy to miss. Although not life threatening, the loss of site even in one eye is a tragedy for the individual, and early diagnosis may prevent this. Swelling may have closed the eye and made it difficult to examine. Pupil reactivity can also be changed following a blow to that part of the face. A traumatic mydriasis for example will cause one pupil to be larger than the other. It is essential to make at least crude assessment of visual acuity at an early stage. This is achievable even if the eye is too swollen to open by pushing a pen torch against the eyelid, and asking the casualty if they are aware of the light. The loss of vision in an eye is potentially reversible with treatment in the first few hours if it is due to a retrobulbar haemorrhage or optic nerve compression. The management of these conditions is beyond the scope of this chapter, but in this situation an urgent assessment by the ophthalmological team is required.

### **Exposure and environmental control**

The casualty is completely undressed to allow a full examination and measures taken to prevent hypothermia developing or worsening as described in Chapter 1.

## **Secondary survey**

The secondary survey is of particular importance in the presence of maxillofacial injuries, as statistically there are often other injuries which are less evident, but which may be life threatening. This is particularly the case in road traffic accidents, and fragmentation wounds from antipersonnel devices. In general, once the whole body has been exposed, the examination proceeds systematically from the head down to the feet. In this section, the important aspects of the secondary survey of the head and neck will be described. Observation and palpation are the key to examination of the head and neck.

### **Scalp**

The scalp should be examined for lacerations and haematomas. The latter may be misleading, and it is sometimes difficult to determine whether there is an underlying depressed fracture of the skull. Where there has been obvious significant blunt trauma to the scalp, regular head injury observations need to be taken. Scalp lacerations should be gently probed with the gloved finger to detect fractures of the underlying skull.

### **Bruising and swelling**

The swelling of the facial soft tissues may take some hours to develop fully. As soft tissue swelling often masks fractures of the underlying facial skeleton, radiographs will be the only way to make the diagnosis. For this reason, a depressed fracture of the zygoma is frequently missed. Similarly, a black eye may mask an underlying fracture of the orbitozygomatic complex, or damage to the globe of the eye. Le Fort fractures of the maxilla in particular are associated with gross facial swelling ("ballooning"). The bruises associated with skull base fractures may take 12 hours or more to become apparent. The "Panda eyes", bruising over the mastoid (Battles sign), or subconjunctival haemorrhage associated with skull base fractures or zygomatic fractures, may therefore not be obvious at an early stage.

Bruising of the neck, particularly when associated with noisy breathing and a hoarse voice should alert you to the possibility of a fracture of the larynx. Gentle palpation around the larynx may reveal crepitus. Once the nature of the bruising has been observed, the bony margins of the facial bones should be palpated to detect step deformities.

### **Cerebrospinal fluid (CSF) leaks**

The ear should be examined for the presence of blood or CSF in the external auditory meatus, often due to a tear in the anterior wall. This is typically a result of a blow to the chin forcing the head of the condyle of the mandible back through the tympanic plate, lacerating the lining of the anterior wall. For this reason a cut on the chin is frequently associated with blood in the external ear. If there is a skull base fracture, CSF will not be seen if the tympanic membrane is intact. However, blood in the middle ear will cause the tympanic membrane to bulge outwards and have a blue appearance through the auroscope.

Detecting a CSF leak from the nostril may be difficult, as in the early stages it is mixed with blood or mucus. A leak of cerebrospinal fluid (CSF) down the nose is usually associated with fractures of the cribriform plate of the ethmoid, as a result of Le Fort II and III fractures of the maxilla, or fractures of the naso-orbital-ethmoid (NOE) complex. A tell-tale sign is “tramlines” running down the cheek. These are caused by blood separating from the CSF to leave two outer lines of blood separated by CSF. They are not however diagnostic. After a day or two, as the bleeding stops, a more obvious watery discharge may become apparent. Where there remains doubt, an MR scan may be required.

### **Lacerations**

Facial lacerations are often closed under local anaesthetic in the Emergency Department before transfer to the ward. Before doing so, consider the following:

- Has any branch of the facial nerve (VII cranial nerve) been divided? Microneural repair of the damaged nerve is much easier if carried out at initial wound closure in an operating theatre. Finding the fine branches as a secondary procedure after suturing can be almost impossible. The branches of the facial nerve are easily tested by; asking the casualty to wrinkle the forehead, screw up the eyes and show the teeth. Particular care is needed with lacerations over the parotid salivary gland in front of the ear. Not only might the facial nerve be divided, but there may also be damage to the parotid duct.
- Is there a foreign body in the wound? Pieces of teeth or even whole incisor teeth may be concealed in a lacerated swollen lip, as may pieces of windscreens glass. If the wound is contaminated with grit, this must be thoroughly cleaned prior to closure or unpleasant tattooing may result.
- If there is a laceration of the neck, which appears on superficial examination to have perforated the platysma muscle, as in a stab wound, do not attempt to explore and suture

the wound under local anaesthesia in the Accident Unit. This may induce severe bleeding if major vessels such as the internal jugular are involved. The correct vascular instruments must be to hand before exploring such wounds. Resist the temptation to blindly try and clamp bleeding vessels in the neck. It is easy to clamp and damage important nerves such as the Accessory.

### **Examination of the eyes**

Although damage to the eyes is not life threatening, detecting an eye injury with deteriorating vision within the first two hours, and seeking urgent help, may prevent permanent loss of vision. The pupil size and reactivity will have originally been noted as part of the AVPU assessment under D of the primary survey. Any change in size or reactivity should be noted over time to detect intracranial bleeding, or decreasing vision due to, for example, pressure on the optic nerve at the apex of the orbit. It is therefore important during the secondary survey to assess the eyes, and remove any contact lens. Do not be deterred by swelling around the eye. It may be necessary to delay a full ophthalmology assessment until the swelling has reduced but by shining a pen torch held against the swollen lids, it is possible to at least determine whether the visual pathway is intact. The conscious casualty will be able to tell you whether he sees the light through the eyelids. If uncooperative, the reaction of the other pupil may indicate that the visual pathway is intact.

The high incidence of missed injuries to the globe associated with cheekbone and orbital fractures has been well documented. In addition to assessing the visual acuity, if the eyelid swelling permits look for the bloodshot eye (subconjunctival haemorrhage), examine the fundus, and test for double vision. A sub-conjunctival haemorrhage may result from direct trauma to the conjunctiva, or, if the posterior limit of the bleeding is not visible, may be due to blood tracking forwards from a fracture of the orbit or cheekbone. Remember that subconjunctival haemorrhage arising from a fracture may take some hours to develop.

Double vision if present may indicate oedema in the orbit, or a blowout fracture of the orbital walls, usually the orbital floor.

### **Where are the teeth?**

As far as possible, any missing teeth or pieces of broken denture should be accounted for. They may have been left at the scene of the accident, but consider the possibility of inhalation, particularly if the casualty was unconscious (see above). The other possibilities to consider are:

- Buried in a lip laceration, as a foreign body
- Inhaled
- Ingested

When examining the mouth, look for any tongue lacerations, which may bleed profusely and cause airway obstruction. A step in the occlusal plane of the teeth or an obvious dental malocclusion may signify a fracture of the mandible or maxilla.

### **Summary**

This chapter has dealt with the management of maxillofacial injuries in the first two hours after arrival in the Emergency Department, and has emphasised the management of the airway and control of bleeding. The need to stabilise the cervical spine, detect decreasing vision, and watch for neurological deterioration in this first two hours has also been stressed. The key to safe management is a seamless interface between pre-hospital and hospital care, and good communication with the surgical teams who will undertake the definitive care and reconstruction of the injuries. The exact diagnosis of the facial fractures at this initial stage has not been necessary other than to identify injuries may be potentially life threatening.

## **Appendix 13.1: Applied anatomy**

For the purposes of this chapter, the head and neck region is best regarded as a closed box (the skull) below which the facial bones are suspended and attached to the inclined skull base. This is supported by the cervical spine, which is easily damaged in deceleration injuries such as road traffic accidents or falls. There is therefore a relationship between facial injuries, head injuries, and injuries to the cervical spine. If a casualty with a significant facial injury is unconscious, there is a 10% chance of an associated injury to the cervical spine. The most important manifestation of maxillofacial injuries is, nonetheless, airway obstruction, and this is the most common cause of death in this type of trauma.

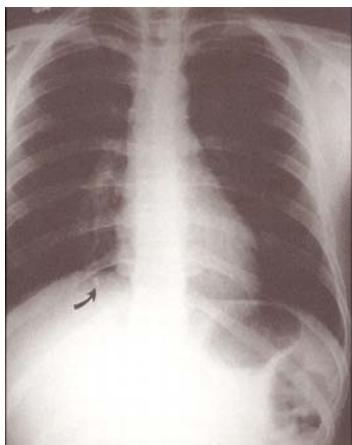
The middle third of the facial skeleton is a complex structure consisting of the two maxillae and nasal bones centrally, and the zygomatic bones laterally. The maxillary bones are thin, but thickened laterally to form four buttresses that pass vertically from the tooth supporting alveolar bone, up to the skull base. These are designed to absorb the vertical stresses of mastication, but collapse relatively easily with anterior forces. As a result of this, the bones of the central midface may function in the same way as the “crumple zone” of a car with the application of a significant anterior force. As the middle third of the face “crumples” it absorbs energy that would otherwise be transmitted to the skull base, increasing the chance of brain injury. As the middle third of the facial skeleton is displaced backwards it slides backwards down the inclined base of the skull, obstructing the airway, and causing a gap between the upper and lower front teeth. In this situation, dragging the upper jaw forwards with fingers behind the palate may relieve the airway. As the central facial skeleton is forced backwards it separates from the skull base at one of three levels originally described by Le Fort early in the last century.

The lateral part of the middle third is formed by the two strong zygomatic bones whose prominence is a protective mechanism for the eye. They also form part of the floor of the orbit, and so zygomatic fractures are frequently associated with eye injuries, which may be masked by the soft tissue swelling, and missed. Always “beware the black eye”.

The mandible forms the lower third of the facial skeleton. It is a strong bone that articulates with the skull base at the temporomandibular joint. It provides the anterior support for the tongue via the muscle attachments to the genial tubercle. If there is a bilateral fracture of the mandible, or comminution of the anterior mandible, the tongue support may be lost, allowing the tongue to fall back and obstruct the airway.

The necks of the mandibular condyles are relatively weak and are a common fracture site. A blow to the chin such as a punch may be transmitted back through the mandible to cause a fracture of the condyle, an injury that is often missed. This injury should always be suspected if there is a laceration on the chin. The fractured condyle may also be forced back into the external auditory meatus causing a laceration of the anterior wall. This results in bleeding from the ear that may initially be misdiagnosed as a skull base fracture.

Teeth are frequently knocked out or fractured in maxillofacial trauma. Wherever possible, any missing teeth should be accounted for, as they may have been inhaled, particularly in the unconscious patient. An inhaled tooth is most likely to be found in the right main bronchus, although smaller fragments may slip further down into the more peripheral airways (Fig. 13.3). An avulsed tooth in the right main bronchus may be overlooked on a standard chest radiograph as it may be masked by the border of the heart. In addition to teeth fragments of acrylic dentures may be inhaled or become lodged in the vocal cords. Early bronchoscopy is indicated to avoid the development of pulmonary complications. Swallowed teeth usually pass through the alimentary canal without complication.



**Fig. 13.2: Chest x-ray showing inhaled fragment of tooth in right lung (arrowed).**

Facial injuries, particularly those to the middle third of the face may cause rapid soft tissue swelling, making it difficult to palpate underlying bone fractures. Gross swelling of the face should always alert the examiner to the presence of a fracture, but radiographs are often necessary to clarify the extent of the injury. The soft tissues of the face and scalp have a good blood supply. Soft tissue facial injuries bleed profusely, but the extent of blood loss is often overestimated. Where there is obvious hypovolaemic shock, it is important to search for covert bleeding elsewhere such as in the abdomen or chest. It is easy for an examiner to be distracted by the appearance of a major facial injury, and to overlook a more life threatening injury elsewhere. The good blood supply also means that tissue necrosis is unusual in facial injury and any debridement should be relatively conservative, preserving facial skin. Nonetheless, wounds

contaminated with debris such as road grit must be thoroughly cleaned to avoid unsightly tattooing of the wound requiring later revision surgery. Extensive lacerations of the face frequently give the impression of tissue loss because muscle retraction pulls the edges of the wound apart.

## **Appendix 13.2: Clinical features of facial and laryngeal fractures**

### *Injuries to the larynx and trachea*

- Evidence of direct trauma to the neck (bruising and swelling)
- Noisy breathing (snoring, gurgling, croaking)
- A hoarse voice
- Crepitus on palpation

### **Fractures of the mandible**

- Pain on jaw movement
- Swelling and bruising
- Bleeding from the mouth
- The upper and lower teeth do not meet properly
- Step in the occlusal plane of the teeth
- Numbness of the chin and lower lip on that side (trauma to the inferior alveolar nerve).
- Mobility at the fracture site.

### **Fractures of the maxilla**

- Ballooning of the soft tissues of the face.
- The upper and lower teeth do not meet properly (possible anterior open bite)
- The middle of the face may look flat before the swelling hides it.
- Numbness of the skin of the cheeks (infra-orbital nerve).
- Cerebrospinal fluid leak from the nose if associated anterior skull base fracture.
- The upper jaw can be moved:
  - a. In a Le Fort I fracture only the tooth-bearing portion of the maxilla is mobile.
  - b. Le Fort II fracture – the bridge of the nose moves with the maxilla.
  - c. Le Fort III fracture – both cheekbones move with the maxilla.

### **Fracture of the zygoma**

- Black eye (circumorbital ecchymosis).
- Bloodshot eye (sub-conjunctival haemorrhage).
- Numbness of the skin of the cheek and upper lip on that side.
- Loss of prominence over body of zygoma.
- Possible double vision, particularly on upwards gaze.
- Unilateral nosebleed

### **Fracture of the naso-orbital ethmoid complex**

- Swollen and deformed bridge of nose
- Deepening of the angle between the nose and forehead (the depression of the root of the nose makes the nostrils more prominent from the front (“pigs snout”))
- Nosebleed with possible CSF leak
- The eyes may appear to be too far apart due to the detachment of the medial canthi (traumatic telecanthus).

## Websites

<http://www.baoms.org.uk/oral%20and%20maxillofacial%20surgery.html>

British Association of Oral & Maxillofacial Surgeons website with guidelines.

<http://www.vh.org/Providers/Lectures/IROCH/FacialTrauma/FacialTrauma.html>

Radiological tutorial.

<http://tristan.membrane.com/aona/case/max/index.html>

Surgical case histories.

<http://www.nlm.nih.gov/medlineplus/eyeinjuries.html>

Good starting point for eye trauma websites.

## **Further reading**

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# **CHAPTER 14**

## **INJURIES DUE TO BURNS**

### **OBJECTIVES**

At the end of this chapter, the reader should be able to:

- Understand the importance of pre-hospital burn management.
- Apply the ABCDE approach to burn management.
- Understand basic burn assessment and calculation of fluid resuscitation requirements.
- List criteria for referral to a specialist Burns Centre.
- Understand how burns patients are prepared for transfer.

The pathophysiology of burns injury and treatment of specific types of burns injury are covered in Appendices 14.1 and 14.2 respectively at the end of the chapter.

### **BACKGROUND**

Burns are most commonly caused by exposure to flames. Scalds are the next most common cause and electrocution and chemical injuries occur only rarely. In the UK an estimated 175,000 acute burn injuries present each year to Emergency Departments. While most are relatively minor, an estimated 16,000 (9%) require admission, 1000 require intravenous resuscitation and approximately 300 die. These figures are similar in many European countries, but are higher in the US. In developing countries, burns mortality is much higher. The definitive care of burns is complex and requires a multidisciplinary team approach. There is no place for the occasional burn surgeon in the management of these patients. Although all large burns should be managed in Burns Centres, the initial emergency management of the burn victim must occur in the pre-hospital environment and the emergency department. Straightforward effective early management of the burns victim before arrival at the specialist centre can have a major effect on outcome. Burn victims are primarily trauma victims and need accurate assessment, careful initial resuscitation and rapid transfer to specialist care. Although the latter is important, adequate resuscitation and careful preparations for transfer are vital.

### **Primary Survey and Resuscitation**

A full primary survey using the ABCDE approach should be carried out on arrival. This will identify any immediately life-threatening injuries. A more thorough assessment of the burn can be undertaken later.

### *Airway, cervical spine control and breathing*

Assessment of the airway and breathing may reveal early signs of inhalational injury and frequently coexisting injury. The development of signs and symptoms from airway oedema and pulmonary injury may take hours to develop. The key to diagnosis is having a high index of suspicion with the frequent re-evaluation of those considered to be at risk. The indicators of inhalational injury may come from:

#### *Signs and symptoms:*

- Hoarseness or other voice changes.
- Harsh cough.
- Stridor.
- Facial burns.
- Singed nasal hair.
- Soot in saliva or sputum.
- An inflamed oropharynx.

#### *History:*

- Exposure to fire and/or smoke in an enclosed space.
- Exposure to a blast.
- Collapse, confusion or restlessness at any time.

#### *Investigations:*

- Raised carboxyhaemoglobin levels.
- Respiratory failure.

All patients should be given high flow oxygen, preferably humidified. If any signs of upper airway obstruction, particularly stridor is present, this indicates that some obstruction already exists and tracheal intubation will be required. An experienced anaesthetist should urgently assess the patient and the safest option is early tracheal intubation as swelling will increase over the first few hours, making this task progressively more difficult. In severe cases a surgical airway may be required.

The cervical spine must not be overlooked in these patients, particularly when the mechanism of injury suggests that the spine may be injured. The classic case would be where the victim of a house fire has jumped from an upper floor to escape the fire and has burns and spinal injuries from the impact of the fall. Tracheal intubation may also be required in those patients with

significant lung injury to optimise ventilation and on the rare occasion of circumferential chest burns restricting spontaneous ventilation.

### **Circulation and haemorrhage control**

Hypovolaemic shock due to burns takes time to develop. If the burn victim shows signs of shock soon after injury other causes of haemorrhage must be excluded. For example, the mechanism of injury (e.g. a fall whilst escaping a fire) should raise the suspicion of other injuries. Where early hypovolaemic shock exists the cause needs to be investigated and patient managed as described in Chapter 5, irrespective of any burns injury. Intravenous access is achieved using two large bore cannulae. Although it is acceptable to cannulate through burnt skin, this should be avoided if possible. If necessary, central veins or the intraosseous route should be used. When blood is sent for laboratory baseline investigations carboxyhaemoglobin levels should be included where an inhalation injury is suspected.

### *Disability of the CNS (neurological assessment)*

Reduced level of consciousness, confusion and restlessness can indicate intoxication and/or hypoxia secondary to an inhalation injury. The possibility of alcohol or drug ingestion and the presence of other injuries may present in the same way.

### *Exposure and environmental control*

Clothing and any restricting items should be removed. The removal of clothing and use of cold water at the scene, during transfer and in the emergency department often leads to hypothermia. This can be minimised by covering uninvolved areas and raising the ambient room temperature (to 30°C). Before progressing to the specific management of the burn and a full secondary survey, the ABCDE of the primary survey should be reassessed.

## **Management of thermal burns**

### *Inhalation injury*

Intubation and ventilation is the mainstay of early management. Patients with suspected inhalation injuries require close observation in an area equipped for immediate intubation. An experienced anaesthetist should accompany the patient until arrival at the receiving burns centre and have an extremely low threshold for elective intubation before interhospital transfer. The burned airway will always become more compromised at a rate that is difficult to predict. Pulse oximetry readings should be interpreted with caution, especially in the presence of

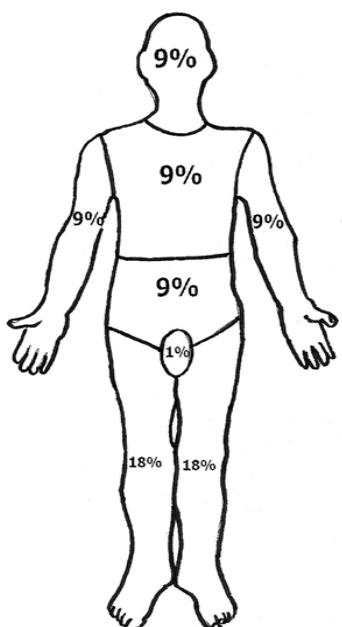
carboxyhaemoglobinaemia. Arterial blood gas analysis and a chest x-ray are also essential. These provide an important baseline as the patient may deteriorate from an initially normal state. There is no evidence that the administration of steroids is beneficial.

#### *The cutaneous burn*

Regardless of the cause of the burn, the severity of the injury is proportional to the volume of tissue damage. Mortality is predicted by the percentage of total body surface area (%TBSA) burned. Functional outcome is more often dependent on the depth and site of the burn.

#### **Calculating %TBSA burn**

There are several techniques for calculating %TSAB. Initial assessment can be made with a 'rule of nines' or a serial halving technique. The rule of nines divides the body into multiples of nine (Fig. 14.1). Serial halving assesses burn size on the basis of asking the question 'is half of the body burned'? If not, is it half of that and so on until an estimate is achieved. A more accurate method that can be used to calculate fluid requirements is the Lund and Browder burns chart (Fig. 14.2). When using this chart it is important to be precise. The burnt areas are accurately drawn onto the chart and then the %TBSA calculated. Erythema should be ignored. In very large burns it can be easier to calculate the size of area not burnt. Differentiating between full and partial thickness burns is not essential. The palmar surface of the patient's hand including the fingers equates to 1% TBSA and can be used to estimate small areas of burn.



**Fig. 14.1: The 'rule of nines' estimates TBSAB by dividing the body surface area in multiples of nine percent. The head and arms equate to 9% each, the anterior and posterior aspect of the chest, abdomen and lower limbs each equate to 9%.**

### *Fluid resuscitation*

Any burn greater than 10% TBSA in a child and 15% TBSA in an adult will require intravenous fluids to prevent the development of burn shock. There are various formulae available to calculate fluid requirements (Box 14.1). The Parkland formula is commonly used:

**2 - 4 mls Hartmann's solution x %TBSA burn x kg body weight**

Use the higher value of 4mls initially. Weigh the patient or ask their weight, estimates are often inaccurate. A child's weight can be obtained by using a recognised formula or a Broselow tape. Having estimated the TBSA as accurately as possible and ascertained the patient's weight, the formula gives a volume of fluid. Half this volume is administered in the first eight hours and the second half over the next sixteen hours. The fluid requirements are calculated from the time of injury (not the time of calculation, so most patients will already be behind with requirements) and does not allow for other losses, or for maintenance needs. It is therefore essential to monitor the adequacy of the fluid resuscitation. This is achieved in the emergency department by measuring urinary output. Urinary catheterisation is therefore mandatory. Aim for urine outputs of:

1 ml / kg / hr in adults

2 mls / kg / hr in children

Clearly where there are other injuries or the TBSA is greater than 15% patients may need blood in addition to crystalloid solutions.

**The Lund and Browder Chart:**

The numbers represent %TBSA  
Letters are age dependent:  
See chart below.

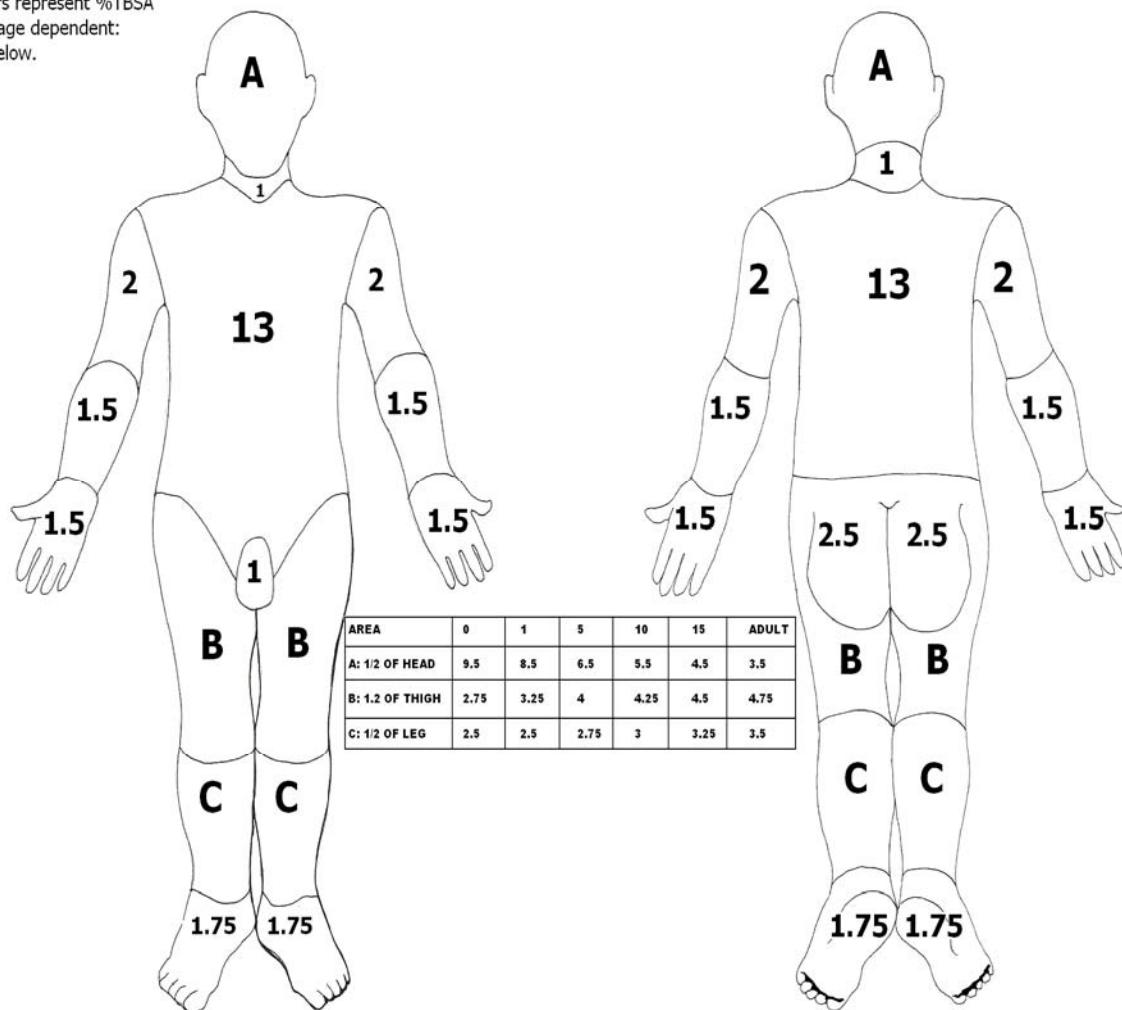


Fig. 14.2: Lund and Browder charts allow accurate calculation of the TBSA burned adjusted for age.

**Box 14.1: Summary of the different formulae to calculate burns fluid requirements**

Parkland	Hartmann's solution 4ml / kg / % burn: half given in the first 8 hours and the rest in the subsequent 16 hours
Muir – Barclay	Human Albumin Solution 0.5ml/kg/% burn administered in each of six periods of 4, 4, 4, 6, 6, and 12hours for the first 36 hours after the burn
Brooke	Hartmann's solution 1.5ml/ kg/ %burn + colloid 0.5 ml/ kg/ % burn + 2000 ml 5% dextrose solution
Modified Brooke	Hartmann's solution 2ml/ kg/ % burn in first 24hours

Evans	Normal saline 1ml/ kg/ % burn + colloid 1ml/ kg/ % burn + 2000ml 5% dextrose solution
Dextran (Demling)	Dextran 40 in saline at 2ml/ kg/ hour for 8 hours + Lactated Ringers to maintain urine output at 30ml/hour + fresh frozen plasma at 0.5ml/kg/hour for 18 hours beginning at 8 hours post burn
SBH-Galveston Paediatric Formula	Hartmann's solution at 5000ml/ square metre body surface area burn + 2000ml/ square metre body surface area: half given in the first 8 hours and the rest in the following 16 hours.

### **Management of the thermal burn wound**

Effective burn wound management aims to achieve maximum functional and cosmetic outcome. Apart from small superficial burns, wound management should be performed in a burns centre. After the wound is cooled and dressed there are rarely any indications for further interventions before transfer.

#### *Initial treatment*

Dressing the wound can be achieved by loosely covering the burn with Clingfilm. Hands can be placed in plastic bags. The patient should then be kept warm with dry blankets. Further accurate assessment of the wound can take place at the burns centre after transfer. Topical antiseptic solution and creams should not be applied.

### **Escharotomy**

A circumferential full thickness burn can act like a tourniquet and compromise circulation. Surgical division of the constriction is known as escharotomy. This should be performed in an operating theatre by skilled persons. There is rarely a need to perform an escharotomy within the first few hours. The exception is a full thickness burn of the entire trunk that is compromising ventilation. In this situation pre-transfer escharotomy must be discussed with the burns centre.

### **Other initial interventions**

Ensure immunity against tetanus. In the absence of any specific indications such as associated contaminated wounds, there is no requirement for antibiotic prophylaxis. In contrast a nasogastric tube and urinary catheterisation will be needed in all patients with complex burns (see below). As burns are painful, adequate pain relief is a priority from an early stage.

Intravenous opiates should be administered until the patient is comfortable. Pain leads to catecholamine release and may increase peripheral ischaemia and potentially burn depth.

### **Transfer to Definitive Care**

**In all cases, early contact should be made with a burn centre so that advice on initial management and transfer can be given. Guidelines for referral take into account the size of the burn and other indicators of complexity. Similar guidelines exist in most countries and those involved in the management of burns patients must be aware of local protocols. In most countries, all complex burns are managed in specialised Burn Centres. The following is a guide to the types of complex burns that should receive specialist attention.**

#### *Complex Burn Injuries*

A burn is defined as complex if one or more of the following criteria are met:

- Age
  - under 5 yrs or over 60 yrs
- Area
  - Burns over 10% TBSA in Adults
  - Burns over 5% TBSA in Children
- Site
  - Burns involving face, hands, perineum or feet
  - Any flexure, particularly the neck or axilla
  - Any circumferential dermal or full thickness burn of the limbs, torso or neck
- Inhalation Injury
  - Any significant inhalation injury, excluding pure CO poisoning
- Mechanism of Injury
  - High pressure steam injury
  - High voltage electrical injury
  - Chemical Injury >5% TBSA
  - Hydrofluoric acid injury (>1% TBSA)
  - Suspicion of Non-accidental injury; adult or paediatric
- Existing conditions
  - Cardiac limitation &/or MI within 5 years

- Respiratory limitation of exercise
- Diabetes Mellitus
- Pregnancy
- Immunosuppression of any cause
- Hepatic Impairment; cirrhosis
- Associated Injuries.
  - Crush Injuries
  - Major long bone fractures
  - Head Injury
  - Penetrating Injuries

Associated Injuries may sometimes delay referral of the burn. In these circumstances advice about burns management should be sought.

### **Preparations for transfer**

Once the decision to transfer a patient to a burns centre is made, preparations for safe transport should begin. Distance to the nearest burns bed and method of transfer will vary both within and between countries. With some longer distance transfers, rotary or even fixed wing aircraft may be required.

Before transfer it is important to carry out the following:

- A thorough secondary survey has been performed and any injuries identified be appropriately managed.
- Maximum inspired oxygen is being administered.
- If there is any suspicion of an inhalation injury, the patient should have been assessed by an experienced anaesthetist and intubated if necessary.
- Adequate intravenous access is secured and appropriate fluid resuscitation has started (as judged by an adequate urine output).
- The burn wound is covered with Clingfilm and the patient is being kept warm.
- There is adequate analgesia.
- There is a urinary catheter in place.
- There is a free draining nasogastric tube in place.
- All findings and interventions, including fluid balance, are clearly and accurately documented.

All patients should be transferred with an appropriately trained escort. If it is likely that a delay in transfer will exceed six hours then the situation needs to be discussed further with the burn centre. In this circumstance it may be deemed necessary for:

- Escharotomies to be performed
- The burn wound to be cleaned and a specific dressing applied
- The commencement of maintenance intravenous fluids and/or nasogastric feeding.

## **Appendix 14.1: Pathophysiology of burns injury**

### *Thermal burns*

Direct thermal injury causes progressive cell death with temperatures over 45°C and almost instantaneous cell death above 60°C. The amount of tissue destruction depends on the degree of heat multiplied by the exposure time. Early cooling of a burn reduces the magnitude of the local inflammatory injury. Heat is also conducted into surrounding tissues and causes a sub-lethal inflammatory injury. Inflammation is seen as increased capillary permeability and loss of fluid from the intravascular space. The clinical impact of the inflammation evolves for several hours and is related to the total volume of tissue injured. This is best expressed as the percentage of total body surface area burned (%TBSAB).

The most superficial of burns cause erythema. This does not cause capillary leakage and should not be considered when calculating the %TBSAB. Burn injury of greater than 15% TBSAB (10% in children) cause sufficient loss of intravascular fluid for compensatory mechanisms to be overwhelmed. Patients with this degree of burn injury will need to be given intravenous fluids to prevent the development of shock. Injuries above about 25-30%TBSAB cause massive activation of inflammatory mediators and the development of Systemic Inflammatory Response Syndrome (SIRS). The initiation of this process takes several hours.

### **Specific types of injury**

#### **Inhalation injury**

There are three components to inhalational injury:

- The true airway burn

This is caused by inhalation of hot gases (flame, smoke and steam). The injury is normally confined to the upper airways because of the protective laryngeal reflexes and leads to oedema with the risk of airway obstruction. The swelling develops over several hours and is maximal between 12 and 36 hours.

- Lung injury

Where products of combustion are inhaled into the lower airways, they dissolve into the fluid lining the bronchial tree and alveoli. This causes a chemical injury of the lungs which can produce varying degrees of respiratory failure, often delayed by hours or even days.

- Systemic toxicity

Absorption of the products of combustion into the circulation through the alveoli can lead to systemic toxicity. This is a common cause of death due to fires in enclosed spaces. The most important agents are carbon monoxide and cyanides. Note that in the presence of carboxyhaemoglobin, pulse oximeter readings are unreliable.

Although the contribution of inhalation injury to burns mortality has not been well quantified, its presence significantly worsens the prognosis.

### **Cutaneous injury**

Classification of the burn wound is descriptive and indicates the depth involved.

- Epidermal burns.

Causes erythema alone, like sunburn. There is rapid spontaneous healing. Not included when calculating %TBSAB.
- Partial thickness burns.

Superficial dermal. Wet and blistered. Marked erythema that blanches on pressure and with intact capillary refill. Deeper skin structures survive. If managed correctly should heal in less than two weeks.

Deep dermal. Darker red that does not blanch. This 'fixed staining' is caused by damage to deeper blood vessels. Often requires skin grafting.
- Full thickness burns.

Total destruction of the dermis, leaving a firm leathery necrotic layer known as eschar. Can be waxy white or lobster red fixed staining. Soot or charred tissue may mask true appearance. Excision and grafting is required except for very small areas.

Skin thickness has a functional relevance. For the same temperature and duration of contact, a functionally deeper burn will be produced in the thinner skin of children and the elderly. Pain levels do not reliably indicate burn depth. Burns are often not homogenous and a mixed depth pattern may be seen. Burn depth has little bearing on fluid requirements but will dictate later wound management. Attempts to accurately define burn depth in the first few hours are unnecessary.

### **Electrical burns**

Burns following electrocution are due to resistance to the conduction of electricity through body tissues. The degree of tissue damage is determined by the resistance of the tissue, duration of contact, and the square of the current ( $I^2$ ). The presence of small surface burns may not reflect

the severity of underlying tissue destruction. Many electrocutions result in falls, and there is therefore a high risk of associated injury. Cardiac arrhythmias may occur following discharge of current across the thorax and are usually the cause of fatal electrocution. Electrical injuries can be divided into three groups: low voltage, high voltage and lightning strike:

### ***Low voltage***

Electrical burns are classified as low voltage below 1000V. These include domestic mains supply (220-240V), single-phase alternating current, and industrial power supply at 415V, three-phase current. Low voltage electrical burns most commonly involve local tissue destruction, with charring of the skin and necrosis of tissues immediately beneath. Duration of contact may be prolonged due to muscle tetany caused by domestic alternating current at 50Hz.

### ***High voltage***

High voltage electrical burns occur over 1000V and where for example high-tension transmission cables are involved can reach 11,000 - 33,000V. High voltage injuries occur through either flash burns or current transmission. Relatively small entry and exit wounds may be associated with massive underlying damage of muscle and bone and entire compartments may be destroyed. Multiple entry and exit wounds may be seen, especially if the current has arced across joints on its passage to earth. Secondary damage as a result of the development of a compartment syndrome may occur.

### ***Lightning injury***

Though uncommon in the UK, lightning injuries result from ultra high tension, high amperage short duration electrical discharge of direct current. A direct strike, where the discharge occurs directly through the victim is almost invariably fatal. More commonly, a 'side strike' or 'splash' occurs, when lightning strikes an object of resistance, such as a tree and is then deflected through the victim. Typically the current flows over the surface of the victim causing partial thickness burns, though there may be significant burns of the feet. Wounds may have an unusual arborescent, or splashed-on appearance known as Lichtenberg Flowers. Though deep organ damage is not often seen, respiratory arrest may occur through the reversible effects of discharge on the medulla. Prolonged resuscitation efforts in these patients are justified.

### Chemical Burns

Two thirds of those who suffer chemical burns are male. The hands and lower limbs are most commonly affected. The number of chemical burns can be reduced by safer working practices and Health and Safety legislation. The majority of work-related chemical burns seen are due to cement. More than 50% of chemical burns are now due to domestic products, often oven and drain cleaning compounds. Less than 5% of these injuries require admission.

With two notable exceptions, chemical burns can largely be divided into those caused by; acids, bases or alkalis, and organic hydrocarbons. Acids produce coagulative necrosis similar to a thermal burn and as such prevent deep penetration of the burning agent through formation of an eschar. Alkalies, in contrast, cause injury through liquefactive necrosis and saponification of fats, and penetrate deeper into tissues. Organic hydrocarbon compounds, such as petrol, can cause a chemical burn without ignition by liquefaction of lipids.

Hydrofluoric acid is used in several industrial processes. In burns caused by hydrofluoric acid, the fluoride ion is absorbed and chelates calcium and magnesium ions causing bone demineralisation, cell death and potassium release. The fall in serum calcium and rise in serum potassium can be very rapid and lead to arrhythmias, refractive VF and death. Hydrofluoric acid burns can be fatal at less than 2.5% TBSA. White phosphorus burns are largely seen in the military. Phosphorus ignites on contact with air. It is very fat-soluble and produces yellow blisters with a characteristic garlic smell. If absorbed hepatorenal toxicity may occur and death has been recorded with small doses. Management of these burns involves careful physical removal of phosphorus particles.

Victims of major burns should initially be assessed using the priorities described in Chapter 1, as airway burns are the most immediate threat to life, followed by injuries to the lung, which in turn are a more immediate threat than the potential loss of fluid from the circulation. In addition to the burn injury, these patients may also have other co-existing injuries, for example fractures. All burns of greater than 10% TBSA and all high voltage electrical injuries should be assessed and treated in a resuscitation area. Cooling of burns is usually commenced in the pre-hospital phase. If not, or if a patient arrives soon after an incident, cooling should commence or continue. Benefit from cooling may still be seen even if cooling has not been started within 30minutes from the time of burning. However, it is important to be aware of causing hypothermia, especially if there are large areas burnt, or the patient is very young or elderly. The decision to stop cooling needs to be based on patient core temperature. It is often possible to warm the patient but cool the burn but this is difficult in very large burns.

## **Appendix 14.2: Management of specific burns**

### **Chemical Burn wound**

The mainstay of treatment is copious and continued irrigation with water. This should be carried out for at least 30 minutes in the case of an acid burn, and for at least an hour following burn due to alkali. Hypothermia should be avoided and the water used to irrigate can be warm. It is important that the diluted chemical is not allowed to pool around the body, as further injury may occur. Indicator paper can be placed intermittently on the wound to see if pH is returning to normal. Chemical burns of the eye require prolonged irrigation and early consultation with an Ophthalmologist. Specific antidotes are rarely required but are indicated for some specific agents (e.g hydrofluoric acid injury should be treated with 1% calcium gluconate). Once all the chemical has been removed and the wound is clean, treatment is as for a thermal burn.

### **Electrical Burns**

#### **Associated Injury**

High voltage electrical injury carries a high risk of fatality at the time of electrocution. Survival has been reported after prolonged cardiopulmonary resuscitation and efforts should not be terminated prematurely. Electrical workers may have been thrown from a height and suffered additional serious injury. Primary and secondary survey of all high voltage electrical injuries should be carried out in the emergency department.

#### **Arrhythmias**

In all cases of electrocution, an ECG should be performed. If abnormal, continuing cardiac monitoring should be employed. In the absence of ECG abnormality or cardiac history, continued cardiac monitoring is of no proven benefit.

#### **Fluid Resuscitation**

As discussed above a cutaneous electrical burn may not reflect the severity of underlying injury. Reliance on fluid replacement formulae may underestimate fluid requirements and careful monitoring of urine output is essential. In patients with deep damage, haemochromogenuria may occur and deposition of haemochromogens (myoglobin, haemoglobin) in the proximal tubules may cause acute renal failure. Resuscitation fluids should be increased to maintain a urine output of at least 2ml/kg/h. If this goal proves difficult with crystalloid resuscitation alone, mannitol may be considered, and alkalinisation of the urine can promote haemochromogen excretion. Some intensivists consider mannitol potentially harmful in these circumstances and prefer to avoid it.

### **Limb Injury**

These patients are at risk of compartment syndrome (Chapter 9). Regular and frequent assessment of injured limbs must be carried out.

## **Web sites**

- [www.burnsurgery.org](http://www.burnsurgery.org)
- [www.baps.co.uk](http://www.baps.co.uk)

## **Further Reading**

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# **CHAPTER 15**

## **HYPOTHERMIA AND COLD INJURY**

### **Objectives**

The objectives of this section are that members of the trauma team should understand:

- How to define hypothermia
- How to diagnose hypothermia
- Initial management of the hypothermic patient and cold injuries
- Rewarming techniques
- Difficulties in diagnosing death in the hypothermic patient

**The pathophysiology of hypothermia and cold injury are covered in Appendix 15.1 at the end of the chapter.**

### **Definition and classification**

**Hypothermia is defined as the lowering of core temperature to below 35°C. When considering signs, symptoms and treatment it is useful to classify it as mild (35-32°C), moderate (32-28°C), or severe (less than 28°C). These temperature ranges are arbitrary and there are other classifications.**

### **DIAGNOSIS, CLINICAL FEATURES AND SIGNS**

Hypothermia should be suspected from the clinical history and a brief external examination as the cues listed in Box 15.1. The symptoms are often non-specific and may easily be overlooked in the early stages, or if other major injuries receive the entire diagnostic focus. The trauma team must always remember hypothermia as one silent threat to the trauma patient. A core temperature  $< 34^{\circ}\text{C}$  may adversely influence the coagulation cascade and the circulatory organs, resulting in increased bleeding, acidosis and infections. Clinical symptoms depend on the body core temperature, but also on the rate of cooling. It is important to remember that there are wide variations in the individual response to hypothermia, and a specific symptom may be absent in one patient but pronounced in another (Box 15.2).

**Box 15.1: Clues that should raise the team leader's suspicion of hypothermia**

Pre-hospital information	During the "5 second" round
<ul style="list-style-type: none"> <li>• Cold ambient temperature</li> <li>• Immersion</li> <li>• Victim trapped</li> <li>• Long time to arrival in hospital</li> <li>• Intoxication</li> <li>• Children</li> <li>• Diabetic</li> </ul>	<ul style="list-style-type: none"> <li>• Wet clothes</li> <li>• Little or no clothes</li> <li>• Shivering</li> <li>• Obvious major injuries</li> </ul>

**Box 15.2: Signs and symptoms of hypothermia**

**Mild: 35 - 32°C**

- Pale and cold
- Shivering
- Increased:
  - respiratory rate
  - pulse rate
  - blood pressure
- **Conscious**

**Moderate: 32 – 28 °C**

- **Pale and cold**
- **Minimal shivering**
- Reduced:
  - respiratory rate
  - pulse rate
  - blood pressure
- ECG changes
- Confused, slurred speech, lethargic

**Severe: below 28 °C**

- Pale and cold
- No shivering
- **Hypoventilation**
- Severe bradycardia or arrhythmia
- Hypotension
- Coma, areflexia
- Dilated pupils

As the core temperature falls below 32°C sinus bradycardia, resistant to atropine, develops that eventually progresses to atrial fibrillation with a slow ventricular response (Fig. 15.1), nodal rhythm, ventricular fibrillation (VF) and finally asystole. Below 28°C, the myocardium becomes very sensitive and even the slightest stimulus such as moving the patient may trigger VF. This sensitive, hypothermic heart has been attributed to excessive sympathetic stimulation, temperature gradients, electrolyte and acid base disturbances, myocardial hypoxia and cold induced myocardial electrical conduction. If VF is triggered, it is usually resistant to defibrillation until core temperature is over 30°C. Box 15.3 lists some of the ECG signs that can be seen in a hypothermic patient. Cerebral metabolism is reduced by the fall in temperature (7-9% per 1°C decrease), gradually reducing the level of consciousness. This reduction in the level of consciousness with loss of gag and cough reflexes, place the victim at increased risk of aspiration and if there is prolonged immobility (lying in one position while unconscious) may also cause rhabdomyolysis, hyperkalaemia and subsequent acute renal failure.

### **Box 15.3: ECG changes associated with decreasing temperature**

- Shivering
- J waves
- Sinus bradycardia
- Atrial fibrillation
- Ventricular fibrillation
- Asystole

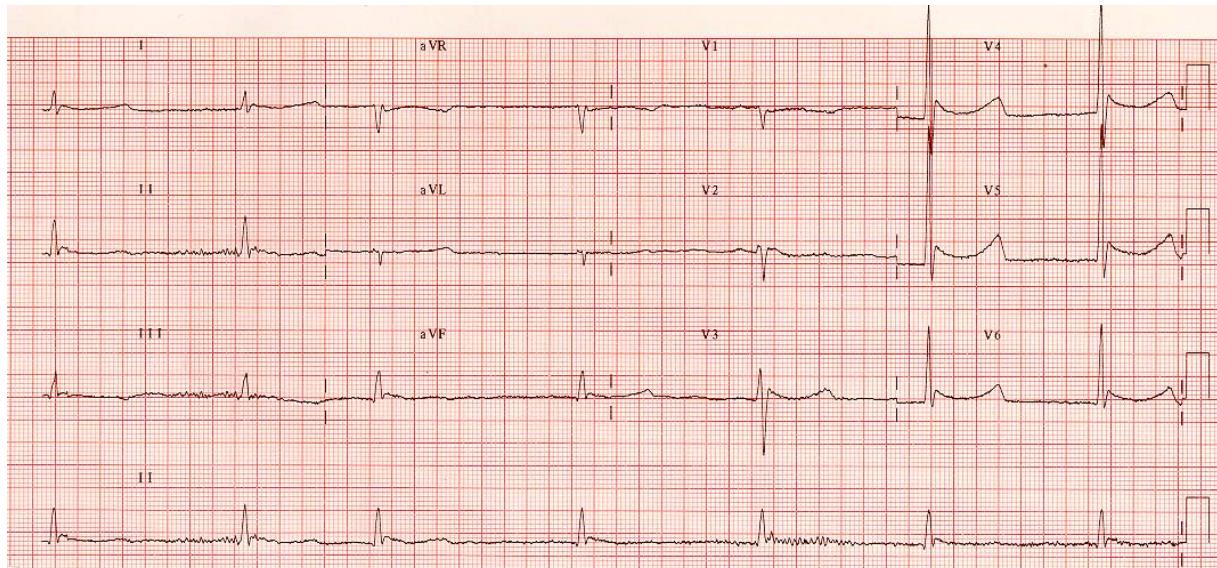


Fig. 15.1: 12-lead ECG of hypothermic patient (core temperature 23C) showing slow atrial fibrillation and J waves (humps at the end of the QRS complex) (Courtesy Dr J Nolan).

#### **Initial management of the hypothermic patient**

The same principles as described in Chapter 1 apply equally to the hypothermic patient.

#### **Primary survey and resuscitation**

Open, clear, and maintain a patent airway and administer oxygen. If there is inadequate or no spontaneous respiratory effort, commence ventilation with a high concentration of oxygen. If tracheal intubation is indicated, it must be performed as carefully as possible, particularly in the presence of severe hypothermia, to avoid the risk of inducing VF. However, in most patients, the advantages of a secure airway and adequate oxygenation outweigh the potential risk of VF secondary to intubation. The cervical spine must be immobilised appropriately; a wet, hypothermic patient should raise the suspicion of a diving injury until excluded. Oxygen should be preferably warmed (40–46°C) and humidified. Obtaining a good quality ECG from regular

adhesive skin electrodes can be difficult due to the low conductivity of cold skin. Placing a subcutaneous needle tangentially through the central part of the ECG electrode (where the gel is) into the subcutaneous tissue can be a useful solution to this.

In severe hypothermia it may be difficult to identify the presence of a pulse, and therefore a major artery must be palpated for up to a minute whilst looking for signs of life before concluding that there is no cardiac output. Capnography or a Doppler ultrasound probe may also be helpful in these circumstances. Peripheral venous access may be difficult and early consideration should be given to alternative techniques, for example a central line or intraosseous infusion (Chapter 5). All fluid must be warmed and care taken with the rate and volume of administration as the cold myocardium is intolerant of an excessive fluid load.

If the patient requires CPR the tidal volumes and rates for chest compression are the same as for a normothermic patient although chest wall stiffness may make this more difficult to achieve. Arrhythmias tend to revert spontaneously with warming and usually do not require immediate treatment. Bradycardia may be physiological and may generate sufficient blood flow for reduced metabolic demands in severe hypothermia. Cardiac pacing is not indicated until after rewarming. Defibrillation may not be effective if the core temperature is less than 30°C. If the patient does not respond to three initial defibrillation attempts, subsequent defibrillation attempts should be delayed until the core temperature is above 30°C.

Central vascular access is preferable for the administration of drugs as they may pool when given peripherally due to venous stasis. However, it must be remembered that the central venous catheter must not be advanced into the right atrium because the increased sensitivity of the hypothermic heart. Drug metabolism is reduced and accumulation can occur to toxic levels in the peripheral circulation if drugs are administered repeatedly via this route in the severely hypothermic victim. Drug efficacy is also reduced and the use of inotropes and anti-arrhythmic drugs is unlikely to be helpful in severe hypothermia until rewarming has been established. These patients need urgent transfer to a critical care setting where full invasive haemodynamic monitoring and effective rewarming can be established. The effects of inotropic drugs and anti-arrhythmic drugs can then be carefully titrated during the warming process.

The patient's neurological state will be affected by the degree of hypothermia and as a result, may lead to an underestimation of their level of consciousness. Alternatively a reduced level of consciousness due to head injury may be wrongly attributed to the patient's temperature. Clearly as the patient rewarms, their conscious level should improve. Any failure to do so should raise

the suspicion of a co-existing head injury. The injury mechanism may also warrant CT scanning of the head to exclude intracranial injury.

During the primary survey, the diagnosis of hypothermia is confirmed by measuring core temperature using a low reading thermometer. No single site is ideal for temperature measurement but the easiest sites are rectal, bladder or oesophageal. If rectal temperature is measured there is often a significant lag between changes in core temperature and the measured temperature. Measuring tympanic temperatures is unreliable, and should be avoided. The accuracy of thermometers and temperature ranges varies, so it is important to be consistent with the site of monitoring when tracking temperature changes and to take repeated readings.

One easy but often forgotten measure is to remove wet clothing from the patient, or wet blankets from around and underneath them as soon as possible to reduce further heat loss. This is best achieved during the log-roll as one examines the back of the patient.

### **Secondary survey**

As with all victims of trauma, a thorough head to toe examination must now be performed to identify any life-threatening injuries. Remember to take an AMPLE history and determine if there is a predisposing condition that may be adding to the hypothermia (e.g. diabetes, alcohol ingestion). It is also at this point that concerted efforts will be made to reduce further heat loss and start rewarming the patient.

### **Rewarming**

The specific management of hypothermia depends on the level of consciousness, cardiopulmonary function and the core temperature. If advanced information is given of the arrival of a hypothermic patient, every effort should be made to receive them into a warm environment. The temperature of the resuscitation room should be raised and all drafts prevented. The team need to also ensure an adequate supply of warm fluids, blankets and access to warming devices.

Rapid rewarming may cause an increase in cardiovascular instability due to fluid and electrolyte shifts. Some believe that victims should be rewarmed at a rate that corresponds with the rate of onset of hypothermia. This is difficult to gauge in practice however, and rewarming a patient too slowly may increase the time that the patient is vulnerable to the harmful effects of hypothermia.

There are three main ways of warming a patient; passive external, active external, or active internal (core rewarming). Passive external warming can be achieved with blankets, hot drinks and a warm room. It is suitable for conscious victims with mild hypothermia, but a disadvantage is that the body core temperature rises slowly. Only supervised victims with mild hypothermia who are otherwise well should be placed in a warm bath. A hot shower whilst standing may cause fainting due to rapid vasodilatation. In moderate hypothermia, warming needs to be more active. The use of warm air blankets (e.g. "Bair Hugger" or "Warm Touch") together with warm intravenous fluids and warm humidified oxygen is probably the most efficient way of active external rewarming. This technique is effective primarily in patients with an intact circulation that can return peripherally warmed blood to the core. However it is important to be aware of the potential for a drop in core temperature after warming started. This "after drop" results from cold blood returning from the increased or re-established peripheral circulation, is difficult to avoid and may complicate the cardiac stability of the patient.

In severe hypothermic patients with profound haemodynamic instability or cardiac arrest, internal active rewarming measures are required. A number of techniques have been described although there are no clinical trials of outcome to determine the best method. Techniques include the use of warm humidified gases along with gastric, peritoneal and pleural lavage with warm fluids at 40°C. The most effective method in these patients is active internal rewarming using extracorporeal devices such as cardiopulmonary bypass, because it also provides circulation, oxygenation and ventilation, while the body core is gradually warmed. This method is highly effective and may increase core temperature by 1-2°C every 3-5 minutes. In practice, facilities for cardiopulmonary bypass are not always available and a combination of methods may have to be employed. An alternative method of extracorporeal warming is to utilise continuous veno-venous haemofiltration. The extracorporeal circuit should be warmed and replacement fluids heated to 40°C. This is only possible in those patients with a circulation.

During rewarming, patients are likely to require large amounts of fluids as their vascular space expands due to vasodilation. Blood loss and fluid extravasation may further increase the fluid needs during hypothermia and rewarming. Vasopressors, for example noradrenaline (norepinephrine), may be required to maintain an acceptable blood pressure and to limit the total fluid load. All intravenous fluids should be warmed prior to administration. Careful haemodynamic monitoring, according to local protocols, is important and these patients are best managed in a critical care environment.

Hypothermic patients often experience respiratory complications after rewarming. Fluid overload, aspiration and cardiac failure may explain these findings, but most of these patients will require a period of ventilatory support. The first option in spontaneous breathing patients is external continuous positive pressure (CPAP). This can be intermittent or continuous, and can improve oxygenation. An alternative non-invasive ventilatory treatment is BiPAP (bi-level positive airway pressure) if the patient is conscious. Ultimately, some patients will need tracheal intubation and controlled ventilation, often with different ventilatory strategies. This is best achieved in a critical care environment.

### **Associated injuries**

While attempts are being made to warm the patient, it is essential that a thorough clinical examination is performed to detect any injuries not identified during the primary survey. This is carried out as described in Chapter 1. If the patient is moved to a critical care area before this has been completed, it is the team leader's responsibility to ensure that this information is imparted to those now caring for the patient.

### **Investigations**

Investigations must include regular measurements of arterial blood gases and electrolytes, particularly potassium, as rapid changes (hyperkalaemia) can occur during the rewarming period. Blood gas analysers measure patient blood gas values at 37°C, and if corrected for the patient's temperature, tend to be lower as gases are more soluble in blood at lower temperatures. To interpret corrected values, results would have to be compared with the normal value for that particular patient temperature. It is therefore easier to interpret uncorrected arterial blood gas measurements, as it is then only necessary to compare them with the well-known normal values for 37°C. This also simplifies comparison of results from serial blood gas samples during rewarming.

Hyperglycaemia is often associated with hypothermia as a result of the reduced metabolic ability of the cold tissues and the body's normal stress response. Insulin must not be administered as this can exacerbate the normal fall during rewarming and render the patient dangerously hypoglycaemic. On the other hand it is documented that hyperglycaemia is bad for the injured brain, and that blood glucose control in intensive care patients is associated with improved outcome. Repeated estimations are therefore required and intravenous glucose may be required in patients whose condition is due to enforced immobility and exhaustion. Blood cultures, thyroid function tests, alcohol levels and a toxicology screen should also be performed.

### **Hypothermia and blood loss**

When temperature falls to below 34°C, coagulation is impaired leading to an increased blood loss if the patient has other injuries. This will again reduce the tissue perfusion, aggravating acidosis and the need for more fluid replacement. Cold fluid and blood will then in turn lower the body temperature, and thereby keep the vicious circle going. The lethal triad of hypothermia, acidosis and coagulopathy is well known and dreaded among trauma surgeons. The only strategic option is to reduce surgical interventions to damage control measures and to restore temperature, circulatory and respiratory homeostasis before definitive surgery is performed.

### **PROGNOSIS**

A full recovery without neurological deficit is possible after prolonged hypothermia even when associated with cardiac arrest, as hypothermia confers a degree of protection to the brain. However, an extremely low core temperature and significant co-morbidity are both predictors of a poor outcome. In severe hypothermia the decision to rewarm the patient on an extracorporeal device must be taken as early as possible and specialised personnel must be called in if not on site. The risk of intra-cerebral haemorrhage in a trauma patient as a complication of the extracorporeal warming must also be kept in mind, since some degree of anticoagulation will result from this treatment.

Hypothermia may mimic death so beware of pronouncing death in the hypothermic patient. Outside hospital, if practical, treatment and resuscitation should commence to allow transfer to hospital. Death should only be confirmed if the victim has obvious lethal injuries or if the body is frozen making resuscitation attempts impossible. Severe hypothermia may protect the brain and vital organs from the effects of hypoxia by slowing metabolism, reducing oxygen needs and decrease free oxygen radical formation. Warming may also reverse arrhythmias associated with hypothermia. Fixed dilated pupils and stiffness can be due to hypothermia. In a patient with cardiac arrest found in a cold environment, hypothermia may be the primary cause but it is difficult to distinguish from secondary hypothermia after cardiac arrest due to myocardial infarction or other causes. Patients who have suffered a non-asphyxial, accidental hypothermic cardiac arrest, have a far better prognosis.

If the first blood tests reveal extreme hyperkalaemia and acidosis this also is a bad prognostic sign. Ideally death should not be confirmed until the patient has been rewarmed or attempts at rewarming have failed to raise core temperature. This may require prolonged resuscitation. In the hospital setting the clinical judgement of senior team members should determine when resuscitation should stop in the hypothermic arrest victim.

**There is not a prescriptive temperature below which death should not be diagnosed**

## **Local cold injury**

### **Frostbite**

This local cold induced tissue injury is normally restricted to extremities and head (nose, ears, chin, and cheeks). Intensity and duration of cold exposure before circulation is restored will influence severity. Superficial tissues freeze, with the formation of ice crystals between and within cells. This leads to dehydration, cellular injury and ultimately destroys the tissue. In addition vasoconstriction will reduce blood flow in the adjacent tissue, and the whole area begins to freeze. Frostbite can be classified in superficial or deep injury, determined before the body part has been thawed.

In superficial frostbite, the frozen area is numb, white and firm when palpated gently. After rewarming, blisters can occur during the following 24-36 hours. Blisters and swelling will disappear after a couple of weeks, leaving a red and extremely tender skin. In deep, unthawed frostbite, the body part is hard and solid. The skin, subcutaneous tissues and even deeper tissue are injured. Huge blisters will form and swelling will be severe and long-lasting. After some time there will be a demarcation line between the injured and normal body parts. The tissue will turn black, dry and shrivelled. This dead tissue can also be infected and become wet, with the whole body part inflamed.

### *Treatment*

Rapid rewarming with warm water (37-40°C) is the best method. Warmer water will be detrimental and should be avoided. This usually takes about 20 minutes. Rewarming will be painful and the patient must receive analgesics. After rewarming the injured area must be covered in sterile dressings. Surgical revision of severe frostbite will be required, and is undertaken only after several weeks.

## **Summary**

Hypothermia is very common in trauma patients. The same principles as described throughout this book for primary survey and initial management apply to victims of hypothermia. Measures to reducing heat loss should be instituted already on the scene and continue en-route and after

arrival in hospital. Passive or active external methods of warming are needed for victims of mild or moderate hypothermia. Hypothermia can seriously complicate the fate of a trauma patient because of negative effects on the coagulation system. In severe hypothermia and cardiac arrest active internal methods such as cardiopulmonary bypass may be needed. Death should be diagnosed with care in victims of hypothermia.

## **Appendix 15.1: Aetiology and pathophysiology of hypothermia and cold injury**

The causes of hypothermia are numerous, and the failure to recognise the condition is associated with increased morbidity and mortality. Humans regulate their body temperature very accurately and even minor variations in the temperature of vital organs can lead to psychological and physiological disturbances. Under normal circumstances, the temperature of the environment is sensed by specialised nerve endings in the skin and the body temperature by nerves in the great vessels and viscera. By balancing heat loss and production, the hypothalamus controls the body temperature. The commonest cause of hypothermia is heat loss. This occurs as a result of:

- *Conduction:* The direct transfer of heat between a warm object to a cooler one, for example when lying on a cold floor or in immersion incidents.
- *Convection:* Heat is transferred to surrounding air or water which moves away taking the heat with it (wind chill).
- *Radiation:* The loss of heat by the emission of infrared radiation from a warm body to a cooler one. Normally, this is the main method of heat loss accounting for up to 60% (primarily from the head or non-insulated body areas).
- *Evaporation:* Liquid water on the surface of the skin, wet clothing, or a wound turns to water vapour. This is an energy requiring process (latent heat of vaporisation) and this comes from the body. The more liquid that evaporates requires more heat energy from the body, which then cools.

**With their larger surface area to volume ratio, children have a greater rate of heat loss by all these mechanisms, while vasodilatation from any cause increase heat loss by conduction, convection and radiation.**

**Those with normal thermoregulation are at high risk of hypothermia when exposed to cold environments, after immersion in cold water, or exposed to wet and windy conditions. Body heat is lost rapidly in these situations via the mechanisms described above. Still air is a good insulator and consequently when blown away by the wind, this insulating layer is lost and body temperature falls.**

**The combination of temperature and wind is called the wind chill factor**

Water has an even greater thermal conductivity than air (up to 25 times) and wet clothes and damp conditions significantly increase the speed of heat loss. Trauma patients may be wet as a result of rain, lying on wet ground or immersion and the failure to take into account this

accelerated heat loss pre-hospital partly explains the fact that many trauma patients are hypothermic when they arrive at hospital. Heat production can also be reduced, usually as a result of a decreased metabolism, for example unconsciousness, hypothyroidism and hypopituitarism, while the elderly have a reduced capacity to increase heat production. Reduced heat production alone is not a common cause of hypothermia, but it is often a contributing factor to hypothermia.

The body's main method of reducing heat loss is by vasoconstriction, but drugs, particularly alcohol, significantly reduce this. The process is usually supplemented by behavioural responses, such as putting on extra clothing, avoiding the cold and reducing surface area by curling up. Again this may be inhibited as a result of the influence of drugs. Shivering increases the metabolic rate (up to 5 times) and increases heat production but at a cost of greatly increasing the oxygen consumption and carbon dioxide production. Unfortunately, this mechanism is lost as the core temperature falls below 33-30°C.

Several studies have shown that trauma victims have lower core temperatures than expected, particularly in cases of severe injury. The cause of this is probably multifactorial and includes environmental conditions, metabolic changes, blood loss, infusion of cold fluids, or the injury itself. Whatever the cause, trauma patients with a low core temperature have a worse prognosis and therefore every effort must be made to prevent further falls in temperature before and after arrival in the emergency department.

## Chapter 16

# **Analgesia**

## **Objectives**

At the end of this chapter the reader should:

- Understand the adverse effects associated with pain.
- Understand the assessment of pain.
- Understand the variety of techniques that can be used manage pain.

The physiology of pain, additional pharmacology of analgesics and local anaesthetic techniques are covered in appendices 16.1, 16.2 and 16.3 respectively at the end of the chapter.

## **Introduction**

Pain is a complex phenomenon that is difficult to describe and measure. The perception of pain involves sensations, feelings, and emotions that are unique and specific to an individual. Pain cannot easily be articulated or interpreted by another person, increasing the likelihood of assessment subjectivity. It is defined by the International Association for the Study of Pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage." Pain is a common human experience that functions as a protective mechanism to both external and internal stimuli. The inability to communicate verbally does not negate the possibility that an individual is experiencing pain and is in need of appropriate pain-relieving treatment.

Assessment, and subsequent treatment, of pain must be carried out early in the management of the trauma patient. It is appropriate to consider administration of analgesia as soon as the primary survey is completed. In addition to pain resulting from the injury, many procedures carried out in the early stages of trauma management are themselves painful. It is very important to administer adequate analgesia to cover these procedures. Very few interventions are so urgent as to excuse administration of appropriate analgesia. This is particularly true with children, both from a humane point of view and also to maintain the patient's trust. Intravenous cannulation in all children, and certainly in adults with cannulae larger than 20G, requires local anaesthesia prior to the procedure. The excuse that local anaesthesia makes intravenous cannulation more difficult is unacceptable and a reflection of poor cannulation technique.

## **The adverse effects of pain**

Patients with severe and complex injuries represent a challenge to the trauma team; their pain is often derived from multiple sources. It may be very severe and can often evoke the strongest of physiological and emotional responses that may exist for a significant length of time. Unrelieved acute pain, however, may lead to chronic pain that may result in considerable morbidity, with longer in-hospital stays, more frequent hospital readmissions, visits to outpatient clinics and emergency departments and increased costs. Pain is a priority in patient management, not least for the relief of suffering, but also because of the adverse pathophysiological responses (see below) that it generates. These responses may result in exacerbation of pain, difficulty in treating the patient, worsening of the patient's condition and long term morbidity:

**Nausea and vomiting.** Pain is a powerful stimulus of the vomiting centre, both through inflammatory mediators and neural afferents from the cortex. Nausea and vomiting in trauma patients may be more effectively treated with adequate analgesia rather than anti-emetic drugs.

**Hyperventilation.** This is a common response to pain, results in respiratory alkalosis and can lead to hypocalcaemia with carpopedal spasm. Hyperventilation also shifts the oxygen dissociation curve to the left/right and may reduce overall tissue oxygen delivery

**Increased oxygen consumption.** Pain increases endogenous catecholamine release that acts to increase basal metabolic rate and oxygen consumption. Reduced oxygen delivery and increased oxygen consumption may jeopardise tissues where perfusion is already compromised.

**Increased intracranial pressure.** Agitation increases intracranial pressure which may reduce overall cerebral perfusion pressure and compromise tissues in watershed areas.

**Increased stress response.** Although not of immediate detriment, ongoing pain results in a stress response that causes an overall negative nitrogen balance, with muscle breakdown. There is evidence that this may adversely affect the patient's recovery from their injury.

**Psychological changes to chronic pain.** Inadequately treated pain is associated with long term mental health problems including post traumatic stress disorder, increased risk of drug abuse, insomnia, self-harm and suicide.

## **Clinical assessment of pain**

Quantifying the intensity of pain is an essential part of initial and ongoing pain assessment. The relationship between the trauma nurse and patient affords the ideal opportunity to assess the patient's pain, plan their care, and intervene with appropriate and timely analgesia. An accurate assessment will also increase the effectiveness of treatment. The picture of pain may include:

- Mechanism of injury
- Patient behavior (e.g. facial expression, mobility)
- Physiological response (e.g. tachycardia, sweating)
- Patient report of pain (assessment tool)

### **Assessment tools**

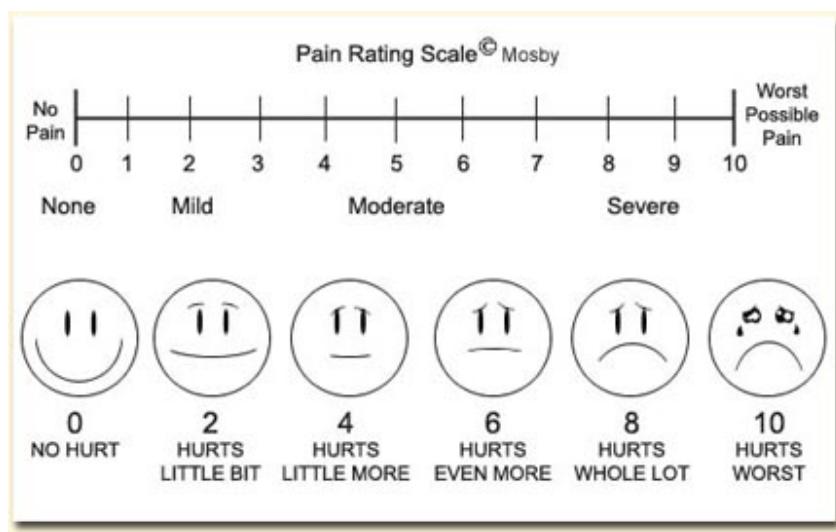
Pain assessment tools have been used in the treatment of chronic pain for some time, but acute care settings have been slow to appreciate the proven advantages. Behavior scales have been introduced into emergency care to offset the subjectivity of the assessment and provide practitioners with a more objective means of establishing pain levels. In the pre-hospital or Emergency Department setting, any tool needs to be quick, accurate, and flexible for varying situations and ages.

The verbal rating scale using groups of words that either describe pain, for example burning, aching or stabbing or its severity for example mild, moderate, severe are common, as they are quick and adaptable. These can be enhanced if used in conjunction with a visual analogue scale. The simplest and quickest consists of a line with a scale ranging from zero to 10, where zero represents "no pain" and 10 the "worst possible pain". The scale may also indicate the parameters associated with mild, moderate and severe pain and patients participate by marking their level of pain on the scale. A horizontal line is preferred as vertical lines indicate a rapid increase and may reduce the sensitivity of the tool (Fig. 16.1). A verbal analogue scale uses similar principles, but asks the patient for a verbal estimate between 0 and 10.

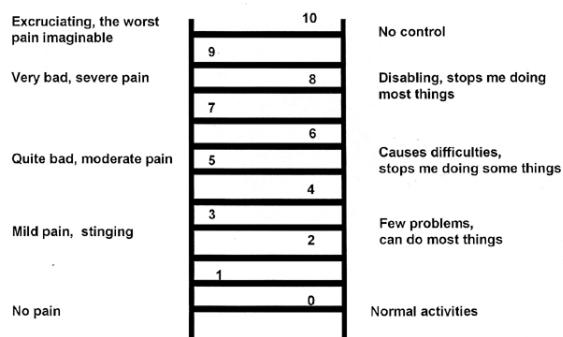
Adequate pain assessment in the child poses a challenge, especially in the very young. Words used to describe the levels can be altered to suit the age group but is of little use to the preverbal child. Sensory, visual pain scales have proven to be effective in children over the age of three (and in patients with learning difficulties). A pictorial rating scale, using a range of happy, smiling faces, to sad, crying faces serves as a quick reference and useful mode of assessment (Fig. 16.1). In addition, it is also useful to observe a child over time and note any

refusal to play or move normally. Acute pain relief in children is possible with accurate assessment, timely analgesia and calm parents and carers.

**Fig. 16.1: Visual analogue and pictorial rating scales for pain assessment in adults and children**



The Manchester Triage System (MTS) (1997), used in over 80% of emergency departments in the UK, combines three assessment tools into one “Pain Ruler” (Fig. 16.2).



**Fig. 16.2: Manchester pain ruler**

## **The management of pain**

A team approach is required to address the psychological, pharmacological and physical needs of patients in order to successfully control their pain, the “three Ps of pain management”.

### **Psychological management**

As anxiety increases, the pain threshold lowers, exacerbating the pain response. In contrast, feelings of confidence and control have an inhibitory effect. This effect can and should be utilized by the trauma team members to limit anxiety and reduce pain by ensuring a calm and reassuring environment, providing frequent reassurance, and demonstrating effective teamwork. Furthermore, the individual response is also related to the acute experience and the events surrounding the injury. Pain associated with major injury in a dangerous and highly charged situation, such as escaping from a burning building, may be ignored or overlooked. Pain associated with relatively minor injuries sustained during civil disturbance, where an innocent onlooker becomes a victim may appear excessive.

Previous painful experiences modify the neural systems creating a memory of past events and enhancing behavior. Repeated exposure to noxious stimuli lowers the patients' threshold and increases their sensitivity. For example, a chronic back pain sufferer who sustains an acute injury may well experience an increased response to the pain stimulus. Once nociceptors have been sensitised, only slight stimuli are needed to produce a significant pain response or hyperalgesia. Early assessment and appropriate control of pain can prevent the development of hyperalgesia and reduce the overall long-term pain response.

An individual's response to illness or injury is also influenced by their cultural background, from which they learn or are taught specific behavior. This influence can be manifested by a stoic acceptance seen particularly in the elderly population who often under report pain or conversely an open display of pain often seen in the very young. Whilst there is no evidence that tolerance is different in various ethnic or cultural groups, outward expression of pain varies enormously. This influence of culture and beliefs also extends to trauma team members who may impose their own values and personal influence when evaluating the pain.

To avoid personal bias in pain assessment, the multidimensional nature of pain, including physical, emotional, social, and environmental components must be considered. The patient's contribution should be valued and included whenever possible.

#### *Specific psychological objectives*

*Reduce fear:* Trauma patients who are conscious are generally frightened by the events associated with the initial injury, fearful of the long-term effect of their injuries and anxious about investigations and tests that may be necessary. The way in which the trauma team work has an important affect on the patient's pain. Confidence is essential and explanation and justification

at each stage in an appropriate and sensitive manner may be as effective as pharmacological adjuncts. Understanding and reassurance will go a long way in playing a part in helping contribute to relief of anxiety and its effects on pain. The presence of a friend or relative may have a calming and reassuring effect on the patient. This is particularly true for children, where the presence of the parent or carer should be considered almost mandatory. Separating parents from their children can also be as equally stressful for the parent as for the child!

*Reduce noise:* A quiet calm environment will reassure the patient and reduce their anxiety. If the patient has a serious condition, do not discuss it with colleagues in earshot of the patient unless the patient has already been informed.

#### *Paediatric patients*

A variety of behavioral responses are seen in children, some of which are cultural, including irritability, grimacing, guarding, pushing away, withdrawal, crying and overreacting to minimal stimuli. Although these may simply be a response to pain, they may also indicate fear and anxiety as a result of previous unpleasant experiences in hospitals. Children who are fearful or mistrust health care workers are more anxious, which ultimately leads to an increased pain response. Trauma team members must be aware of child development in relation to chronological age in order to understand the expected behavior and language skills, thereby improving communication with a distressed child, gaining their trust and cooperation.

#### *Elderly patients*

This group of patients often present with pre-morbid conditions that complicate, and compound, the acute trauma phase. Many elderly patients live with a degree of pain daily and accept this discomfort as part of the ageing process. Again the observation of pain behavior is important, as is the discussion of the options for pain relief. The latter may be pharmacological, but could also include appropriate comfort measures such as appropriately positioning and supporting the patient.

### **Physical treatments**

Many factors contribute to the subjective perception of pain. Although severe pain will generally require the use of drugs, consider the following factors that may reduce the overall analgesia requirements.

**Fracture stabilization:** Unstable fractures are extremely painful. Early splinting not only is effective at reducing the severity of pain, but it will also reduce fracture-associated bleeding, the risk of neurovascular damage, tissue inflammation and fat emboli.

**Surface cooling:** of an injured limb, particularly around joints, will help to reduce oedema and pain. This is best achieved using commercially available ice packs, which are stored at 5°C and reusable. Ice should never be applied directly to the skin.

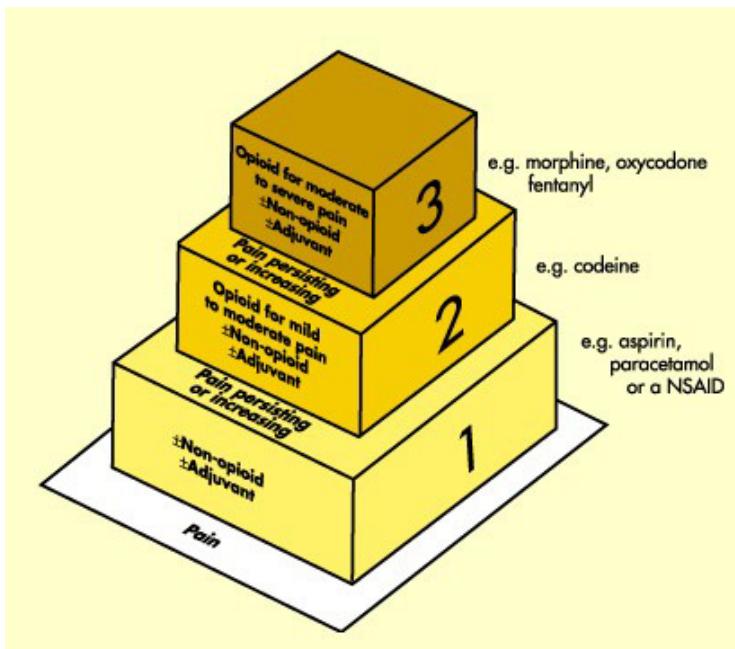
**Wound dressings:** Superficial burns are very painful and hypersensitive to touch, including exposure to air currents. Covering the burned area with a sterile dressing will reduce pain and help protect from contamination. A variety of propriety dressings are available in an emergency, however, PVC is cheap, sterile and non-adherent. PVC dressings should be placed in longitudinal strips to prevent limb constriction.

**Spinal boards:** are uncomfortable even after a short duration. Moving patients to softer surfaces reduces pain and reduces the risk of pressure sores.

**Hypothermia:** Patients suffering prolonged entrapments or even short duration exposure to a cold environment may be hypothermic on arrival at hospital. Shivering increases pain intensity and early warming, preferably using a forced-air warming blanket, should be instigated as soon as possible. Although full exposure of the trauma patient is usually necessary, cover the patient as soon as possible. Even a simple cotton sheet will halve the patient's thermal losses.

### **Pharmacological management**

Drugs are the mainstay of providing pain relief after trauma and a wide variety of agents can be used. The general principles of pain relief are a step-wise escalation in analgesic administration. This 'analgesic ladder' (Fig. 16.3) commences with a simple analgesic, such as paracetamol, progressing to the addition of a non-steroidal anti-inflammatory drug and then an opioid if necessary. Analgesic drug combinations generally have a synergistic effect and reduce the overall dose needed of individual drugs. This is of particular benefit with opioids where the reduction in total dose reduces the numerous side effects of this class of drugs. In patients with severe pain, a stepwise approach is inappropriate, with opioid-based analgesia being required immediately. Once pain is controlled, other classes of drug can then be added which will reduce the opioid requirements; effectively climbing down the steps rather than up.



**Fig. 16.3 : The WHO analgesic ladder for pain relief. (Source: ABPI)**

### Opioid analgesics

Opioids and morphine in particular, are regarded as the gold standard in analgesic efficacy. No other drug is able to match opioids in their analgesic properties and they are therefore the drugs of choice in all patients suffering acute, severe pain. However, many studies have documented the inadequate analgesia given to both adult and paediatric trauma patients. The main reason for this is concern over the possible side effects, in particular, respiratory depression. Typical effects seen on ventilation after their use are shown in Box 16.1.

#### Box 16.1: Typical changes in PaCO<sub>2</sub> seen after the administration of opioids

PaCO <sub>2</sub>	Comment
Low	In the conscious patient, consistent with pain and/or anxiety. If in pain, it is likely to be safe to proceed with opioid titration
Normal	Commonest situation - proceed or continue with analgesia
High	Indicates that ventilation is inadequate and need to distinguish between: Mechanical cause of inadequate ventilation e.g. pneumothorax or flail segment – repeat primary survey Pain from chest trauma – administer effective analgesia Central respiratory depression e.g. opioids, head injury – repeat

## primary survey

**Overcaution regarding the side effects of opioids is one of the major obstacles to analgesia being achieved. Any patient conscious enough to complain of pain is somehow off suffering life-threatening respiratory depression.**

### Individual drugs

#### Morphine

Most patients will get rapid and effective analgesia, with following intravenous morphine in an appropriate dose, although the full analgesic effect may take 15-20 minutes to evolve. Morphine is available for administration by a number of other routes but it must be remembered that in patients with renal failure the metabolite, morphine-6-glucuronide, is more potent than morphine and will accumulate.

#### Intravenous analgesia

Intravenous administration of opioids is the quickest route to deliver effective analgesia. Small repeated doses are necessary to achieve adequate analgesia without overdosing the patient. The usual dose required ranges from less than 4mg in the frail elderly patient up to 50mg in younger opioid-tolerant patients. The safest method of giving morphine is by diluting it; in an adult, a 10mg ampoule diluted to 1mg/ml using normal saline. Incremental doses of (0.015 – 0.03mg/kg) can then be given (1 - 2 mg). Although the same dose is used for children, it is often easier to dilute 1mg to 10ml (0.1mg/ml). In the frail, shocked or elderly, the dose must be reduced. Intravenous opioids work quickly and subsequent doses can be given after 3 - 5 minutes, (longer in the elderly with slower circulation). It is important that observation of conscious level, respiratory rate, pulse and blood pressure are performed at 5-minute intervals and continued for at least 15 minutes after the last dose.

#### Intramuscular analgesia

This route has a slower onset of action than intravenous administration and in the trauma patient; poor peripheral blood flow will further reduce absorption making it difficult to achieve initial adequate pain control. However, when opioid requirements have been established it may be the preferred method of maintaining analgesia, particularly in elderly patients unable to understand and use PCA (see below). There is little justification for using the intramuscular route for analgesia in children; intravenous access often needs to be established anyway and the intranasal route has been shown to be a very effective alternative to intramuscular injections.

### Oral route

Even in the absence of nausea and vomiting, gastric stasis and reduced gastrointestinal blood flow reduce the rate of oral absorption of drugs and make it an unsuitable route for acute control of pain. Once patients are stable and able to take oral medication, the oral route however is an ideal way for continuing analgesia. Severe pain may need high oral doses of opioids and they can be continued after discharge from hospital. There is a potential for misuse in some populations.

### Intranasal

Intranasal administration of opioids is particularly useful in children where it avoids painful intramuscular injections. Nasal diamorphine is preferred method of pain relief in children and teenagers presenting to emergency departments in acute pain with clinical fractures. Pain relief is achieved quicker with intranasal spray than with intramuscular morphine. A suitable dose of nasal diamorphine is 0.1mg/kg, mixed to a small volume (0.1-0.2 ml) squirted up each nostril as the child reclines at 45 degrees.

### Patient Controlled Analgesia (PCA)

This is best thought of as a method of maintaining analgesia rather than attaining it. Having achieved an agreed level of analgesia using the technique described above, the PCA patient device can allow the patient to self medicate as the initial doses wear off.

#### Terminology for PCA

- Loading dose – quantity of drug to establish analgesia (as above)
- Bolus dose – incremental dose of drug by patient demand to maintain analgesia
- Lockout interval – duration of time after a successful bolus dose during which the device will not respond to further requests

Typical settings for an adult are:

- Bolus dose of 1mg
- Lockout interval of 5 minutes

Opioid requirements are very variable between patients and therefore the maximum dose (for example, amount permissible in 4 hours) is an unnecessary restriction. Opioid tolerant patients may need high bolus doses, for example 4mg but usually the lockout interval remains constant. Many elderly patients will not or are unable to use PCA effectively, even after a seemingly good explanation and other routes must be used. Other opioids, for example fentanyl, may be also used by the PCA route. For PCA purposes 20 mcg fentanyl is equivalent to 1 mg morphine.

### *Side effects of opioids*

- Nausea: often exacerbated by the delay in gastric emptying as a result of trauma.
- Respiratory depression: Respiratory rate may fall towards normal levels with effective analgesia, changes in respiratory rate are more helpful than specific values. Where there is doubt about the effects of opioids on central respiratory drive, analysis of arterial blood gases can help. Respiratory depression is best monitored using the respiratory rate. Normal oxygen saturation is not an adequate monitor, particularly for patients being given supplementary oxygen.
- Pruritus and urinary retention: usually occurs with high doses.
- Sedation: This is usually minimal with judicious titration of opioids. In patients with a head injury, a reduction in level of consciousness beyond a calming and analgesic effect will be detected by an appropriately performed assessment of the patient's Glasgow Coma Score.

### *Concerns over use of opioids*

- The potential to mask pathology: This is now known to be unfounded and relief of severe pain allows more effective clinical examination in a less distressed patient.
- Miosis: This is a result of a central effect on the brainstem. It does not mask the development of pupillary abnormalities associated with intracranial pathology. A unilateral fixed dilated pupil will be more obvious as a result of the miosis in the unaffected pupil.
- Hypotension: The degree of hypotension induced by opioids is most marked with morphine that causes an anaphylactoid-like reaction characterized by histamine release. The degree of hypotension is related to the speed, dose and route of administration, with a large intravenous bolus causing the greatest degree of hypotension. The haemodynamic response to morphine is related to the degree of hypovolaemia, and hypotension is most marked in patients who are already volume depleted. In the trauma patient, pain may mask the presence of hypovolaemia as cardiovascular stimulation increases the pulse rate and helps maintain blood pressure. Therefore do not assume pain is the cause of a tachycardia in the trauma patient with a normal or low blood pressure. Instead treat any hypovolaemia before with fluid resuscitation before using opioids.
- Addiction: Addiction to opioid use only occurs with long-term use, and is not a concern for the short-term management of acute pain.

Occasionally, opioids may need to be reversed if an excessive dose has been administered. This may be done with small doses of naloxone – 400 mcg diluted in 10 ml saline and given

initially in 1ml increments. Excessive naloxone will antagonize morphine to the extent of causing severe pain. This pain will respond poorly to further opioids and alternative methods of analgesia e.g. nerve blocks should be considered.

Details of other commonly used opioids are given in Appendix 16.2.

### **Non-opioid drugs**

#### *Non-steroidal anti-inflammatory drugs (NSAIDs)*

This group of drugs reduces prostaglandin formation by inhibiting cyclo-oxygenase (COX). There are two types of COX:

COX-1, which is involved in production of prostaglandins responsible for gastric mucosal protection and platelet aggregation

COX-2, involved in the production of prostaglandins in injured tissues and involved in inflammation and pain

Older, non-specific NSAIDs (e.g. aspirin, mefanamic acid, ibuprofen) inhibit both forms of COX. Recently COX-2 specific NSAIDs have been introduced which have less adverse effects, although some (celecoxib and rofecoxib) have recently been withdrawn because of excess mortality. The analgesic effect is similar across all agents and there is a ceiling effect because other mechanisms of pain generation are not affected.

A summary of preparations and their potential for side effects is shown in Box 16.2 in Appendix 16.2.

#### Paracetamol

Paracetamol is an effective adjuvant in many forms of acute pain. It is probably under used, particularly in patients in whom NSAID administration is contraindicated. It is inexpensive by either the oral or rectal route. An intravenous preparation is increasingly available in European countries.

**Once acute severe pain has been controlled with opioids, all patients will benefit from having a combination of an opioid and either a NSAID or paracetamol.**

*Entonox*

Entonox is a mixture of 50% oxygen and 50% nitrous oxide. It is administered to spontaneously breathing patients via a demand valve using a facemask or mouthpiece and results in both an analgesic and euphoric effect. It should not be used in patients with possible pneumothorax or pneumocranum because of the risk of expansion of gas within enclosed spaces. When used correctly, Entonox can achieve the same analgesic efficacy as approximately 15 mg morphine. Like opioids, it may cause significant nausea and vomiting.

#### *Local Anaesthetic Creams*

These creams are simply and easily applied and allow intravenous access in a relatively pain-free manner in children. If time permits, local anaesthetic cream should be used prior to cannulation in all children. There are two preparations available:

##### *Ametop (= topical amethocaine gel)*

Ametop should be applied in a generous layer over veins suitable for cannulation (most commonly the dorsum of the child's hands). It should be applied to a minimum of 2 sites. The cream is covered with an occlusive dressing. For best results, it should be applied 30-45 minutes prior to cannulation/venepuncture.

##### *EMLA cream (= eutectic mixture of local anaesthetic: lidocaine and prilocaine)*

EMLA cream is applied in a similar manner to that described above, but is of slower onset. It may cause vasoconstriction and thus Ametop is preferred if possible. It is not suitable for use in children under one year.

#### **Local anaesthetic techniques**

These can be extremely useful in the trauma patient and range from simple local infiltration analgesia to nerve blocks. Whatever technique is used, it is important that a safe dose of local anaesthetic agent is calculated for each patient to avoid the risk of toxicity (CNS and cardiac excitation and/or depression). Further details are given in Appendix 16.2.

#### **General anaesthesia for pain relief**

Occasionally, the extent and nature of injuries may be so severe that adequate pain relief can only be achieved with general anaesthesia. This is particularly true for patients with extensive partial thickness burns where morphine may be not be able to provide immediate and adequate pain relief. Those with appropriate anaesthetic skills must administer general anaesthesia for these patients. Sedation is not a substitute for analgesia and the administration of intravenous benzodiazepines to the point of inducing a state resembling general anaesthesia is not appropriate. The use of the anaesthetic drug ketamine is covered in Appendix 16.2.

## **Summary**

Pain is a common feature of all injuries. Its subjective nature makes it difficult to assess and inadequate pain control is well-documented feature of acute injury. Pharmacological control of pain is usually necessary for most injuries, but patient reassurance and non-pharmacological adjuncts can contribute significantly to reducing drug requirements. Children are particularly challenging to treat; they should not be separated from their parents where possible and painful intramuscular injections avoided. Most acute pain will require an opioid-based drug to control the pain, but NSAIDs and paracetamol have an important morphine-sparing effect once the pain is controlled. Concerns over opioid-induced respiratory depression should not be a reason to deprive patients of adequate analgesia

## **Appendix 16.1: Physiology of pain**

An understanding of the physiology of pain will assist all the team members in assessing the patient's pain and allow them to make informed choices regarding appropriate and effective modes of analgesia.

As discussed, the perception of pain can be affected by many external stimuli such as anticipation, comprehension, anxiety, fatigue or a sense of danger. These stimuli have the potential to exacerbate an individual's perception of pain, particularly in the unplanned, stressful situation associated with accidental trauma. The body's physiological reaction to a noxious stimulus is termed nociception and pain is the associated unpleasant experience. The nervous system is the sensing, thinking, and controlling system of the body. Sensory information is collected from many sources, such as the skin, internal tissues, through sight and sound, and eventually transmitted to the brain for cognitive processing and interpretation. The peripheral and central nervous systems are the main components involved in the transmission and interpretation of pain. The manifestation of pain from injury is the result of a complex chain of communication between the body and brain

### **Peripheral nervous system**

Pain receptors (nociceptors) are located in the skin, subcutaneous tissues, arterial walls, viscera, periosteum, joint surfaces, muscles and fascia; all common sites of trauma-related injury. The majority of deep tissues have relatively few pain receptors, resulting in a less specific location of the pain stimulus. In the skin, the perception of pain begins in nerve fibers, the density of which varies; large quantities are found in highly sensitive areas for example the thumbs, fingers, or lips while smaller quantities are found in areas such as the back and internal organs. Each nerve ending has a receptor that responds to mechanical, thermal, and chemical stimuli resulting in appreciation of heat, touch, pressure, joint position and pain sensation.

### **Tissue damage**

Tissue damage causes the release of powerful inflammatory mediators such as serotonin, histamine and bradykinin, resulting in stimulation and sensitisation of chemoreceptors. The effect of these inflammatory mediators on chemoreceptors is thought to produce the pain of inflammation, particularly in those tissues surrounding the actual area of damage. These chemicals decrease the threshold of other pain receptors in the body. The effect of inflammatory mediators, released as a consequence of pain due to injury, is therefore to increase the intensity of response to the original stimulus.

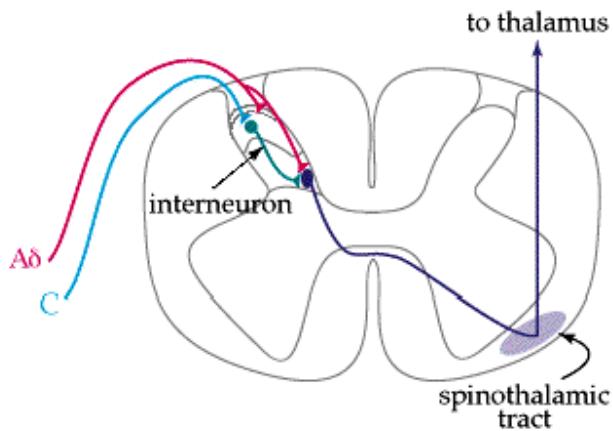
Once an injury has occurred, the stimulated nociceptors convey their afferent signal along A $\delta$  (delta) or C peripheral nerve fibers. A $\delta$  fibers are fine, myelinated fibers that conduct rapidly (5 - 30 m/sec). This is the first pain sensation sensed, usually sharp, pricking and well localized. C fibers are smaller, unmyelinated and are stimulated by mechanical, thermal, and chemical sources producing dull, aching, and burning sensations. C fibers are widely distributed in both the skin and deep tissues and mediate "slow pain" sensations. The conduction velocity of C fibers is one tenth that of A $\delta$  fibers, resulting in sensations less easily defined as to their intensity or point of origin.

Somatic pain originates from bones, tendons, ligaments, blood vessels and nerves and is detected by somatic nociceptors. Somatic pain is generally localised and typically described as aching, squeezing, stabbing, or throbbing. A sprained ankle or bone fracture is an example of somatic pain. Visceral pain originates from body organs and is detected by visceral nociceptors located within body organs and internal cavities. The relative scarcity of nociceptors in these areas produces more of an aching pain that is usually of longer duration than somatic pain. Visceral pain is difficult to localise, and may present as referred pain, where the sensation is localised to an area unrelated to the site of injury. Examples of visceral pain include that of myocardial ischaemia that may present as an aching or pain in the arm or jaw and diaphragmatic pain that may be referred to the shoulder.

Normally, most nociceptors lie dormant. Inflammation sensitizes this vast population of nociceptors, making them far more sensitive to stimulation (hyperalgesia). Hyperalgesia may be primary (felt at the site of stimulation, related to sensitization of the neurones innervating that area) or secondary (felt at a site remote from the original injury). Spinal mechanisms result in areas surrounding the injury also developing allodynia (secondary hyperalgesia). This results in the injured part and a surrounding zone being protected, by withdrawal from normal use. In most circumstances this promotes resolution of the pain and subsequent healing.

### **Central nervous system**

Nociceptive messages enter the spinal cord in primary afferent nerves through the dorsal horn of the spinal column. A $\delta$  fibers enter the spinal cord to synapse on a second set of neurons. These are the secondary afferents that cross to the opposite (contralateral) side of the spinal cord and ascend in the spinothalamic tract. The C fibers also synapse, but they do not synapse directly on secondary afferents. Instead, they synapse on interneurons that carry the signal to the secondary afferents that also cross to the opposite side of the spinal cord and ascend in the spinothalamic tract (Fig. 16.4).



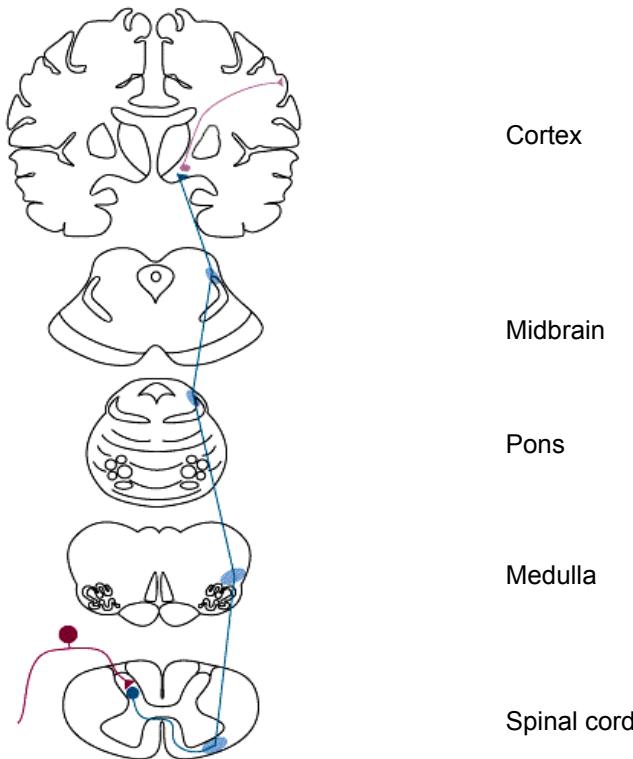
**Fig. 16.4: Pathways of A $\delta$  and C fibers within the spinal cord**

The spinothalamic tract ascends through the medulla, pons and midbrain to enter the thalamus, where it synapses, and is finally carried to cortex by the thalamocortical neurons (Fig. 16.5). There is no discrete area of the brain responsible for pain; pain perception, emotion, autonomic function, memory and motor responses are linked in a complex matrix.

#### *Pain modulation*

Spinal reflexes may increase pain following trauma. The stimulation of motor reflexes results in the muscle spasms often seen in trauma. Muscle spasm heightens the pain response possibly by inducing local metabolic changes. This often accounts for the dramatic improvement in pain control achieved after immobilization of a fracture.

**Fig. 16.5: Ascending pain pathways**



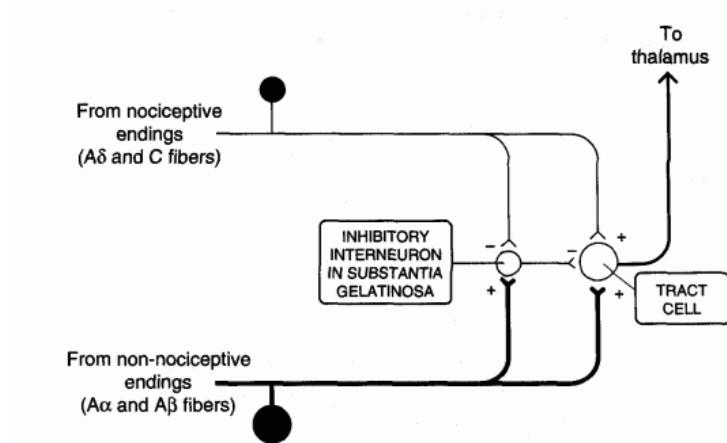
The transmission of pain signals to higher centres is a balance between ascending excitatory and descending inhibitory pathways. The ascending pathways have been discussed. Inhibition of afferent nociceptive signalling occurs at three levels:

*Tissue:* Drugs may inhibit the action of peripheral nociceptors, particularly in the terminals of C fibers that are already sensitised by inflammation.

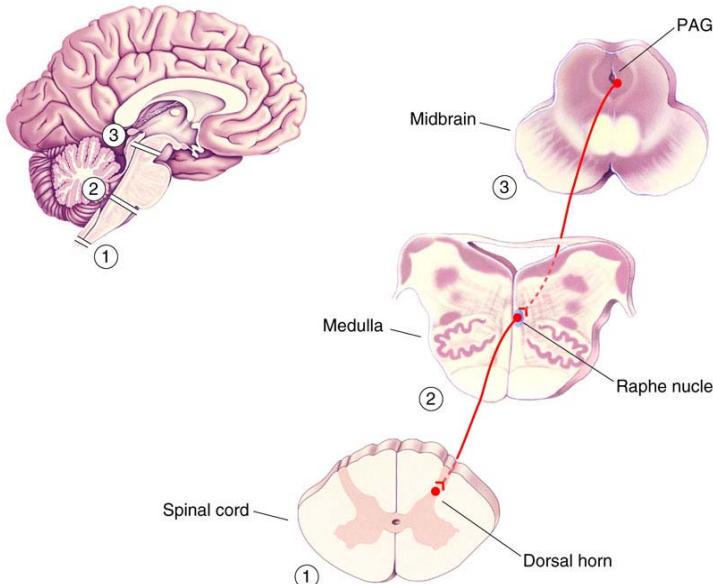
*Spinal cord:* Inhibitory interneurons in the substantia gelatinosa of the dorsal horn modulate afferent input to the ascending spinothalamic tract. Stimulation of these interneurons by touch and pressure afferents acts to stimulate the interneurons and reduce afferent signalling to the spinothalamic tract. This is known as the 'Gate control theory of pain' and was first described by Melzack and Wall in 1965 (Fig. 16.6).

*Brain:* Descending inhibition pathways originate at the level of the cortex and thalamus, and descend via the brainstem (periaqueductal grey) and the dorsal columns to terminate at the dorsal horn of the spinal cord (Fig. 16.7). Noradrenaline, serotonin (5-HT), and opioids (either endogenous or exogenous) release these pathways from GABAergic inhibition, increasing activity in the descending pathways and thus suppressing pain. Stimuli for endogenous opiate

release in these pathways includes pain stimuli ascending from the spinal cord, and stimuli passing along the multiple connections that the brainstem pain structures have with other local nuclei, the midbrain, and higher centres.



**Fig. 16.6: The gate control theory of pain (Melzack & Wall, 1965).** Non-nociceptive sensory nerve endings (e.g. touch, pressure) stimulate the inhibitory interneurons, whereas nociceptive afferents inhibit them. An increase in non-nociceptive afferent signalling will reduce the rate of firing of the spinothalamic tract neurones.



**Fig. 16.7: Descending inhibitory pathways**

## Appendix 16.2: Additional pharmacology

### Other opioids

#### Fentanyl

- An alternative morphine, for instance when there is allergy
- An initial dose of 100 micrograms is equivalent to approximately 10 mg of morphine
- Has a short duration of action
- As with all opioids, careful titration and observation is required

- 1-2 micrograms/kg intranasally in children is an effective analgesic.
- Available as a transdermal patch for slow release.

*Pethidine*

- Limited in its analgesic efficacy
- Relatively impotent against severe pain.
- It is also a partial agonist that limits the effectiveness of morphine given subsequently.
- It is best avoided for control of acute pain.

*Codeine (methyl morphine)*

- Metabolised to morphine to become active
- 10% of the population is unable to metabolise codeine to morphine (e.g. no analgesic effect). Conversion is therefore unreliable, providing variable analgesia
- Can only be given orally or intramuscularly; intravenously associated with severe hypotension
- Traditionally used for analgesia in patients with head injury; increasingly replaced with morphine

*Tramadol, Buprenorphine and Nubain*

- Little clinical benefit in most emergency situations compared to morphine and are generally unsuitable for acute pain relief.
- Less potent than morphine.
- Side effects similar to morphine.
- The use of this group of drugs (partial agonists) may compromise the efficacy of morphine at a later stage, making control of severe pain difficult to achieve.
- Much more expensive than morphine.

*Oxycodone and hydromorphone*

- Relatively new opioid antagonists
- Do not have significant active metabolites
- May be an advantage for patients in renal failure

**Non-steroidal anti-inflammatory drugs**

Although NSAIDs are very useful in the management of trauma pain, their potential for side effects (Box 16.2) and relatively slow onset of action make them unsuitable for the initial

management of patients with major trauma. They should therefore be reserved for use in those patients who have been fully resuscitated.

<b>Box 16.2: NSAID preparations and their potential side effects</b>		
	<b>Non-specific NSAIDs</b>	<b>COX-2 NSAIDs</b>
<b>Oral preparations</b>	Ibrufen 200 mg qds Diclofenac 50 mg tds	Rofecoxib 50 mg daily
<b>Intravenous preparation</b>	Ketorolac 10-30 mg	Parecoxib 20-40 mg
<b>Asthma</b>	Both are avoided in aspirin sensitivity and asthma associated with recurrent nasal polyps. Safe if history of uneventful NSAID usage	
<b>Upper GI ulceration</b>	Avoid if significant recent history	Reduced risk of ulceration
<b>Renal failure</b>	Both are avoided in critical illness, hypovolaemia or established poor renal function	
<b>Haemorrhage</b>	May potentiate bleeding by inhibiting platelet aggregation	Little or no effect on platelet function
<b>Bone healing</b>	Both may inhibit bone healing (animal model evidence only)	

### *Ketamine*

Ketamine is a unique potent intravenous anaesthetic and analgesic agent. Its analgesic efficacy far exceeds that of morphine and it is able to control the most severe pain, far quicker and more effectively than any opioid. Although it is traditionally advocated for use in extreme situations such as emergency amputation, it is generally underutilised for the control of acute pain in any situation. Its side effects such as salivation and emergence delirium as the drug wears off have hindered its acceptance into the routine frontline management of acute severe pain. When used carefully, these side effects are usually not a significant problem. Emergence delirium is worse with repeated doses, particularly in young adults, and can be limited by concomitant administration of an intravenous benzodiazepine e.g. midazolam 1-2 mg i.v. or diazepam 2-5 mg i.v., repeated as necessary.

Ketamine excels for the provision of acute analgesia to cover painful procedures such as extrication from a car or reduction of a fractured limb. An intravenous dose of 1 mg/kg provides short-term analgesia to cover these procedures. Intramuscular doses of 3-4 mg/kg provide similar analgesia, but with delayed and subsequently prolonged action. Intranasal ketamine (5-6

mg/kg) has been used successfully to provide acute analgesia to cover painful procedures in children.

At higher doses, ketamine has anaesthetic effects. Unlike other anaesthetic agents, the airway and circulation are relatively well maintained, but this should not be used as an excuse for complacency. Those using the drug at anaesthetic doses must be competent in acute airway management. A dose of 2-3 mg/kg i.v. will produce anaesthesia for several minutes although movement and vocalisation may still occur. In extreme circumstances, ketamine can be administered intramuscularly for general anaesthesia (10 mg/kg i.m.), but the effects are very unpredictable, particularly in circumstances where peripheral perfusion may be impaired.

### **Analgesia for special circumstances**

#### *Drug abusers or patients on methadone maintenance programs*

As always, a careful assessment of pain and analgesia is required. These patients suffer pain from injury just like any others and are often fearful that requests for analgesia will be ignored which may increase their demanding behaviour. Intravenous access may be difficult in these patients, necessitating the use of other routes to achieve acute pain control. A traumatic episode is unlikely to be a good time to institute a drug withdrawal programme – remember that they already have a substance abuse problem and withholding adequate analgesia for obvious injury is unlikely to be helpful. Opioids will be relatively ineffective but if used, must be at an appropriately high dose. Other forms of analgesia such as NSAIDs and local blocks will usually be very welcome. Opioid maintenance will need to be continued. Liaison with the local drugs team may provide future support.

### **Appendix 16.3: Local anaesthetic techniques**

Recommended maximum doses of local anaesthetics:

Lidocaine plain – 3 mg/kg

Lidocaine with adrenaline – 5 mg/kg

Bupivacaine  $\pm$  adrenaline – 2 mg/kg

(Levo-bupivacaine is less toxic and has a wider safety margin).

The relationship between the amount of drug, concentration and volume is given by the formula:

$$\text{Volume (ml)} \times \text{concentration (\%)} \times 10 = \text{dose of drug (mg)}$$

For example: 20 ml of 2% lignocaine

$$20 \text{ (ml)} \times 2 \text{ (\%)} \times 10 = 400 \text{ mg}$$

Toxicity is also increased using more concentrated solutions therefore always use the lowest effective concentration, for example, 0.5% lignocaine is adequate for infiltration analgesia. Lower concentrations allow larger volumes that are more effective for nerve blocks.

Although there are a large number of nerve blocks that can be usefully applied in the trauma patient, there are two that all trauma team leaders should be familiar with; intercostal and femoral nerve blocks. These blocks are very effective and relatively free from complications. Those interested in other blocks should refer to one of the many excellent texts available.

#### **Intercostal nerve block**

Useful for rib fractures to aid coughing and deep breathing. The intercostal nerve runs in the subcostal groove along the inner, inferior border of the rib, accompanied by the intercostal vessels. The nerves associated with the lower seven ribs are most accessible and blocked at the angle of the ribs. There is a small risk of pneumothorax, so patients need to remain under close supervision after they have been attempted.

- The patient is best placed in the lateral position, affected side uppermost. The upper arm is then placed above their head to remove the scapula from over the ribs.
- The site of injection is approximately a hands breadth from the spine
- The site is cleansed with antiseptic and the area draped
- The lower border of the rib is identified by the operator's non-dominant hand.

- The skin over the rib is then “pushed” upwards (cranially)
- The syringe containing local anaesthetic solution, with needle attached, is held like a dart and the needle introduced perpendicularly through the skin to make contact with the rib
- The tension in the skin is then released allowing the needle to be “walked” down the rib until it just slips off the inferior border
- The needle is then angled at 45° and advanced a further 0.5 cm to place the tip in the groove beneath the rib
- The syringe is then aspirated looking for blood or air to ensure that the tip of the needle is not in the vessels or within the lung respectively
- Local anaesthetic, usually 3-4 ml of 0.5% bupivacaine, is then injected and the needle withdrawn.
- The procedure is then repeated at the next rib.

### **Femoral nerve block**

The femoral nerve supplies most of the periosteum of the femur and a block allows fractures to be reduced or traction applied effectively with minimal discomfort, avoiding the need for opioid analgesia. It is important to use an appropriate technique and regional anaesthesia needle to minimise risk of damage to the nerve.

- The inguinal ligament is identified, running from the anterior superior iliac spine to the pubic tubercle.
- The femoral artery is identified at the midpoint, just below the ligament and the area cleansed.
- A finger is placed on the pulsation of the femoral artery.
- A 21 g, short bevel needle, attached to a syringe containing local anaesthetic, is inserted 1cm laterally to the artery, at 45° and advanced to a depth of 3-4 cm.
- In the conscious patient, if paraesthesia is elicited, withdraw the needle slightly.
- Aspirate to ensure that the needle tip is not within a blood vessel.
- Inject local anaesthetic, usually 20 ml of 0.5% bupivacaine.
- If paraesthesia is not obtained or the patient is unconscious, inject the local anaesthetic in a fanwise manner from adjacent to the artery, moving laterally, 3-4 cm.

### **Topical analgesia for chest drains**

A chest drain can cause severe pain as it irritates the pleura covering the lungs and ribs. Although opioids can be used to treat this pain, local anaesthetic instilled down the chest drain washes over the pleural surface to provide very effective analgesia. Absorption of local

anaesthetic across the pleura is rapid and therefore lidocaine is often used in preference to more toxic bupivacaine. Ten ml of 10% lidocaine, repeated no more than 2 hourly, is often spectacularly effective.

## **Further reading**

1. Manual of Pain Management. 2<sup>nd</sup> Edition. Ed: Warfield CA, Fausett HJ. Published by Lippincott Williams & Wilkins, Philadelphia. ISBN 0-7817-2313-2.
2. Mackway-Jones K. Eds. (1997) Emergency Triage. BMJ Publishing Group. London.

# **CHAPTER 17**

## **CHEMICAL, BIOLOGICAL, RADIOLOGICAL & NUCLEAR (CBRN) INCIDENTS AND TRAUMA**

### **Objectives**

At the end of this chapter, the reader should understand the:

- Effects of chemical, biological, radiological and nuclear (CBRN) hazards
- Generic response to CBRN and trauma.
- Definitive management of CBRN exposure and combined injury.

### **Introduction**

The presence of CBRN agents during an incident whether deliberate or accidental requires extra expertise for both scene and clinical management. In general, the term 'CBRN' is used for deliberate release, while the term 'hazardous materials' is used for accidental release. In most cases the distinction is academic. Where there is a contamination hazard, an interim (warm) zone is required for decontamination close to the scene of the incident. Within this zone, assessment and treatment is usually limited but still possible.

Combined injuries (trauma and CBRN exposure) may occur for a number of reasons:

- Exposure to hazardous materials, such as asbestos or toxic industrial chemicals, may be secondary to a traumatic incident.
- The delivery method used to disperse the CBRN agent may cause trauma ('dirty bomb').
- A CBRN device can cause traumatic injury (e.g. nuclear detonation).
- The manifestation of the exposure causes trauma-like injuries (e.g. chemical burns).
- The injuries may be due to the population's response to the incident (e.g. evacuation and stampede).

A review of deliberate release suggests that the release of chemical and biological agents is generally covert, although the effects of a chemical attack may be seen immediately. Radiological incidents are more likely to be combined with traumatic injuries because a dispersal device (explosive) is required to maximise effect.

## **Generic (all hazards) approach**

When an incident presents an assessment of possible hazards should be made. This may be in the pre-hospital phase, outside, or in the emergency department. The presence of an unknown (unusual) agent should be suspected if there are multiple casualties presenting with similar symptoms or a single casualty with an unusual presentation. Concurrent trauma may or may not exist. A simple assessment method is the STEP 1-2-3:

<b>ONE casualty</b>	<b>Approach using normal procedures.</b>
<b>TWO casualties</b>	<b>Approach with CAUTION, consider all options.</b>
<b>THREE casualties or more.</b>	<b>Do NOT approach.</b>

Depending on the hazard, the need for Personal Protective Equipment (PPE) and further measures such as containment, decontamination or isolation should be made based on the possibility of *contamination* or a *contagious* agent. If there is a risk on contamination, decontamination should take place as soon as possible and ideally before transport to hospital. Life-saving interventions should be concurrent and, in radiation incidents, take precedence.

The level of PPE should depend on the type of hazard – chemical (gas, vapour, particulate), biological (toxin, particulate) and radiological (direct gamma radiation, radioactive particulate matter). Within a hospital, after decontamination has taken place the normal universal precautions appropriate for trauma care are applicable for post-decontamination patient care. In cases of chemical ingestions of agents such as cyanide salts and organophosphates, precautions should be made to limit any exposure from off-gassing and may include gas scavenging systems.

In severe cases, patients may be taken directly to hospital. In addition, ambulatory patients may self-present to Emergency Departments while still contaminated. All Emergency Departments should have facilities to decontaminate casualties, both ambulatory and non-ambulatory. Some form of triage will be required and should take place prior to decontamination, as triage will prioritise casualties requiring urgent decontamination as well as medical interventions. The provision of life-saving interventions may include:

- Basic airway management.
- Ventilatory support, including the use of adjuncts such as LMAs.
- Decompression of tension pneumothoraces.
- Early antidote treatment either by intramuscular or intraosseous routes.
- Haemorrhage control.

The effects of CBRN agents as well as trauma can be remembered as the *four I's* – *intoxication, infection, irradiation* and *injuries* respectively. Each incident can be assessed by using the pneumonic 'T.C.I.P. – To Come In Please' standing for:

	<b>Chemical</b>	Biological	Rad/Nuclear	Trauma
<b>T</b> o	<b>TRIAGE</b>			
<b>C</b> OME	<b>CONTAMINATION</b>		<b>CONTAGIOUS</b>	
<b>I</b> N	<b>INTOXICATION</b>	<b>INFECTION</b>	<b>IRRADIATION</b>	<b>INJURIES</b>
<b>P</b> LEASE	<b>PROPHYLAXIS (AND TREATMENT)</b>			

Traumatic injuries associated with CBRN or hazardous material exposures can be classified as:

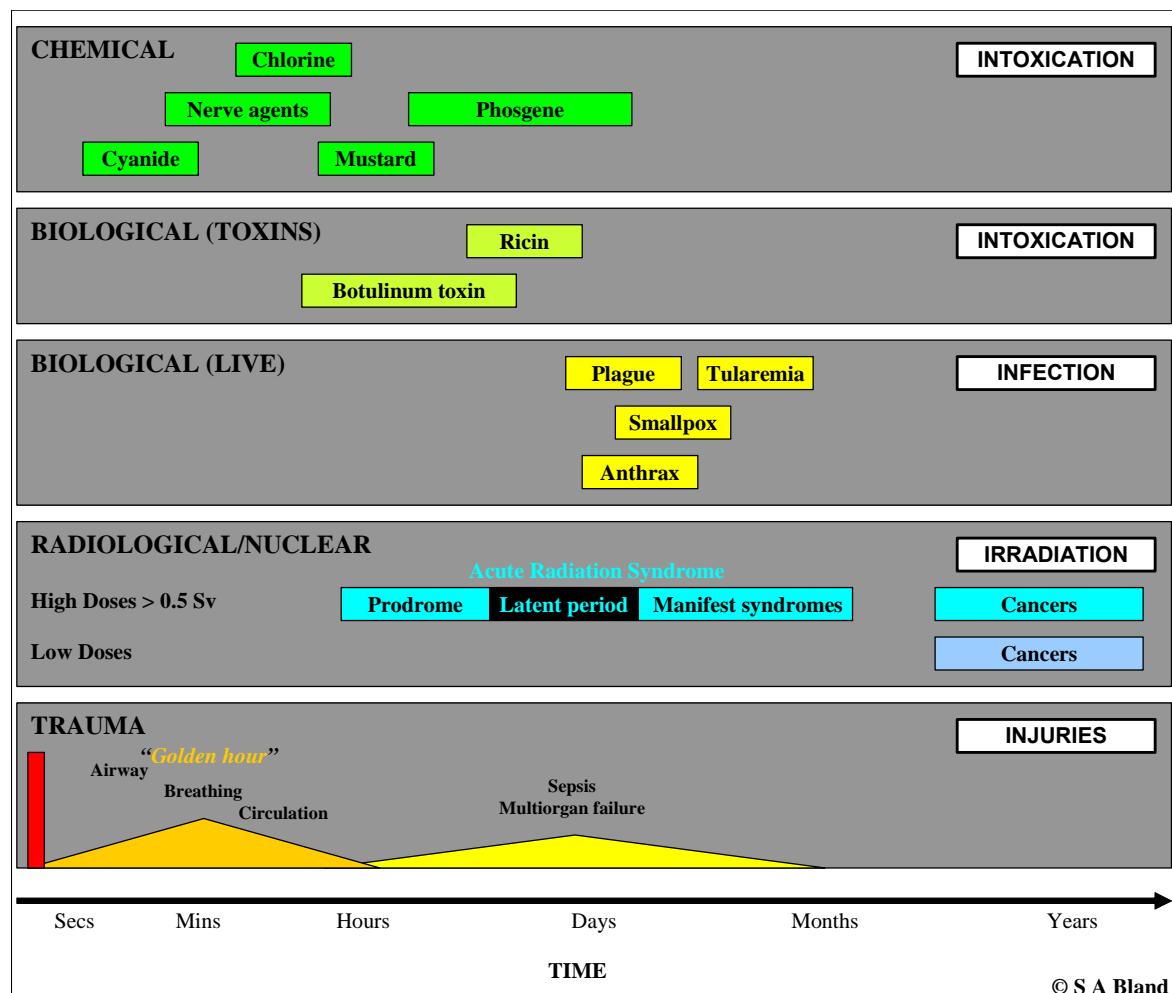
- Primary – injuries due to the direct effect of the CBRN component of a device (chemical burns, thermal / blast injuries from a nuclear detonation)
- Secondary – injuries resulting from the CBRN device delivery method (bomb blast, artillery bombardment). Blast injuries also have their own classification.
- Tertiary – injuries resulting from the indirect effects of a CBRN device (evacuation, stampede, crush injuries)

Because of the potential for combined injuries and the wide spectrum of CBRN agents a generic approach is recommended. Chemical agents tend to have an early onset, followed by biological toxins. Live biological agents have a slower onset due to an incubation period. Fig. 17.1 highlights the onset times of these agents and compares them with the priorities (ABC) for trauma and the distribution of mortality.

### Chemical

The effects of chemical agents can be wide ranging from irritant to lethal. Agents include household chemicals, toxic industrial chemicals and chemical weapons. The approach to chemical hazard is determined by the specific characteristics of the agent:

- *Toxicity* – the amount of a chemical required to cause a toxic clinical effect. Classification includes incapacitating, damaging and lethal. These can be further sub-typed into classes of agents specific to their mechanism of action i.e. nerve agents or vesicant.
- *Latency* – the period of time between exposure and clinical effect.
- *Persistency* – the physical properties of the chemical that determine how long the agent remains a threat after release. This influences the risk of secondary contamination of rescue and hospital staff.

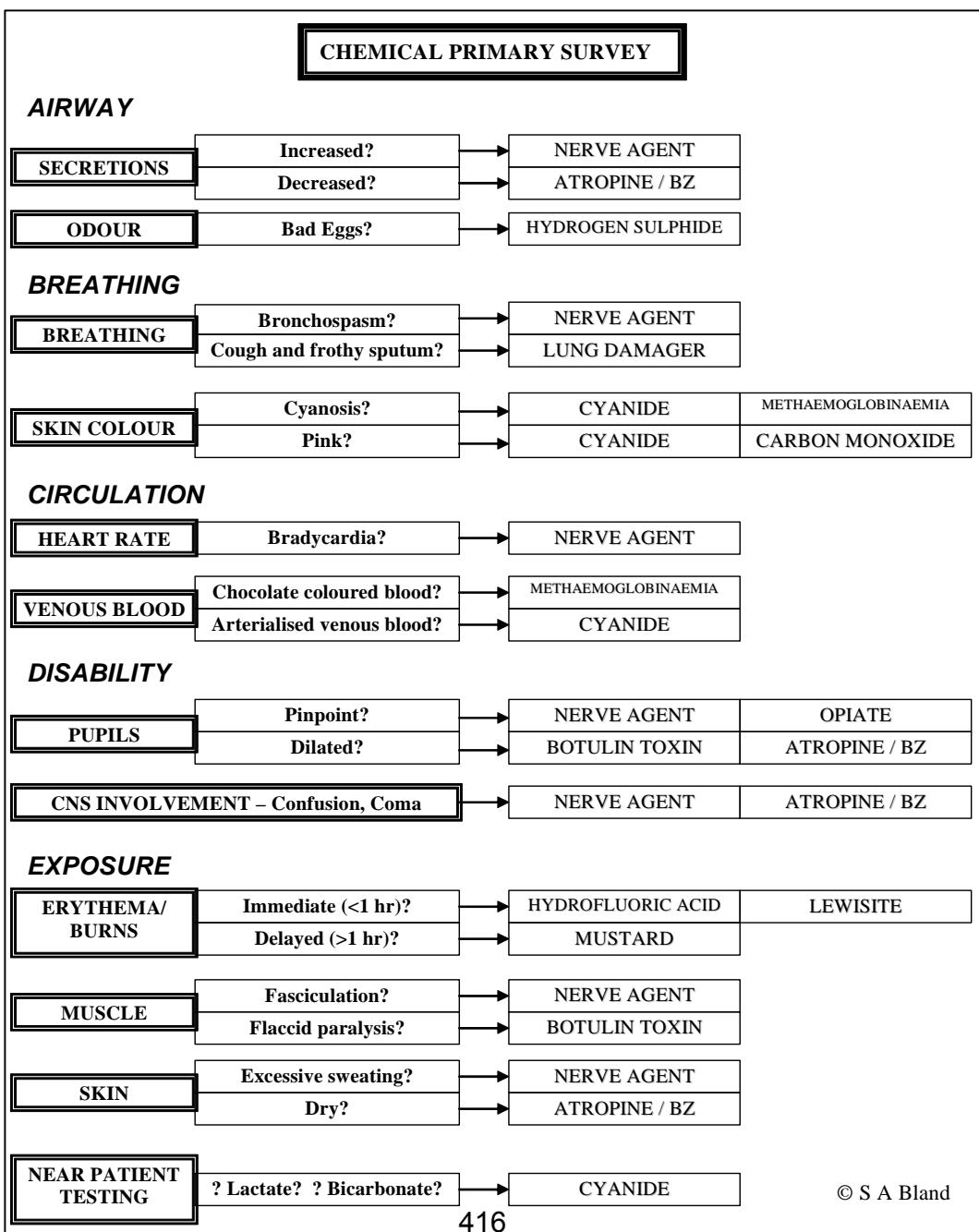


**Fig. 17.1: Comparison of onset times for CBRN insults compared with trauma**

Priorities in the management of these cases are based on the triage category of the casualty, the contamination risk to responders and the requirement for life saving interventions and early antidote administration. The definitive management of specific chemical agents includes the use of antidotes. During the initial assessment of the

casualty, the primary survey is used to identify any immediately life-threatening injuries that can be addressed by primary resuscitation. Information collected during the standard ABCDE trauma assessment can be used to identify clinical signs associated with specific chemical agents. These are listed in Box 17.1. This 'chemical primary survey' looks for signs that identify specific chemical syndromes (toxicoses) or agents that have specific antidotes or management options. It is not inclusive of all agents and its specificity varies with co-morbidities and injuries and is therefore only a guide to some of the commoner chemical syndromes

**Box 17.1: Clinical signs associated with different chemical agents**



Where chemical exposure has taken place, the agent may further compromise the traumatised casualty both in the acute and later phases of recovery. Chemical agents may alter the casualty's response to trauma, mimic traumatic injuries (chemical burns) or interact with drugs used in trauma management. These effects are agent specific and detailed in Box 17.2.

<b>Box 17.2: Specific chemical agent effects on the traumatised casualty</b>	
CHEMICAL AGENT	POTENTIAL COMPLICATIONS DURING COMBINED INJURY
Nerve agents (acute)	Excessive airway secretions and bronchospasm may further compromise airway obstruction and failure to ventilate lungs. Increased airway pressures due to bronchospasm may mimic tension pneumothorax during positive pressure ventilation. The pupillary effects of NA (miosis) and its treatment with atropine (mydriasis) will effect head injury assessment. NA effects on the autonomic nervous system (including bradycardia) may cause cardiovascular compromise in a compensating casualty.
Nerve agent (late)	Intermediate effects of NAs may lengthen time required in intensive care because of muscle weakness.
Sulphur mustard (late)	Acting as an alkylating agent, systemic toxicity may include bone marrow suppression and increase the likelihood of infection.
Pulmonary agents (choking agents)	Pulmonary agents, such as chlorine and phosgene, may present as pulmonary oedema. This may exacerbate respiratory failure due to chest trauma. Phosgene should be noted for its delayed onset and potential for misdiagnosis.
Cyanide	Severe cyanide casualties will not self-refer to hospital and treatment should be initiated early with specific emphasis on rescuer personal protection from off-gassing where cyanide salts have been ingested. Moderate casualties that survive beyond the pre-hospital environment may have a significant metabolic (lactic) acidosis. This may be misleading when assessing shock and oxygen delivery. Cyanide poisoning should be considered in all cases of smoke inhalation. Supportive management with high flow oxygen is recommended.

Iatrogenic (atropine)	Inadvertent use of atropine may lead to anticholinergic toxicity. This typically includes dry mucosa, large pupils, tachycardia and confusion. The last three signs may confuse clinical assessment in the presence of trauma, especially when assessing shock and head injuries. Thermoregulation may also be disturbed because of reduced sweating, leading to hyperthermia particularly in warm environments.
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Vesicants and in particular the *mustards* have been used widely used, most recently in the Iran-Iraq War. Despite their widespread use, sulphur (HD) and nitrogen mustard have no specific antidotes and rapid decontamination remains the optimal way to reduce absorption and minimise injuries (skin burns, ophthalmic, mucosal and airway injury, and systemic toxicity). Lewisite (an arsenical based vesicant with significant systemic toxicity) does have antidotes available, including British Anti-Lewisite (BAL / dimercaprol), that act as chelating agents.

Recent work on the management of exposure to these agents makes a number of recommendations:

- Cooling (~15°C) of the affected areas has been shown in animal models to reduce the effect of the HD on comparison with controls. Care should be given not to cause iatrogenic hypothermia.
- General management, such as fluid balance and escharotomy when indicated, is similar to that of thermal burns with particular attention to electrolyte balance.
- Topical antimicrobials should be considered, but systemic antibiotics should not be given routinely. There is a risk of immunosuppression due to systemic toxicity.
- Treatment of burns with laser debridement or dermabrasion has been shown in animal models to reduce healing time by breaking down the eschar.
- There is no clear evidence for the use of steroids.
- Ophthalmic injuries are common and treatment includes the use of a mydriatic such as tropicamide, with topical antibiotic. Steroids have been advocated for topical use, especially in severe cases. In the most severe cases, corneal grafting may be required.

Further research into the treatment of mustard exposure is looking at the use of anti-oxidants and includes work on vitamin C and E, and N-acetylcysteine.

Chemical burns that require particular attention are those due to *hydrofluoric acid* (HF). This is because of systemic toxicity. HF's effects can be broken down into those due to the acid ( $H^+$ )

and those due to the fluoride ion ( $F^-$ ). The systemic toxicity of HF is due to the high affinity of the fluoride ion for calcium. A relatively small burn (~3%BSA) may be enough to cause life-threatening hypocalcaemia with cardiac manifestations. Treatment includes the use of topical calcium gluconate gels, local infiltration of the burn site with calcium gluconate and where systemic toxicity is seen, intravenous calcium chloride. Observation, ECG monitoring and laboratory support is required.

## **Biological**

Biological hazards can be differentiated into *live biological agents* and *toxins*. This is important as the risks and medical management differ for each type of agent. Live biological agents include bacterial, viral, fungal, rickettsial and chlamydial organisms. Effects due to infection may range from incapacitation (salmonella) through to death (SARS / avian flu/ inhalational anthrax). Infected casualties may be contagious as well as contaminated. Risk from live agents is based on lethality and transmissibility. Biological toxins (botulinum toxin, ricin, staphylococcal toxin B) are essentially chemical agents of biological origin. They are non-transmissible but present a potential contamination hazard. Risks from toxins can be classified in the same way as for chemical agents - toxicity, latency and persistency.

Combined injuries with live biological agents are less likely to be a problem during the initial phases of medical management. This is because live agents usually require an incubation period before a manifest illness is observed. Antibiotics used in trauma are likely to provide some chemoprophylaxis to potential bacterial agents (assuming normal sensitivities). Delivery methods for live biological agents also tend to be more covert without the use of explosive dispersal devices.

In contrast, biological toxins may work synergistically with traumatic injuries. While atraumatic methods of dispersal have been used for toxins, absorption of small but lethal doses can be achieved if the agent penetrates the skin. Biological toxins such as botulinum and ricin have an onset time that could affect the initial management and recovery of the casualty. These effects can mimic other trauma-associated complications, such as respiratory failure, cardiovascular collapse and sepsis. The use of toxins is not unprecedented with examples including the use of ricin on Bulgarian dissident Georgi Markov and the alleged use of botulinum toxin for the assassination of Hitler's potential successor, Reinhard Heydrich. Markov was killed after inoculation with a small ricin impregnated pellet delivered from an umbrella although the diagnosis was post-mortem. Heydrich died a few days after an assassination attempt in Prague,

using a grenade. The injuries were originally thought to be serious but survivable. However, Heydrich's condition suddenly deteriorated and he went into a coma with respiratory failure, and subsequently died. Botulinum toxin remains a possible cause of death introduced into the body by the grenade fragments, although secondary wound sepsis was cited as the original cause of death.

## Radiological / Nuclear

Radiological and nuclear hazards are managed similarly. Radiological incidents may include any event involving ionizing radiation (alpha, beta, gamma and X-ray) and so can involve industrial sites, transportation and medical sources. An example of deliberate release includes a radiological dispersion device ("dirty bomb"), which may use explosives. Casualties from radiological or nuclear incidents may present in a variety of ways, including contaminated, irradiated, traumatic and combined. Scenarios that lead to *Acute Radiation Syndrome* (ARS), through high dose radiation exposure, are unlikely unless one of the following has occurred:

- The nuclear process (*fission*) to have taken place, including
  - Nuclear reactor incident, either as an accident or deliberate act
  - Criticality (brief period of critical mass) incident, such as a laboratory accident
  - Nuclear detonation
- Prolonged exposure to highly radioactive sources, including
  - Industrial radiography sources
  - Radiotherapy sources
  - Nuclear power sources used in remote locations ('nuclear batteries')

The effects of high dose radiation are primarily due to cell death. Because a certain number of cells have to die before there is organ / system dysfunction, there is a threshold level before the effects are seen as ARS (~0.5Sv); an average chest x-ray is 20 $\mu$ Sv. This syndrome is a result of the death of cells with a rapid turnover. After prodromal symptoms (nausea, vomiting) and a latency period, ARS results in bone marrow suppression and loss of gastrointestinal mucosa. Without medical management, the lethal dose to kill 50% of the population at 60 days (LD50/60) is 3.5Sv. This can be increased to at least 5-6Sv with medical support and definitive management. The main cause of death is sepsis and coagulopathy due to haematological and gastrointestinal failure. At very high doses, cerebrovascular failure will contribute to the mechanism of death. The effects of low-level radiation are associated with cell (DNA) damage leading to mutagenesis and carcinogenesis. ARS can be managed by supportive therapies, including antibiotics and blood product replacement. Definitive management includes the use of cytokines such as colony stimulating factors and bone marrow (stem cell) transplant.

The most likely scenario is the use of an explosive device to spread radioactive material. High levels are unlikely but may be possible where there are shrapnel injuries caused by radioactive fragments. A mixture of contaminated and injured casualties is likely with some contaminated wounds. These casualties should be prioritised, both for decontamination and medical support. While decontamination will allow more effective medical interventions, it should not delay lifesaving medical interventions; gross decontamination (removal of clothing) is an integral part of a trauma primary survey and can achieve up to 90% removal. Where combined injury includes trauma and irradiation, outcome predictions are significantly worse than for trauma or irradiation alone. Wound contamination should be managed as for any contaminated wound. Irrigation and surgical debridement remains the mainstay of treatment. Any foreign bodies should be treated with caution, not handled and placed in a shielded container.

#### *Injuries from nuclear detonation*

Following the detonation of a nuclear device, the following “conventional” traumatic injuries will be seen:

- Flash – causing afterimages, retinal damage and blindness.
- Blast injuries – caused by the initial positive and negative overpressures, and blast winds. In addition, further injuries will occur due to flying debris and crush injuries due to falling masonry. Treatment will follow conventional guidelines.
- Thermal injuries – these are due to the initial intense flash and fireball with further injuries secondary to any fires ignited. These thermal injuries are different to burns caused by ionising radiation.

High levels of radiation should be suspected in casualties presenting with prodromal symptoms: nausea, vomiting, diarrhoea and erythema within hours of the event. ARS (> 2Sv) will over a period of time cause the casualty to experience bone marrow suppression leading to immunosuppression, coagulopathy and anaemia. Bacteraemia is also likely, due to the gastrointestinal manifestation of ARS. (I have deleted this next part as it is beyond the scope of the course). Burns, in the context of radiation injury, are associated with a poor prognosis. Once a burn exceeds a surface area of 50%, even in the absence of combined injury, a poor outcome is much more likely. When combined with radiation, prognosis will be significantly reduced.

#### **Summary**

Combined injuries present a difficult and unusual problem with little human data. Much of the management is based on expert opinion, consensus and animal studies. There is some

historical data on antidote use and their use is based on a risk / benefit assessment. The assessment has to include the additional risks to a traumatised patient. This is particularly important when considering interactions between chemical agents and anaesthetics, likely to be used for trauma casualties. Any anaesthetic agent should be used with caution. CBRN incidents are likely to generate mass casualties and difficult decisions will have to be made with regard to resource allocation and the potential use of triage. Management should be based on:

- Personal safety for health care workers dealing with the patient
- Triage.
- Assessing the risk of contamination and contagion.
- Identification of specific agents and life threatening conditions.
- Supportive management, including resuscitation and prophylaxis.
- Definitive management, including surgery, antidotes and antibiotics.



## CHAPTER 18

# INTER- AND INTRA-HOSPITAL TRANSFER OF THE TRAUMA PATIENT

### Objectives

At the end of this chapter, members of the trauma team responsible for transferring a patient should understand:

- The indications for transfer
- The risks of transfer
- The composition of the transfer team
- The equipment and drugs required
- The phases of transfer

**The following advice is based upon experiences within the UK. Some aspects will not apply in your country. In such cases local protocols should be followed, providing they meet the same standard of care and safety during patient transfer.**

### Introduction

Each year in the United Kingdom, thousands of critically ill patients are transferred between hospitals. A significant proportion of these transfers involve trauma patients undergoing secondary transfer from district general hospitals to specialist units. Typically, a hospital serving a population of 250,000 will need to transfer out 1-2 major trauma patients per month, mainly for neurosurgical care. Inter-hospital transfer of critically injured patients is a more serious and potentially complicated process than many people realise and the occurrence of adverse incidents is well documented.

### The need for transfer

The fundamental reason for transferring a patient is to achieve better care in a different geographical location. This may be because the patient's injuries require specialist treatment that is not available in the primary receiving hospital or because the hospital's intensive care beds are all full. Ideally, such a patient would have been taken directly from the scene to a hospital with all the necessary resources, making secondary transfer unnecessary. To do this may require a longer primary journey to hospital, delaying initial hospital treatment. Without a high speed, fast response transport system (e.g. with helicopters ready to scramble) that can

deliver head-injured patients rapidly to a regional neurosurgical centre from anywhere within the region, it is inevitable that the need for secondary transfer will continue.

### **Reasons for transfer**

There are three main categories of transfer *to* or *from* hospitals:

- Primary Transfer – from the scene to the initial hospital (this is not discussed here)
- Secondary Transfer – between hospitals for clinical benefit (this is the main type of transfer to be considered)
- Tertiary Transfer – between hospitals for social or other non-clinical reasons (e.g. repatriation for humanitarian or economic reasons after sustaining injuries abroad). These transfers are usually less urgent and involve patients who have been already stabilised before transfer is even considered.

Transfers *within* hospitals are more frequent than *between* hospitals and need the same attention to detail. Although it may seem that help is always close at hand, the fundamental risks are similar and deterioration may occur in the corridor at an alarming rate. The overarching reason for transfer is still the same – the patient should benefit from being moved, whether it is for a CT scan, for surgical intervention or to step up to intensive care.

### **Indications for secondary transfer**

Major trauma patients require secondary transfer for a variety of clinical reasons, including a *lack* of:

- Surgical expertise (e.g. a neurosurgical service)
- Specialist intensive care (e.g. a neuro-critical unit or paediatric intensive care unit)
- Facilities for specialist investigation (e.g. 24-hour computerised tomography)
- Intensive care beds, due to inadequate staffing or just being full

Specific clinical indications for secondary transfer usually relate to the anatomical injury and the corresponding specialist service (Box 18.1). The decision to transfer must take account of all the patient's problems. A few isolated specialty hospitals still exist (e.g. cardiothoracic and spinal centres) and secondary transfer to such hospitals may solve one problem but create others: major blunt trauma rarely affects an isolated body region.

**Box 18.1: Indications for secondary inter-hospital transfer**

1. Severe head injury
  - Requiring a neurosurgical operation
  - Requiring neurosurgical assessment or intensive neurological monitoring
2. Suspected mediastinal injury
  - Aortic tear
  - Tracheo-bronchial rupture
  - Ruptured oesophagus
3. Burns
  - Extent (surface area and depth)
  - Particular site (e.g. airway and flexures)
4. Spinal injuries
  - Unstable fracture
  - Spinal cord injury
5. Limb and pelvic injuries
  - Pelvic (including acetabular) reconstruction
  - Vascular injuries
  - Open tibial fractures with extensive soft tissue injuries
  - Severe hand injuries
6. Severe facio-maxillary injuries
7. Severe liver injuries
8. Severely injured children
  - Requiring specialist paediatric surgery
  - Requiring paediatric intensive care
9. Severely injured patients
  - With multiple injuries, according to regional trauma system guidelines, where it has been agreed to centralise major trauma in a trauma centre
  - Requiring regional or supra-regional intensive care techniques (e.g. intracranial pressure monitoring or extra-corporeal membrane oxygenation)
  - Requiring rehabilitation
  - Requiring repatriation or treatment in their home area

## **The risks of transfer**

Transfer is never free of risk. The universal rule is that whenever a decision to transfer is made, the potential benefits of receiving care at the destination must outweigh the risks of transfer. Otherwise the decision to transfer cannot be justified. Key aims of transport medicine are to identify and then to minimise the overall risk of transfer.

### **Risks to the patient**

The patient is starting from a position of risk because of the injuries sustained. The mobile environment presents a further physiological challenge to the patient. For example, sudden movement may trigger hypotension in a vulnerable patient, though the stimulation may lead to hypertension in others. The risks to the patient associated with transfer are listed in Box 18.2.

#### **Box 18.2: Risks to the patient associated with transfer**

- Deterioration of the underlying condition
- Physical disturbance resulting from vibration, acceleration, deceleration (including impact in the event of a crash), the thermal loss or ambient pressure changes
  - Dislodgement of endotracheal tubes, chest drains, intravascular lines and monitoring leads
  - Ventilator disconnection
  - Haemodynamic de-stabilisation of hypovolaemic patients associated with physical movement
  - Displacement of fractures with risk of pain or neural compression
  - At altitude, reduced partial pressure of oxygen and increased size of gas-containing spaces (e.g. pneumothorax, pneumocephalus)
- Inadequate care due to poor access and a moving environment
- A limited range of drugs and equipment – it is impractical to take too much
- Limited expertise in the escorts – while it is impossible to supply the full range of skills available in hospital, training and experience can mitigate this risk
- Damage to or failure of equipment en route interfering with monitoring or therapy
- Unexpected delays en route

### **Risks to escorts and others**

Escorts are at physical risk when travelling at high speed in an emergency ambulance. They are vulnerable mentally too, as their clinical decisions and practical skills are being tested in potentially stressful circumstances. Knowledge, judgement and concentration are essential to

prevent mistakes. While the escorts are absent from their base hospital, the emergency cover may be under pressure, potentially putting other patients at risk.

### **The speed of transfer**

In general, the journey should be rapid so that time spent outside hospital is minimised, so long as the speed does not itself lead to de-stabilisation of the patient. The preparation time too should be as short as possible if emergency treatment of the underlying condition at the receiving centre is time-critical. However, setting off without adequate stabilisation is a recipe for disaster.

### **The urgency of the condition**

The urgency of transfer is dictated primarily by the underlying injuries and their planned treatment. The speed of response varies according to whether the transfer is an *emergency* for life-saving surgery, *urgent* for life-threatening states that require specialist intensive care but no surgery, or *scheduled/elective* for reconstruction or rehabilitation. It is imperative that a de-compensating extradural haematoma is evacuated as soon as possible (and certainly within four hours). Pelvic reconstruction, on the other hand, may be best planned to take place after the vulnerable period, typically between days 2 and 4, when major operative interventions are associated with a greater risk of organ failure.

### **Stabilisation versus delay before setting off**

In general, meticulous stabilisation minimises the risk of decompensation en route. Hasty preparation with inadequate evaluation may lead to a separate injury being missed. On the other hand, the need to stabilise must not become an excuse for sluggish or disorganised arrangements. The underlying injury that needs emergency specialist intervention at the receiving centre may continue to deteriorate and be unsalvageable by the time the destination is reached.

In the severely head-injured patient, it is easy to overlook intra-peritoneal bleeding on clinical examination. An ultrasound investigation or CT scan is an essential adjunct to the head CT in an unconscious trauma patient. If significant bleeding is discovered, it must be controlled before transfer so as not to compromise cerebral perfusion en route. The general rule is to control an A (airway), B (breathing) or C (circulation) problem before a D (disability i.e. nervous system) one.

### **De-stabilisation versus delay en route**

It is important to minimise travel time, during which the patient is separated from the extensive resources found within a hospital. Nevertheless, travelling too fast round corners and roundabouts is unnecessary and may cause cardiovascular compromise in vulnerable patients, as well as putting more strain on straps and other attachments.

Spinal-injured patients were previously taken by road extremely slowly to avoid jolts, but this led to prolonged journey times and continual pressure on susceptible areas of skin. With an efficient police escort, the road ahead can be cleared, minimising the need to brake. A useful maxim is to go fast on the straights and slow round the bends.

In severely hypoxic patients (e.g. with severe ARDS being transferred for ECMO therapy in a supra-regional centre), two difficult risk-benefit decisions may be required: (1) Is the patient fit for transfer? (2) Does the speed benefit of a helicopter outweigh the adverse effects of altitude?

### **The mode of transport**

Most inter-hospital transfers within a range of 50 miles are by land ambulance. Air transport offers advantages for longer distances, especially if one of the hospitals is poorly accessible by road. The choice of transport depends not only on the provision and response time of local services, but on weather and traffic conditions at the time.

### **By trolley on foot**

Every intra- and extra-hospital transfer involves a journey on a trolley (or a bed). It is easy to overlook this essential component, focusing on the vehicle alone. As in journeys by ambulance, the trolley route must be planned and personnel assigned to perform the equivalent of the police escort through a town – clearing the corridors, freeing lifts and unlocking doors.

For inter-hospital transfer, the trolley and equipment used to reach the ambulance is generally used for the entire journey. Sometimes, it is tempting to switch over to a dedicated ventilator on entering the ambulance (especially a helicopter). However, if the patient has a critical head injury with a well-controlled  $p_aCO_2$ , confirmed by repeated arterial blood gas analysis, it makes good sense to continue using the same ventilator rather than to copy the settings to a separate ventilator at that stage. Alternatively, the ambulance ventilator can be taken into the hospital and used to stabilise the patient there, though this may slow down the transfer process.

### **Land ambulance**

Although continually improving in design, the back of an ambulance is still a confined space with limited access to the patient and temporary isolation from help, extra drugs, and extra equipment. It is not a hospital. Vibration, noise and poor lighting affect practical procedures and interfere with the reliability of monitoring devices.

Land ambulances are widely available, can be mobilised rapidly for high priority cases and are cheaper to run than air ambulances. They run at night and can stop in an emergency for critical interventions to be performed. Single cot vehicles with a centrally placed stretcher allow reasonable access to the patient. They represent the optimal means of transport for journeys of less than 20 miles and are worth considering up to 100 miles.

The design considerations for intensive care land ambulances shown below in Box 18.3 are taken from the latest Intensive Care Society Guidelines for the transport of the critically ill adult.

### **Helicopter**

An increasing number of secondary inter-hospital transfers are being carried out using helicopters. They are more than twice as fast as land vehicles, but are only faster overall if mobilisation is slick and helipad facilities are available close to the start and end points of the patient's journey. Supplementary transfers by land vehicles slow down the entire process, add to the amount of manual handling and increase the chance of de-stabilisation.

Helicopters are more expensive to run, are restricted by weather conditions and are not always available at night. Escorts need specific training in helicopter safety (e.g. approaching the vehicle in defined directions to avoid the rotors) and must follow instructions from the flight attendants carefully. The noise levels interfere with communication and vibration limits the performance of intricate tasks. They are generally not pressurised and typically fly at about 3000 feet, leading to the expansion of gas-filled spaces, such as a pneumothorax, pneumocephalus, bowel or endotracheal tube cuff. Nevertheless, in a well-organised system, secondary transfer by helicopter is ideal for emergency transfers between 30 miles and (given a larger vehicle, such as a Sea King) 200 miles.

### **Box 18.3: Design considerations for intensive care land ambulances**

(from Intensive Care Society 2002)

#### **Vehicle**

- Driven by suitably trained personnel
- Able to carry up to four members of hospital staff in addition to ambulance crew
- Seats for staff should ideally be rear facing or forward facing (not side facing)
- Seats to be fitted with head restraints and three point inertia reel seat belts
- Hydraulic ramp, winch or trolley system designed to enable single operator loading
- Patient trolley central mounted allowing all round patient access
- Stable comfortable ride with minimal noise and vibration levels
- Regular service and maintenance contracts

#### **Services (for UK)**

- Standard 12-volt DC supply. In addition:  
240-volts 50Hz AC power supply from an inverter or generator (Recommended minimum output 750 watts. This is generally sufficient to power a portable ventilator, monitor, and infusion pumps)
- Minimum of two standard 3 pin 13 amp outlet sockets in the cabin
- Minimum of two F size oxygen cylinders in secure housings  
Manifold system with automatic cylinder change over, and audible oxygen supply failure alarm  
Minimum of two wall mounted outlet valves for oxygen  
(Oxygen concentrators may be an alternative)
- Medical air supply is also desirable but the space required by additional cylinders or compressors may be a limiting factor
- Adequate lighting, heating, air conditioning, and humidity control

#### **Equipment**

- Mobile telephone to enable communication with referring/receiving hospital
- Defibrillator and suction equipment
- Adequate storage and stowage for ancillary equipment

#### **Fixed wing aircraft**

Fixed wing aircraft are most valuable in remote areas separated from urban centres by large distances, especially islands (e.g. Orkney and Shetland). They are faster and even more expensive than helicopters. They generally require two dedicated landing strips, usually at airports. Longer supplementary journeys by land ambulance are inevitable. Nevertheless, for

journeys of more than 200 miles, the overall journey time is often less than other forms of transport, though the logistic barriers must not be under-estimated. It is difficult to achieve an instant time slot at a busy airport, even for an emergency situation.

Fixed wing vehicles are generally pressurised to 5000 – 8000 feet, creating more hypobaric problems than a helicopter flying at 3000 feet. They are generally more spacious, less noisy and less prone to vibration and sudden movement than helicopters.

### **Retrieval versus transfer**

The patient may be transferred out by escorts from the primary receiving hospital or retrieved by clinicians from the secondary receiving hospital or by a dedicated stand-alone transfer team. Retrieval teams are routinely used in the secondary transfer of paediatric patients to specialist paediatric centres. A key advantage of retrieval from a specialist unit is that the escorts may have particular training and expertise in dealing with the patient's condition (e.g. neuro-critical care) or with the particular group of patients (e.g. small children). A regional stand-alone transfer service is able to provide escorts with a more concentrated experience of transfer, allowing them to enhance their skills. By providing a separate service, there is no depletion of staff from the primary and secondary hospitals, but separate funding arrangements are needed.

Retrieval requires the escorts to make two journeys before the patient can be treated at the destination, leading to an intrinsic delay. Nevertheless, a slick retrieval service that can be mobilised promptly and liaise en route with the clinicians stabilising the patient may still be quicker than an ad hoc transfer team assembled at the primary hospital.

A retrieval service covering a small area will have prolonged slack periods. A service covering a larger area will be busier, but with a higher risk of simultaneous transfer requests and the need to cover greater distances. If the retrieval service is unavailable, the primary hospital may be asked to perform the transfer, an activity which they may have become deskilled.

An alternative to both transfer and retrieval is taking a specialist surgeon to the patient in an extreme situation. While this may appear to be an attractive option for the individual patient, it depletes resources at the specialist centre and is rarely a practical option. The intervention is best carried out by a practised operating team rather than by a surgeon working with unfamiliar staff and ongoing specialist care will generally be needed afterwards.

## **Escort selection and training**

Inter-hospital transfer is a multidisciplinary activity and a team approach is required. Due to the isolated working environment and the often complex nature of the patient's medical condition, team members need to acquire specific skills and judgement.

### **Competencies and experience**

Medical, nursing and paramedic training is increasingly focusing on competencies, rather than simply on seniority and experience. The Inter-Collegiate Board for Training in Intensive Care Medicine has set down broad requirements in specialist training guidelines (Box 18.4). A more detailed listing of competencies can be found in the STaR manual.

#### **Box 18.4: Competency-based Training in Transport Care**

(from Intercollegiate Board for Training in Intensive Care Medicine)

**Overview:** Critically ill patients are frequently moved, either within the ICU to a different bed space, or within hospital for diagnostic radiology or for surgical procedures, or between hospitals. The principles of safe transfer are the same, regardless of the distance travelled. All trainees should gain supervised experience in safe transfer. Inter-hospital transfer in particular requires a high level of expertise because additional help cannot be obtained if problems occur.

#### **Knowledge**

- Principles of safe transfer of patients
- Understanding portable monitoring systems

#### **Skills**

- Intra-hospital transfer of patients requiring ventilatory support alone
- Inter-hospital transfer of patients with single or multiple organ failure

#### **Attitudes & behaviour**

- Insistence on stabilisation before transfer
- Pre-transfer checking of kit and personnel
- Planning for and prevention of problems during transfer
- Communication with referring and receiving institutions and teams
- Insistence on adequate support from senior / more experienced colleagues

#### **Workplace training objectives**

- Supervised intra-hospital transfers of ventilated patients to theatre for diagnostic procedures (e.g. CT scanning)
- Inter-hospital transfers of ventilated patients with or without support of other organ-systems

It has been recommended that anaesthetists should accompany ventilated head-injured patients. Other medical specialists (e.g. emergency physicians and non-anaesthetist intensivists) also make suitable escorts, provided that they have proven competency in acute trauma management, resuscitation, airway care, mechanical ventilation, other organ support and intensive care monitoring techniques. In some parts of the world, paramedics or nurse practitioners have been trained to serve as extremely competent escorts for major trauma patients.

Component roles from four different clinical disciplines provide the necessary elements for the transfer team (Box 18.5).

**Box 18.5: Component roles for a transfer team**

1. Medical (anaesthetist, intensivist, emergency physician, or other doctor with appropriate training for clinical assessment and intervention)
2. Nursing (emergency department or intensive care nurses to provide holistic care and communicate with team members, the patient, and the patient's family)
3. Technical (operating department practitioner or intensive care technician to operate and troubleshoot problems with machinery such as monitors and ventilators)
4. Transport (ambulance paramedics or technicians to handle and secure the patient together with being responsible for the mobile environment).

**Training courses**

All escorts should undergo formal training and be directly supervised until deemed competent. Attendance on courses specific to training in inter-hospital transfer, such as the Safe Transfer and Retrieval (STaR) Course (Advanced Life Support Group 2006), should become a mandatory requirement.

Multidisciplinary training allows for members from each of these backgrounds to learn to perform complimentary skills. The exact number of accompanying personnel will then depend on the clinical circumstances in each case. A complex, unstable case may require a team that draws from all of the specialist areas. Overlapping skills will allow a smaller team to manage most cases safely. Good communication is essential at all stages in the transfer process and must be incorporated into training courses as a fundamental consideration.

## **Guidelines and working practices**

### **National guidelines**

The Association of Anaesthetists of Great Britain and Ireland have recently updated their guidance on transfer of the brain-injured patient. So far as possible, these recommendations have been kept in line with those from the Brain Trauma Foundation, the National Institute for Clinical Excellence and Advanced Trauma Life Support. Examples of other national guidelines are given in as further reading (below).

While national guidelines should not be considered mandatory, they deserve serious respect when based on current evidence and when tailored to be as compatible as possible with other accepted trauma care guidelines.

### **Local arrangements**

National guidelines can never take into account all of the detailed arrangements needed at the local level. Local arrangements should be defined proactively so that valuable time is not wasted making ad hoc arrangements at the time of need.

One of the most frustrating recurrent situations for district hospitals to deal with is the lack of a suitable intensive care bed for a severely head-injured patient in the regional neurosurgical centre. Who should be responsible for finding an alternative bed in another region: the referring hospital clinicians who are busy stabilising the emergency patient or the neurosurgeons, the recognised experts who have assessed the transmitted CT scan and are responsible for severe head injury management throughout the region? By default in most regions, the burden falls on the referring team.

Some head-injured patients require emergency decompression of immediately life-threatening intra-cranial haematomas. They need an operation rather than an intensive care bed, at least in the first instance. The neurosurgical centre may need to accept the patient even when there is no bed available. An example of guidelines for this situation is shown in Box 18.6 (with permission from STaR).

### **Box 18.6: Guidelines for the Transfer of Patients with Acute Life-threatening Intracranial**

#### **Space-occupying Lesions**

(from Safe Transfer and Retrieval, Advanced Life Support Group, 2006)

In the following circumstances, the Consultant Neurosurgeons are empowered to accept an emergency transfer from those hospitals in the Transfer Group for which the Neurosurgical Service is specifically responsible, even if there is no available intensive care bed in the Neurosciences Centre, provided that an appropriately staffed and equipped operating theatre can be made available for emergency neurosurgical intervention:

#### **Acute life-threatening intracranial space-occupying lesions:**

- **That are predicted to deteriorate rapidly with a high risk of death or disability unless treated surgically without delay**
- **That are judged to be sufficiently urgent to transfer straight to the operating theatre for life-saving surgery before admission to a ward**
- **In which a favourable outcome is potentially achievable with prompt surgical treatment**

**In all other circumstances**, the availability of an intensive care bed must be established before accepting a transfer from another hospital with a neurosurgical emergency which is likely to need intensive care.

If acute neurosurgical intervention is considered to be futile, the Consultant Neurosurgeon may refuse the patient, irrespective of bed availability. Advice on continuing care or withdrawal of care should still be provided to the referring clinician.

#### **Equipment, drugs and fluids**

A critically-injured patient should be transferred in a portable intensive care environment. All equipment must be robust, durable and lightweight. Everything should be serviced regularly and checked both daily and immediately before using for transfer. The transfer team must be familiar with the equipment (Box 18.7) and drugs (Box 18.8) in the transfer kit and with that available in the transfer vehicle itself.

#### **Airway equipment**

Patients with a vulnerable airway should be intubated before transfer and all tubes must be well secured. Nevertheless, a self-inflating bag-valve-mask, pharyngeal tubes, a pair of working laryngoscopes, a range of endotracheal tubes and a bougie are all essential. Equipment for cricothyroidotomy is also needed as a backup.

### **Oxygen supply**

A monitored oxygen supply is required with enough reserve to cover a doubling of the expected journey time in case of breakdown or other delay. Lightweight D-cylinders containing 340 litres of oxygen when full and lasting for up to  $\frac{1}{2}$  hour at 10 litres/minute are often used to transfer patients to and from the ambulance. Ambulances usually have F-cylinders that each contain up to 1360 litres and last up to  $2\frac{1}{4}$  hours at 10 litres/minute. Ambulance suction devices are commonly run from the oxygen cylinders and this need should not be overlooked on long journeys. Intensive care units commonly use E-cylinders (680 litres lasting 1 hour at 10 litres/minute) for journeys to and from the CT scanner.

### **Power supply**

Electrical equipment needs to be able to run reliably on batteries and spares, with a much longer life than the maximum possible journey time. Maintenance programmes should be in place to ensure that certain batteries are periodically fully discharged and re-charged to maximise battery life. Modern ambulances are equipped with electrical power inverters and equipment such as syringe pumps can usually be connected directly into the ambulance power supply.

**Box 18.7: Essential equipment for transfer**

- Portable monitor with ECG, NIBP,  $\text{SaO}_2$ ,  $\text{EtCO}_2$ , three invasive pressures, and temperature
- Portable sucker (which does not depend on electricity or gas)
- Self-inflating bag-valve-mask and intubation equipment, including back-up cricothyroidotomy
- Portable ventilator with disconnection and high pressure alarms, and variable settings ( $V_T$ , rate,  $\text{FiO}_2$ , PEEP, I:E ratio)
- Extension hose with Schraeder probe (as extension to ventilator oxygen hose to reach distant supply)
- Rucksack transport kit with respiratory and cardiovascular equipment rolls (including venous access equipment) and a drug pack
- Chest drain equipment
- Battery powered syringe drivers
- Battery powered IV volumetric pumps
- Defibrillator
- Urinary catheters
- Warming blanket
- Spare batteries
- Head light
- Protective clothing (high visibility clothing and protective helmet)
- Communication aids
- Scissors and tape

**Transport ventilator**

Portable mechanical ventilators must have disconnection and high-pressure alarms. Variable inspiratory/expiratory (I/E) ratio capability is invaluable in severe pulmonary contusion or ARDS. The ability to provide pressure controlled ventilation, pressure support and continuous positive airway pressure (CPAP) is desirable, but rarely available on transport ventilators.

**Transport monitor and infusion devices**

A multi-channel, portable monitor with a clear illuminated display and resistance to motion artefact is essential. Alarms need to be visible, as well as audible, in view of the noisy environment. The following modalities should be available: pulse oximetry, end-tidal capnography, ECG, non-invasive blood pressure, at least two invasive intra-vascular pressures

and temperature. Capnography serves as an excellent disconnection warning and is now considered to be essential for transfer of the intubated patient. The end-tidal CO<sub>2</sub> readings are often used as a rough guide to arterial values, but remember that the end-tidal value may be considerably lower than the arterial in the face of hypotension, low cardiac output or serious lung injury. When the end-tidal CO<sub>2</sub> is high, so is the arterial pCO<sub>2</sub>. When it is low, we cannot assume that the arterial value is low too.

Syringe drivers and infusion pumps should be used in preference to gravity-dependent drips, which are inaccurate and unreliable in moving vehicles. Ideally, pumps should be mounted below the level of the patient and infusion sets should be fitted with anti-siphon valves.

### **Transport trolley**

A well-designed, dedicated transfer trolley can simplify the whole transfer process. This should be chosen in full consultation with the local ambulance service and must be compatible with local ambulance specifications to fit in place in the vehicle. The trolley should comply with CEN standards so as to be able to withstand the substantial forces applied to it in a range of conditions, including collisions.

Ideally, transport trolleys should be capable of being tipped head down to help deal with a vomiting patient who is not intubated, though this may require a more complex and accordingly heavier design.

Some vehicles use collapsible trolleys to minimise the lifting involved in loading the trolley. This manual handling advantage is offset by the need to store heavy items of equipment above rather than under the patient, leading to a higher centre of gravity and amore unstable trolley.

### **Drugs and fluids**

The main drug requirement in transit is for nervous system control: analgesia and, if intubated, sedation and paralysis. Vasoactive drugs may occasionally be needed (e.g. vasopressors for head injuries to help achieve an adequate cerebral perfusion pressure or vasodilators in contained aortic injuries to help prevent catastrophic vessel disruption. Cardiovascular resuscitation drugs must always be at hand. Mannitol should also be available to buy time in the face of a de-compensating intracranial haematoma.

Blood that has been cross-matched should be taken en route in a dedicated cool-box, together with the formal documentation to allow it to be used safely en route or at the destination.

Crystalloids and colloids should be taken in sufficient quantity to allow for traffic delays, although the stocks in the ambulance will provide a supplementary supply.

#### **Box 18.8: Essential drugs for inter-hospital transfer**

- Hypnotics/sedatives e.g. midazolam, propofol, etomidate, ketamine
- Muscle relaxants e.g. suxamethonium, atracurium, cis-atracurium, vecuronium, rocuronium
- Local anaesthetics e.g. lidocaine, levo-bupivacaine
- Analgesics e.g. fentanyl, morphine
- Anticonvulsants e.g. diazepam, thiopentone (thiopental)
- Mannitol
- Inotropes/vasopressors e.g. epinephrine, norepinephrine, metaraminol, ephedrine
- Resuscitation drugs according to Resuscitation Council guidelines

#### **The phases of transfer**

Inter-hospital transfer can be considered in five phases as outlined in Box 18.9.

#### **Box 18.9: The five phases of inter-hospital transfer**

1. Initial assessment: identification of injuries with resuscitation and stabilisation of physiological systems
2. Consultation and referral: decision to transfer and communication with the receiving centre and transport agency
3. Preparation for transfer
4. Transportation: handling the patient and the environment
5. Handover and return

##### ***1. Initial assessment***

Assessment, resuscitation and stabilisation should follow the recommendations as laid out in Section 1.7. Trauma care is often time-critical and unnecessary delays must be avoided. However, failure to identify injuries and thoroughly stabilise the patient prior to transfer may lead to serious, life-threatening complications in transit. A patient persistently hypotensive despite resuscitation must not be transported until all possible causes of the hypotension have been addressed. Correctable major haemorrhage should be controlled prior to transfer. A fundamental requirement before transfer is to ensure satisfactory and stable organ perfusion

and tissue oxygen delivery. Following severe head injury, it is essential to avoid hypoxia, hypotension and hypercarbia to reduce the risk of secondary brain injury.

## **2. Consultation and referral**

It is not necessary to wait until the entire set of initial investigations is complete before considering transfer or indeed contacting the receiving unit. However, the referring clinician must possess sufficient knowledge about the patient's overall condition in order to present a sufficiently accurate account for which to base the consultation: make sure you have a clear understanding of the sequence of events, the list of injuries so far defined and the patient's current physiological status. The initial contact with the receiving hospital can be a daunting process. Be prepared for discerning questions from the receiving specialist.

The need to transfer a patient to another unit may be evident from the outset (e.g. a severe head injury with lateralising features) or it may be less obvious and only revealed after detailed investigation. The need may arise from other factors, such as a lack of intensive care beds. Whatever the circumstances, the final decision to transfer a patient to another hospital must be made at consultant level.

It is essential for a plan of transfer to be agreed between the referring and receiving consultants. This will include:

- Any further investigations or interventions prior to transfer
- The mode of transfer (e.g. land or air ambulance)
- The timing of transfer
- The exact location to which the patient should be transferred

As soon as a decision has been made to transfer, the ambulance control should be contacted. The ambulance service needs to be involved in deciding on the mode of transfer and will need some time to mobilise sufficiently experienced crews. Any delay contacting the ambulance service will lead to unnecessary delays in setting off.

Sensitive communication with the patient (if awake and competent) and the family must not be forgotten. They should be made fully aware of the clinical situation and the reasons for transfer.

**Good communication is a cornerstone of a successful and uneventful transfer. This is never more important than during the initial referral and at the time of the eventual handover.**

### ***3. Preparation for transfer***

Preparation for transfer begins with proactive investment and training. Checklists prior to transfer are invaluable (Martin 2001) and will ensure that no aspect of the transfer process is omitted, thereby reducing the potential of adverse incidents.

As soon as the transfer has been agreed with the receiving centre, the ambulance service should be contacted to arrange an appropriate transfer vehicle at an agreed time or, in a life-threatening situation, as soon as possible.

The transfer team should be assembled as soon as possible to assimilate all the details relevant to the patient and to contribute to the preparation for transfer. Indeed, some of the team members may have been involved from the outset as trauma team members. Communication within the team is essential if they are to cope with a complex case. They must be fully aware of each other's roles and responsibilities.

Some of the interventions prior to transfer may be in response to advice from the receiving unit. For example, a burns specialist may suggest adjusting the intravenous fluid regime in a major burn. Until handover has been completed, the transferring team must still retain responsibility and use initiative to make further adjustments according to the urine output and the overall clinical picture.

Some preparations are generic, such as securing lines and tubes, and keeping the patient well wrapped and warm. The patient should have been moved off the spinal board during the initial assessment. Spinal precautions should be continued if a spinal injury has been identified or if one cannot be excluded. For the secondary transfer, a vacuum mattress is preferable to a hard spinal board, though a well-padded board may be acceptable for a short transfer.

#### **When in doubt, intubate prior to transfer**

### *Airway*

A key consideration at this stage (as at all stages) is airway management. Intubating a complex trauma patient, with an uncleared spine, is difficult enough in the resuscitation room, let alone in the confined space of an ambulance. The indications for intubation after head injury are shown in Box 18.10.

### *Breathing*

Ten to fifteen minutes after connection to the portable ventilator, the arterial blood gases should be analysed and adjustments made to the ventilator settings if necessary. If the preparation time is prolonged, blood gases analysis should be repeated at least hourly and always just before transfer. Intubated head-injured patients should be transferred with a  $P_aO_2$  greater than 13 kPa and a  $P_aCO_2$  of 4.5 – 5.0 kPa. In the face of clinical or radiological evidence of raised intracranial pressure, it is acceptable to reduce the  $P_aCO_2$  to 4.0 – 4.5 kPa.

In the presence of a pneumothorax or significant fractured ribs, a chest drain should be inserted prior to transfer. A large bore gastric tube must be passed in an intubated patient.

### *Circulation*

Hypovolaemic patients tolerate transfer poorly and intravenous volume loading may need to be undertaken prior to transfer. Even after surgery, do not underestimate ongoing losses from other sources, such as fracture haemorrhage and third space losses. A central venous catheter can be used to optimise filling and administer drugs or fluids during transfer. Other haemodynamic monitoring devices (PiCCO, LiDCO) or oesophageal Doppler techniques may be used to optimise filling cardiac output prior to transfer, but are impractical in transit.

### *Nervous system*

If intubated, the patient must be kept adequately paralysed and sedated. Ongoing sedatives and analgesic drugs are best delivered by infusion.

In the severely head-injured patient without an intracranial pressure monitor, the mean arterial blood pressure should be kept above 80 mmHg. In patients with potential trunk bleeding, who cannot be operated on at the referring hospital (e.g. in a tear of the thoracic aorta being referred to a cardiothoracic centre), the systolic blood pressure should be kept low (ideally at about 100 mmHg). This may necessitate the use of vasodilators. On the rare occasion where both injuries co-exist, a compromise will be needed, though when in doubt act according to priorities in ABCD order.

### *Injuries*

Dislocations and significantly displaced fractures should be reduced and immobilised prior to transfer. Open wounds should be irrigated with sterile saline, if contaminated and then covered to reduce the risk of infection. Open fractures should be inspected once and a photograph taken to allow others to visualise the wound. Depending on the urgency of transfer and the degree of contamination, formal debridement in an operating theatre may be necessary before commencing the transfer. The need for tetanus prophylaxis should always be considered.

#### **Box 18.10: Indications for intubation and ventilation after head injury**

(from The Association of Anaesthetists of great Britain & Ireland 2006)

- Glasgow Coma Score  $\leq 8$
- Significantly deteriorating conscious level, even if not in coma (fall in motor score of two points or more)
- Seizures
- Loss of protective laryngeal reflexes
- Ventilatory insufficiency as judged by blood gases
  - Hypoxaemia ( $\text{PaO}_2 < 13 \text{ kPa}$  on oxygen)
  - Hypercarbia ( $\text{PaCO}_2 > 6 \text{ kPa}$ )
- Spontaneous hyperventilation causing  $\text{PaCO}_2 < 4 \text{ kPa}$  or significant respiratory arrhythmia or other abnormal breathing pattern
- Bilateral fractured mandible
- Copious bleeding into mouth (e.g. from skull base fracture)

### *Final preparation*

Just prior to setting off, a pre-transfer checklist should be run through to minimise the risk of forgetting anything. Case notes (preferably the full set of originals rather than copies), investigation results, diagnostic images (films or digital imaging files with hard copies too if the receiving centre does not have a compatible digital system in the operating theatre) and a referral letter should accompany the patient. Cross-matched blood and other blood products if appropriate should be taken, together with the formal documentation. A sample transfer checklist for neurosurgical patients is shown in Box 18.11.

**Box 18.11: Transfer checklist for neurosurgical patients**

(from Association of Anaesthetists 2006)

System	Checklist
Respiration	<p><math>\text{PaO}_2 &gt; 13 \text{ kPa}</math>, <math>\text{PaCO}_2 4.5 - 5.0 \text{ kPa}</math> (or <math>4.0 - 4.5 \text{ kPa}</math> if clinical or radiological evidence of raised intracranial pressure)?</p> <p>Airway clear and protected adequately?</p> <p>Intubation and ventilation required?</p> <p>Nasogastric or orogastric (if risk of basal skull fracture) tube in situ (if intubated)?</p>
Circulation	<p>Mean arterial blood pressure <math>&gt; 80 \text{ mmHg}</math> (adults)?</p> <p>Pulse <math>&lt; 100/\text{min}</math> (adults)?</p> <p>Peripheral perfusion adequate?</p> <p>2 reliable large iv cannulae in situ?</p> <p>Estimated blood loss already replaced?</p> <p>Arterial line sited?</p> <p>Central venous access needed?</p>
Head injury	<p>GCS, GCS trend (improving/deteriorating)?</p> <p>Any focal signs?</p> <p>Skull fracture?</p> <p>Anticonvulsants?</p> <p>Mannitol needed (discussed with neurosurgeons)?</p>
Other injuries	<p>Cervical spine injury (protection in place), chest injury, fractured ribs, pneumothorax excluded/treated?</p> <p>Intrathoracic, intra-abdominal bleed?</p> <p>Pelvic, long bone fracture?</p> <p>Extra cranial injuries toileted, dressed and splinted?</p>
Escort	<p>Doctor, nurse, ODP, ambulance crew adequately experienced?</p> <p>Instructed about the case?</p> <p>Adequate equipment and drugs?</p> <p>Adequate oxygen supply?</p> <p>Can use equipment and drugs?</p> <p>Case notes, investigation results, radiology films and reports, and referral letter?</p> <p>Transfer documentation prepared?</p> <p>Contacted the receiving unit and know exactly where to go to?</p> <p>Telephone numbers programmed into portable phone?</p> <p>Portable phone battery fully charged?</p> <p>Name and bleep number of receiving doctor?</p>

Cash/credit cards in case of emergency?

Family aware of details?

The relatives require accurate directions to the receiving hospital, together with contact telephone numbers. These should be pre-printed for the hospitals used as regular destination units. On no account should relatives attempt to keep pace with the transferring ambulance. Ideally, they should follow on at least fifteen minutes after the patient and transfer team have left for the receiving hospital.

#### **4. Transportation**

On leaving, the receiving hospital should be re-contacted and given an estimated time of arrival. Provided the patient has been meticulously investigated, stabilised, and packaged prior to setting off, transportation should be relatively straight forward, requiring few if any, interventions en route. If there is any doubt about the patient's stability, then the transfer should be delayed. Monitoring must be continuous throughout the transfer.

Sedation and paralysis should be optimised before departure (and just before arrival at the destination) to ensure that sudden movement (e.g. moving the trolley into the vehicle or transferring the patient on to ambulance trolley), does not cause coughing, gagging or limb movements.

The senior medical attendant present should determine the speed of travel in liaison with the driver. The aim is a smooth and steady ride. The use of a police escort will be dictated by local policy.

Staff should remain seated and belted whenever possible. If an escort has to undo the seatbelt to attend to the patient, then the tripod position should be used (kneeling with widely spaced knees). Do not stand and sway!

A continuous written record must be kept during the transfer, with particular reference to critical incidents.

#### **5. Handover and return**

Responsibility for the patient remains with the transfer team until an agreed time after handover at the receiving hospital. The handover should include a clear, concise verbal and written account of the patient's history, vital signs, therapeutic interventions, and significant clinical

events both prior to and during transfer. A template for a structured handover at the receiving hospital is recommended in Box 18.12.

After handing over, the transfer team may need a brief period of refreshment before returning back to their hospital. A debriefing session should be considered after complicated transfers. All transfers should be audited and a system for reporting critical incidents should be in place.

#### **Box 18.12: Handover template**

##### **Immediate information**

- Personnel: introduce yourselves
- Patient: introduce your patient
- Priority: indicate any major problem that needs immediate attention

##### **Case presentation**

- Presentation: mechanism and time of injury
- Problems: simple list of injuries and other major problems
- Procedures: simple list of major interventions and investigations
- Progress: system review
  - Respiratory (e.g. airway, oxygenation and ventilator settings)
  - Circulatory (e.g. haemodynamic status and blood transfused)
  - Nervous system (e.g. conscious level and sedation/paralysis)
  - Metabolic (e.g. urine output and glucose level)
  - Host defence (e.g. temperature, antibiotics and steroids)

**Do not forget to return with ALL the equipment you took with you!**

#### **Summary**

The purpose of transferring trauma patients is to provide better care elsewhere. The risks involved must be outweighed by the likelihood of benefit. Careful stabilisation without undue delay reduces the chance of problems occurring en route. Good communication is vital throughout the transfer process. Multi-disciplinary escort training and the development of national and local guidelines are important components of an effective transport system.

## **Further reading**

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# **CHAPTER 19**

## **PSYCHOLOGICAL ASPECTS OF TRAUMA**

### **OBJECTIVES**

At the end of this chapter, the reader should be able to:

- Describe the presentation and management of
  - Normal reactions to highly stressful and traumatic events
  - Common acute psychiatric conditions
  - Grief reactions
- Provide guidelines for the breaking of bad news
- Highlight issues concerning the presence of relatives in the resuscitation room
- Describe the impact of patient care on staff
- Identify measures to help staff cope with the demands of their work

### **INTRODUCTION**

If the contemporary media were to be believed the resuscitation room would represent a high profile crucible in which the dramatic issues of life and death, romance, heroism, scientific inspiration and awesome clinical judgment were uniquely blended. The reality is different. Undeniably, of course, this setting is frequently dramatic and professionally challenging, but its demands can also take its toll of staff that work in it, and it poses major challenges in terms of patient management and staff welfare.

#### **Normal reactions to highly stressful and traumatic events**

For patients and relatives, the resuscitation room is likely to provoke anxiety and concerns about privacy, unfamiliar procedures and faces, and intimidating equipment. Our own familiarity with these matters should not prevent us from realising how alien these maybe to our patients and their relatives. Also, we need to remember that accidents and medical emergencies can cause marked reactions with which the layman may not be familiar.

The following are some of the reactions commonly observed:

- Numbness and shock: This is probably Nature's way of shielding us from otherwise overwhelming experiences.
- Fear: In particular it is helpful to reassure the "stiff upper lipped" male that fear is a normal biological reaction.

- Depression, apathy, and helplessness: Such reactions are commonly associated with sudden and unexpected “loss”, and medical emergencies are nearly always associated with real or anticipated loss. *All suicidal talk should be taken seriously, and therefore recorded and reported. Do not be reliant on the old adage, “If they talk about it, they won’t do it”.*
- Irritation and anger: These reactions may compromise safety and the stability of catheters, intravenous cannulas and our own composure. It is important, however, not to take such reactions personally.
- Guilt: If patients express guilt about their contribution to a tragic event it is important not to offer superficial reassurance, as it is unlikely we will know all the facts.
- Cognitive and perceptual distortions: Following intensely emotional events individuals’ memory may be distorted either in terms of the order of events or the speed at which they are perceived to have happened.
- Autonomic hyperarousal and hypervigilance: Heightened autonomic reactions and an increased sensitivity to risk represent Nature’s way of preparing us for the next threatening event. From a medical point of view, it is important to be aware of these changes as they may lead in the longer term to self-medication i.e., the use of alcohol and other psychoactive substances as patients try to “calm down”.
- Intrusive experiences: “Flashbacks” are commonly experienced following some traumatic event. These may involve any modality but are characteristically distressing and beyond conscious control. Nightmares are a related phenomenon. They represent an inability of the brain to process deeply disturbing memories and experiences.

### **Psychological first aid**

This approach was first described in the 1980s and subsequently developed by Alexander in 1990. Described below are some basic ways we can help victims of tragic events in the early stage to ensure matters do not become worse.

- Comfort and protect: A traumatic event leaves people feeling bewildered and shocked. Sometimes, they lose sight of further risk to themselves and of the need to protect themselves against further risk. As a consequence, they may even fail to recognise the need for urgent medical care.
- Counteract helplessness: One of the hallmarks of trauma is that dreadful moment when the individual feels totally helpless and hopeless in the face of some distressing event. That feeling may carry itself over into the resuscitation room. Whenever possible, therefore, counteract that sense of hopelessness and helplessness by involving individuals in their own care or in the care of others, for example, by providing information, comfort and reassurance or by contacting other relevant persons in the family.

- Reunion with friends or relations: Family networks are an essential ingredient in trauma care. It is very important that professionals do not usurp the role of the family who are in a better position to provide a degree of care and support for their loved ones. Thus, we should allow time and provide the facilities for victims to contact their loved ones.
- Expression of feelings: It is not helpful to attempt to dredge up deep felt and painful feelings. It is better to reassure patients and their relatives that it is alright to express their feelings however unpleasant.
- Provision of accurate information: Information is a powerful antidote to uncertainty and anxiety. Giving people accurate information helps them to feel more in charge of their circumstances and welfare. It is, however, important to remember two things. First, the information must be accurate. (If we do not know the answer to a question, we should be honest about this and confirm we will do our best to find out the appropriate information.) Second, stressed individuals can only take in so much information at a time. We need, therefore, to *titrate* the dose of information.
- Re-establish order: Many of the above steps will contribute to this. A characteristic feature of trauma is that victims feel their world has been turned upside down and they are out of control and helpless. The composure of staff, conveyed by word and by behaviour, can do much to indicate that they are in safe and competent hands.
- Triage: Identify individuals who may need additional support or expert help. Certain circumstances may make it particularly difficult for patients and their relatives (see below). Beware also of the individuals who are particularly quiet; they may be more at risk than those expressing openly their distress.

## **Acute psychiatric conditions**

The resuscitation room is not normally the domain of mental health professionals; their contribution is usually made once the patient is physically stable. Occasionally, however, acute psychiatric conditions may require expert help. Below are a number of such conditions, which may confront clinical staff in the resuscitation room. For those interested, details about such conditions and their management can be found in further reading.

### **Panic attacks**

The dominant symptoms are likely to be:

- Sudden onset of palpitations and chest pain
- Choking sensations
- Dizziness
- Sense of unreality

- A fear of dying or going mad
- Waves of intense fear, provoking flight

### **Delirium**

Delirium is characterised by impaired consciousness and attention, and is commonly associated with hallucinations, delusions, disorientation, social withdrawal, impaired memory for recent events, impaired sleep, and either underactivity or overactivity. In addition to these physical and intellectual symptoms, there may be emotional disturbances such as depression (or, sometimes, euphoria), anxiety, irritability, apathy and perplexity. The onset is usually rapid, and the symptoms show diurnal variation (commonly worse at night). The causes are varied and include:

- Hypoxaemia
- Hypotension
- Cerebral haemorrhage
- Hypoglycaemia
- Meningitis
- Renal or hepatic failure
- Extended sleep deprivation
- Drugs (including steroids) and drug withdrawal
- Heavy and extended alcohol misuse or withdrawal ("delirium tremens" – see below)

### **Acute stress reaction**

This transient disorder is caused in otherwise emotionally stable individuals by an exceptionally stressful or traumatic event. The symptoms usually resolve within three to four days or sometimes even within a few hours after the individual is removed from the source of stress or threat.

The symptoms include a mixture of autonomic, mental, and psychological ones.

<b>Autonomic</b>	<b>Mental</b>	<b>Psychological</b>
tachycardia sweating flushing	disorientation confusion amnesia	dazed depression anxiety overactivity anger despair

### **Dissociation**

In dissociative conditions individuals commonly display marked denial of problems or events that are obvious to others (even the death of a loved one). These symptoms may be triggered by traumatic events, and they may help the victims to cope with otherwise intolerable stress.

There may also be otherwise inexplicable physical symptoms, such as deafness, paralysis, “pseudoseizures”, or an amnesia for events relating to the trauma. Such amnesias are usually selective and partial. They may show a curious lack of concern about such symptoms.

### **Alcohol-related problems**

Resuscitation room staff will be all too familiar with the problems of alcohol (in their professional lives!). About a quarter of emergency admissions to hospitals in England and Wales are due to excessive alcohol consumption, and alcohol is implicated in over three quarters of fatal road traffic accidents.

- Patients who are acutely intoxicated may display problems of violence (see below), but, fortunately, such behaviour tends to be short-lived and dose-related.
- Those who have an extended history of alcohol misuse may present with the characteristic “alcohol withdrawal symptoms” outlined in Box 19.1.

**Box 19.1: Alcohol withdrawal symptoms**

- Tremor (fine and rapid)
- Nausea
- Excessive sweating (especially in the morning)
- Tinnitus
- Hyperacusis
- Muscle cramps
- Impaired sleep (particularly with early morning wakening and nightmares)
- Variable mood (including irritability and anxiety)
- Perceptual disturbances (including visual hallucinations)
- Convulsions (which characteristically appear about 24 hours after alcohol withdrawal)

*Delirium tremens (DT)*

About 5% of physically addicted drinkers experience delirium tremens (the DTs) following withdrawal, and, if untreated, may result in cerebral obtundity, convulsions, and death. It tends to occur between one and four days following withdrawal. Common features are shown in Box 19.2.

**Box 19.2: Typical features of delirium tremens**

- Vivid visual hallucinations
- Delusions
- Ataxia
- Hyperarousal
- Insomnia
- Agitation
- Impaired concentration
- Profound confusion and disorientation
- Low grade pyrexia
- Fear

*Management of alcohol withdrawal*

- The acute management comprises rehydration, restoration of electrolyte imbalance, and thiamine injections when there is a risk of acute encephalopathy.

- If there is also evidence of hypoglycaemia, oral or parenteral glucose replacement is essential, supplemented by thiamine to avoid Wernicke's encephalopathy.
- Since there is a risk of fitting, long half-life benzodiazepines (e.g., diazepam or chlordiazepoxide) are recommended. In the cases of head injury and respiratory disorder, care must be taken with the use of such medication.
- As a general principle the administration of a vitamin supplement is advisable.

### **Post-traumatic stress disorder (PTSD)**

This condition is associated with very distressing reexperiencing of the traumatic event in the fashion of flashbacks and nightmares (see case example), avoidance of any reminders of the event (e.g., talking about it, going back to the scene, or even meeting people associated with it), and hyperarousal and hypervigilance (see above).

This condition is not diagnosed until about a month after the trauma, therefore, it is rarely a condition that will confront staff of the resuscitation room. However, since it has obtained such a high profile (thanks, in large, part to the media and to the legal profession) the features are described in Box 19.3.

#### **Box 19.3: Case example of post-traumatic stress disorder**

- The senior author was asked to examine a badly burned patient who would not use his PCA despite being in obvious pain. This puzzled the nursing and surgical staff.
- The explanation demonstrates how distressing such intrusive phenomena can be.
- The patient explained that if he used his analgesic he had no pain; if he had no pain he became sleepy; if he became sleepy he experienced flashbacks and nightmares in which he was burned and his son died.
- He preferred pain.

### **Grief reactions**

Grief is not an illness. Most individuals will deal successfully over time with tragic loss, but grief reactions are varied, occasionally dramatic, and may interfere with clinical management.

Because most deaths now occur in hospital and not at home most laymen have little personal experience with death and dying. Also, because of impressive technological advances in medical care, society now has become "death defying", in that they feel that no patient should

ever die once in expert medical care. (Staff may themselves see death as a “failure” – see “Impact on staff”.) Some deaths are particularly difficult to cope with (Box 19.4).

**Box 19.4: “Difficult” deaths**

- Sudden, unexpected
- Painful, horrifying, mutilating
- Where medical mismanagement is suspected
- Where there is no body (a body helps to confirm the reality of death and provides the opportunity for the bereaved to say their “goodbyes”)

It is also worth bearing in mind that, although the death of a loved one is the most obvious source of grief, in medicine there are many “mini deaths”. These include, the loss of a limb, the loss of function or the loss of looks (e.g., through traumatic disfigurement).

There is some overlap between the normal reactions to other stressful and traumatic events (see above) and those to loss. However, the following acute reactions should be noted in particular:

- Shock and denial: Patients and relatives often cannot take in the reality of the loss. This may be Nature’s way of shielding them from overwhelming stress. Thus, it is best not to be confrontational with regard to the “truth”.
- Apathy: Patients may show a lack of interest in their physical welfare (and, as a consequence, may not contribute to medical examination and treatment).
- Acute distress: There are commonly paroxysms of anxiety, depression, pining, agitation and crying. (With regard to the last, the archetypical “stiff upper lipped” male needs to be reassured that crying is a universal human response.)
- Guilt: This is commonly seen in events that have resulted in a fatality. It is important not to idly reassure that the individual is “not guilty” because, at the early stages, all the relevant facts will not be known.
- Anger: Relatives and patients may be extremely angry about an accident or medical emergency. They may not always know with whom to be angry; thus, staff may become the target. (See “Dealing with the bereaved”.)

**Dealing with the bereaved**

It is likely that how we deal with the bereaved in the early stages will have a marked bearing on how well they cope subsequently. It may also determine how they react to staff in the future.

Thus, however uncomfortable dealing with grief may make us feel, we must do our best for those who suffer a major loss. Below are some guidelines:

- Consider the setting: Try to find a quiet room, which guarantees a degree of privacy. We can be sure our pager will go off at the most intimate and inappropriate moment! Leave it with a colleague or switch it off.
- Listen: (When in doubt about what to say, say nothing!) The acutely bereaved rarely welcome being talked at. It is usually best, therefore, to confine ourselves to providing an opportunity for individuals to express their feelings and to ask questions.
- Tolerate their reactions: The bereaved are commonly angry (*"What did I do to deserve this?"*), bewildered, impatient and irritable (as described above). As caregivers, we may become the target of their feelings but it is important not to defend ourselves. (*"There is no point in being angry with me, I'm just doing my best."*) It is far better to show empathy and to "normalise" their reactions. (*"I can understand why you feel so upset and angry just now. I think anybody would feel like that."*)
- Ban the cliché: Dealing with those in grief can make us feel uncomfortable, insecure and anxious. Thus, we commonly are not sure what to say, and we may fall back on clichés. (*"It could have been worse, you know, your son might also have been killed."* – said to a mother whose daughter died in a road traffic accident.) Such comments can be hurtful.
- Identify sources of help: It is helpful to give people information about what they should do if their difficulties continue. There is of course the family doctor but, in almost any community, there are local voluntary agencies for example, CRUSE (see web sites) which are available to the bereaved. A brief (and accurate) leaflet listing such agencies and how they can be contacted is particularly helpful to the bereaved.

#### **10.6: Dealing with the bereaved**

- Consider the setting
- Listen
- Tolerate reactions
- Ban the cliché
- Identify sources of help

#### **Breaking bad news**

How the bereaved react to news of a tragic death (or other loss) is likely to be determined in part by how we tell them what has happened. However, it is never easy to do this, and there is no

blueprint for success. There are however some extended guidelines provided in the further reading.

Nobody wants to be the bearer of bad tidings, and, therefore, there is a strong temptation to avoid being in this position. (*"I'll leave it to Sister, she's had more experience!"*). It is the responsibility of the team, therefore, to consider who is in the best position (for whatever reason, e.g., age, seniority, gender or knowledge of the incident) to speak to the patient or relative. However difficult, it is a challenge that cannot be avoided.

Below, are three key questions you might like to consider if you are charged with this difficult responsibility:

1. Am I properly prepared in relation to:

- Whom I will meet?
- What information I might need?
- Where we can speak in privacy?
- How I look (clothing stained with bodily fluids may distress relatives)?

2. Have I thought about how I am going to share the bad news?

- It is usually helpful to provide our name, status and involvement in the proceedings.
- Find out what they already know and what they want to know – bear in mind, they may already have gleaned false information from bystanders, the emergency services or others.
- It is usually best to speak slowly and with pauses, and, every now and again, confirm that they have taken in what has been said. Do not forget that when we are anxious we tend to speak too quickly. Also, those in a distressed state may be confused, and their attention and memory will certainly not be at their best. Diagrams and notes can be particularly helpful.
- In an effort to protect people, we may be tempted to deceive or to, put it more bluntly, "lie". Well intended this may be, but it carries the strong risk of backfiring. If they find out we have been dishonest we are likely to lose their trust. Also, it is worth remembering, that contemporary society is now very much better informed about medical matters than it used to be.

3. What will I do once I have told them the bad news?

Sharing bad news with somebody is not just an event but also the start of process of helping him or her to deal with a tragic circumstance. Below, are some points we should bear in mind:

- Allow the individual time to digest what they have been told.
- Ask them if they would like a member of staff to be with them or if they would prefer to be alone.
- Ensure that they know what will happen next (e.g., legal proceedings, postmortem and recovery of possessions).
- Establish how they will get home and to whom and to what they are going home. (Individuals' ability to cope with a tragedy may be strengthened by the support they have at home or in the community but weakened by other pressures in their lives.)

### **Relatives in the resuscitation room**

This is an emotive and contentious issue about which much has been written recently (Mitchell & Lynch, 1997). Resistance to the presence of relatives in the resuscitation room is often based on the anxiety it creates in staff, the threat of litigation and the suspicion that relatives would not cope with what they see. More recent evidence confirms that relatives may gain considerably from their presence in the resuscitation room. The advantages of relatives being present could include:

- They can see that everything is being done to help their loved one.
- It may remove some of the "mystery" of what happens in the resuscitation room.
- It may help to develop a constructive bond between staff and relatives which may be very helpful in the aftermath and follow-up.

However, these gains are unlikely to be achieved unless we take the following steps:

1. Relatives need to be briefed as to what they will see, hear and, possibly, smell. Otherwise, they are likely to find the proceedings in the resuscitation room very disturbing and bewildering.
2. We need to ensure that there is a competent member of staff with them who can advise what is happening and offer support and reassurance as the proceedings unfold.
3. Ideally, the same member of staff should be able to spend some time with them after the proceedings have been completed to discuss their reactions and to answer any questions.

## **Impact on staff**

Staff are the most valuable resource in the National Health Service; we need to take steps to protect their welfare, physical and emotional. However, there is now evidence to confirm that, whilst healthcare staff gain much job satisfaction, what they achieve may be at some cost to their health and welfare. “Burnout” and “compassion fatigue” are recognised risks. The authors have also described recently how the adverse effect of work may spill over into the personal and family lives of healthcare staff.

- Sources of stress: There are two primary sources of stress for staff. The first relates to specific demands of their work, including dealing with death, dying, pain and suffering, and insufficient time to recover between incidents. The second relates to poor management and organisational practices. These include a lack of appreciation, work overload, underfunding, and poor communication and relationships. Thus, the institution has no right to assume that the responsibility for staff welfare rests solely with individual members of staff.

***“Burnout” is a professional occupational disease manifest in the many specialities of healthcare and will be a disorder as long as human values and worth are disregarded by inept policy makers and managers of human resources (Felton, 1998)***

- Staff reactions: Inexperienced and younger staff may be vulnerable to work-related pressures. However, it must not be assumed that senior and experienced staff have some special immunity to the pressures of their work. Alexander and Atcheson (1998) found that the senior trauma surgeons and nurses were more likely than their less senior and experienced colleagues to admit to the adverse emotional impact of their work.

## **Violence against staff**

A regrettable and increasingly prevalent source of distress and, sometimes, injury to staff is violence by patients and relatives. Obviously, the institution has a “duty of care” to ensure that every reasonable step is taken to protect staff. However, we ourselves can take some steps to protect our safety. First, we should be aware of early warning signs, and, second, we should conduct ourselves in a way that reduces the risk of violence and confrontation.

### *Early warning signs:*

- A history of violence
- Overtly aggressive, abusive attitudes and/or behaviour
- An identifiable serious psychiatric condition\* (e.g., a paranoid illness [i.e., one in which the person feels that he/ she is being persecuted], alcohol/substance misuse, organic cerebral

damage [including epilepsy and damage to the frontal lobes], and severe personality disorder)

**This does not mean that anybody who has a mental illness is likely to be violent.**

**However, there is an increased risk of violent behaviour in certain conditions.**

*Preventative steps:*

- Whenever possible do not deal alone with volatile situations
- Violence is commonly provoked by anxiety and uncertainty. We can reduce this by explaining in advance what we are going to do (particularly if we are behind the patient) by way of, for example, removing clothing, examining, injecting and suturing.
- Bear in mind that what is done in the resuscitation room may be regarded by the layman as intrusive and invasive not only to the person's body but also to their "psychological space". A courteous acknowledgement of this will make this "invasion" more acceptable to those who might otherwise react adversely.
- If there is a risk of violence, it is wise to keep doors and screens open whenever it is reasonable to do so. This may not only help the individual to stop feeling trapped (and, therefore, violently anxious) but it also provides a ready exit.
- A confrontational and aggressive (or "macho") approach by staff is more likely to be inflammatory rather than defusing and reassuring. A calm, courteous and confident (even if we don't feel it!) approach is more likely to help.
- Beware of persistent eye contact; this may be perceived as confrontational.
- Never underestimate the strength of a disturbed individual, whatever the age, gender or physical stature.

If physical restraints are to be used:

- Ensure they are administered safely (for patients and staff)
- They should be removed only slowly and when the risk of violence has significantly lessened
- Ensure the airway is maintained
- They should be introduced in accordance with well practised procedures. (A chaotic rugby scrum of flailing, unidentifiable limbs is certainly unprofessional and, more importantly, potentially hazardous to patients and to staff.)

If sedation is required:

- Injections should be administered only once immobilization has been achieved

- Avoid the prolonged use of heavy doses of anti-psychotics because of the risk of sudden death

*Important signs that staff may need help*

We need to be alert to signs in our colleagues (and also in ourselves) that might indicate that there are problems of coping. These include:

- Excessive and unusual use of alcohol, food, cigarettes and other substances
- Unusual level of carelessness and accident proneness
- Unusual irritability and moodiness
- Reduced work competence and poorer time-keeping
- Underworking (or, sometimes, overworking since some individuals seek to over compensate when they feel they are not coping)
- Pre-occupation with a specific event
- Social withdrawal
- Excessive denial about emotional difficulties or impact of an event.

**WHAT CAN WE DO TO HELP STAFF?**

Staff in the resuscitation room are usually resilient and emotionally robust individuals who are self-selected for this important and demanding form of work. They will develop their own ways of coping, including “black humour” and viewing incidents as “challenges” or “problems” to be solved rather than overwhelming “crises”. Others may neutralize what would otherwise be disturbing to them (Box 19.5).

**Box 19.5: Case example**

During a major exercise to retrieve human remains after a major disaster, one police body handler reported to the senior author:

*Sir, when I go in there [a large badly damaged structure] as far as I am concerned I'm going into a spaceship looking for Martians.*

In addition, good training and selection will reduce the risks of work-related problems. On the other hand, there are other steps that should be considered:

### *Peer support*

Talking openly with colleagues is a well-recognized source of mutual support. However, it is important that time and a suitable facility are available for this to take place. (We should also consider the wisdom of sending members of staff home following a particularly distressing event. They could miss out on the discussion among and support from their colleagues).

### **SUPPORT GROUPS**

There is an implicit assumption that getting together a number of staff on a regular basis will necessarily be helpful. This has been challenged (Alexander, 1993) because commonly insufficient attention is paid to the aims of the group, the methods by which it will be run, and the choice of the group leader.

**NB:** Although support from colleagues may indeed be a powerful antidote to work-related stress sometimes such support is not available because:

- Colleagues do not wish to be seen as intrusive by asking personal questions.
- Colleagues may find it difficult to deal with the distress and personal problems of other staff (particularly if they are more senior and experienced staff).
- Those who may need help may fear that by admitting this they may compromise their career prospects and run the risk of breaches of confidentiality.

### **ORGANISATIONAL AND MANAGERIAL PRACTICES**

HOW THE “SYSTEM” IS RUN APPEARS TO BE A POWERFUL ANTIDOTE TO THE ADVERSE EFFECTS OF STRESSFUL DUTIES. EFFECTIVE INGREDIENTS INCLUDE:

- Good team spirit
- A clear definition of duties and responsibilities
- Good communication within the team
- Explicit appreciation and recognition of “work well done” (rather than the common practice of identifying colleagues only when they have made a mistake)
- Adequate training (Training can help to establish realistic expectations of what staff can do, and also it should include opportunities to rehearse how individuals will cope with things when they do not go to plan, e.g. in a failed CPR.)

### **PSYCHOLOGICAL DEBRIEFING**

In the 1980s and early 1990s, there was a strong move towards setting up debriefing sessions following “critical incidents” (i.e., those events which threaten to overwhelm the ability of staff to cope). The aims of debriefing were to:

- Confirm that the individuals’ reactions are normal

- Enable them to describe their reactions to the incident
- Identify what has been learned and what gains have been made from the incident
- Reinforce the mutual supportiveness among the group
- Help individuals to disengage from (i.e., leave behind) the incident

Strong claims were initially made for debriefing as a means of preventing the onset of post-traumatic reactions such as PTSD. More recently, however, a more conservative approach has been advocated because there has been some evidence that certain individuals, under some circumstances, may feel worse after debriefing. This is a contentious issue, and there are many important questions to be answered. For those who wish to introduce debriefing, they are advised to consult the relevant literature on the principles, practices and evaluation of debriefing to be found in further reading.

If it is decided to introduce debriefing sessions after critical incidents, the following guiding principles should be considered. Debriefers should be:

- Properly trained
- Familiar with normal and pathological reactions to critical incidents
- Familiar with group dynamics (a group is more than the sum of the individuals)
- Familiar with the nature of the incident
- Familiar with the “culture” of the resuscitation room (in terms of what goes on in that environment, who does what, and what are the prevailing values, fears and needs among the staff).

## **SUMMARY**

The profile of contemporary medicine is usually cast in terms of technological advances. Whilst these are certainly impressive, we will not attain the highest levels of medical care unless we address with equal commitment the psychological needs of patients and their families. Moreover, however well trained and carefully selected, staff working in the resuscitation room have a right to have their emotional needs and welfare considered in order that they are able to fulfill their important and demanding duties.

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## **CHAPTER 20**

### **EPIDEMIOLOGY OF TRAUMA**

#### **Objectives**

At the end of this chapter, the reader should understand the:

- Definition of injury
- Magnitude of trauma as a public health problem
- Main causes and types of traumatic injuries
- Distribution of injuries and their causes by geography, age, gender and income
- Most common patterns of injuries and distribution of death
- Some aspects of the prevention and/or mitigation of injuries

#### **Definition of injury**

An injury is defined as "a body lesion at the organic level, resulting from acute exposure to energy (mechanical, thermal, electrical, chemical or radiant) in amounts that exceed the threshold of physiological tolerance. In some cases for example drowning, strangulation and freezing, the injury results from an insufficiency of a vital element" (Baker SP et al. 1984).

#### **Magnitude of trauma as a public health problem**

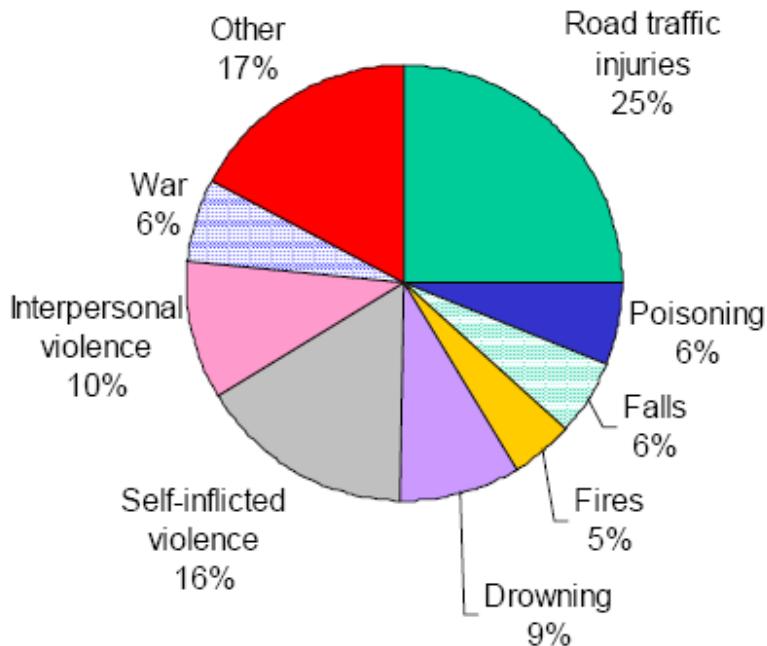
A World Health Organisation (WHO) report in 2003 estimated that 5.18 million people worldwide died from trauma in 2002 and approximately 80,000 in Europe. Injuries account for the 9% of the total number of world's deaths and ranks fourth among all causes of mortality after cardiovascular diseases, infections and malignancies. In 2000, the overall mortality rate for injuries in high-income European countries was 47.6 per 100,000 of the population. More impressively, in the USA the odds of dying from an injury in one year are 1 in 1,755. The younger population are especially affected, with trauma being the commonest cause of death in Europe in people under the age of 45. The actual burden of injuries can only be truly appreciated if disabilities are also taken into account. In the USA in 2003, for every death due to trauma it is estimated that 2-4 cases of temporary or permanent disability occurred. The fact that injuries affect the younger members of society magnifies the negative impact on communities because the young have the longest expectancy of productive life. The concept of Disability Adjusted Life Years (DALYs) was developed to account for this, with a DALY being defined as "one lost year of healthy life, either due to premature death or disability". Injuries cause 15% of DALYs worldwide.

The estimated annual European cost of treating injuries that result in death is €1–6 billion and treating non-fatal injuries €80–290 billion.

### Main causes of traumatic injuries

Road traffic injuries are the leading cause of injury-related deaths worldwide, followed by self-inflicted violence and interpersonal violence. The worldwide injury mortality by cause in the year 2000 is shown in Fig. 20.1.

**Fig. 20.1:**  
**Distribution of global injury mortality by cause, 2000**



The International Classification of Diseases (ICD 9) separates injuries into intentional and unintentional categories. These are then subdivided further (table 19.1).

Table 19.1

External causes of injury and their corresponding ICD codes			
Type of external cause of injury	ICD-9 code	ICD-9 BTL code	ICD-10 code
All injuries	E800–E999	B47–B56	V01–Y98
Unintentional injuries	E800–E949	B47–B53	V01–X59, Y40–Y86, Y88, Y89
1 Road traffic injuries	E810–E819, E826–E829, E929	B471–B472	V01–V89, V99, Y850
2 Poisoning	E850–E869	B48	X40–X49
3 Falls	E880–E888	B50	W00–W19
4 Fires	E890–E899	B51	X00–X09
5 Drowning	E910	B521	W65–W74
6 Other unintentional injuries	E800–E807, E820–E848, E870–E879, E900–E909, E911–E949	B49, B52 (minus B521), B53, B47 (minus B471)	V90–V98, W20–W64, W75–W99, X10–X39, X50–X59, Y40–Y86, Y88, Y89
Intentional injuries	E950–E978, E990–E999	B54–B55, B56 (minus B560)	X60–Y09, Y35–Y36, Y870–Y871
1 Self-inflicted	E950–E959	B54	X60–X84, Y870
2 Interpersonal violence	E960–E969	B55	X85–Y09, Y871
3 War	E990–E999	B561	Y36
4 Other intentional injuries	E970–E978	B569	Y35

ICD, International Classification of Disease; BTL, basic tabulation list.

From: Peden M, McGee K, Sharma G. *The injury chart book: a graphical overview of the global burden of injuries*. Geneva, World Health Organization, 2002.

Based on 2002 estimates for Europe, unintentional injuries are responsible for nearly two thirds of deaths following injury. Among these, road traffic accidents (RTA) are the leading cause, followed by poisoning (especially from alcohol and in Eastern Europe) and falls. Amongst intentional injuries, self-inflicted trauma is the leading cause of death, followed by interpersonal violence. However, there are important differences between high-income countries (HIC) and middle to low income countries (L/MIC) - see below.

Although the above information is authoritative and valuable, the taxonomy of injuries is limited because precise information on the mechanism of injury (MOI) may be mixed with information on intent. Most Trauma Registries therefore categories cases according to their blunt, penetrating or thermal cause. These broad categories are then sub divided according to intent and by more detailed information of the MOIs. This list has usually some degree of overlapping with the WHO classification at least for the main causes i.e. RTA and falls, but the further definitions are less standardized, though a recent plea for uniformity is expected to have some effect. A further

problem with the available data is that it is usually limited to patients who make it to hospital with a defined severity of injury. It therefore does not consider both the victims dead at scene and patients with minor injuries.

EuroTARN is an example of the type of trauma register described above. This was established in 2002 with the aim of developing a shared trauma database among European hospitals. Currently it collects data from 9 countries and focuses on hospitalized patients with an Injury Severity Score (ISS) >15. The latter criterion is the universal definition of major trauma, because it corresponds to a steep increase in the mortality curve. The distribution of the types and causes of injuries according to these data is shown in table 19.2.

**Table 19.2: Distribution of injury types and mechanisms of injury in Europe\***

Injury Type	Totals	Overall %
Blunt Injury	12959	96
Penetrating	543	4
	<u>13502</u>	<u>100</u>

Mechanism of Injury	Totals	Overall %
Road Traffic Incidents	6971	51,6
Major Falls	2441	18,1
Stabbing Assault	192	1,4
Blunt Assault	758	5,6
Gunshot Wounds	175	1,3
All Other	2873	21,3
Not known	98	0,7
<b>Totals</b>	<b><u>13508</u></b>	<b><u>100</u></b>

\* Variable data collection between 2000-2004 based on 11 submissions from 9 countries (Austria, Bosnia-Herzegovina, Croatia, Denmark, Italy, Macedonia, Portugal, Slovak Republic, UK). Burns are coded as blunt injuries.

From: "Comparison of European Registries of Trauma - first report of the EuroTARN Group" – unpublished abstract presented at the Third Mediterranean Emergency Medicine Congress. Nice, France. September 1-5, 2005. Courtesy of TARN and EuroTARN - Salford UK.

Population-based studies have fewer of the limitations mentioned above but are available only from single European countries. Overall, they report similar data, with a strong (>95%) prevalence of non-penetrating injuries. RTAs and falls are also the commonest mechanisms of injury. When considering only trauma fatalities the prevailing MOIs remain the same, though with different percentages.

## Distribution of injuries and their causes by geography, income, age and gender

There are wide differences in the frequency of injuries and the distribution of their causes according to income, geography, age and gender. Oversimplification has to be avoided because these factors are often intertwined, but some generalizations can be made. First, mortality rates for all seriously injured adults has been shown to be inversely related to income, rising from 35% in a high-income (HIC) setting, to 55% in a middle-income setting and reaching 63% in a low-income setting. Even within the European Region, the injury-related mortality rates are more than triple in low and middle income (L/MIC) countries, for example the Czech Republic, Romania and the Russian Federation compared to the HICs of U.K., France, Germany and Italy. In L/MIC interpersonal violence also prevails, whereas in HICs, mortality rates from falls are higher. Interpersonal violence and consequently penetrating injuries are also more frequent in North America compared with Western Europe. The latter does however have a mortality rate from suicide that is second only to China, with very high mortality rates from poisoning amongst European LMICs. Combining these data we now know that self-inflicted injuries, falls and RTAs are the commonest causes of death in HIC European countries and they have roughly similar mortality rates. However, as already mentioned, it is the younger members of the population that suffer the most, being injured and killed more frequently than the elderly; those between the ages of 15 and 44 years account for almost 50% of the world's injury-related mortality. When considering all causes, males have injury mortality twice that of females and with only self-inflicted being a greater problem amongst the female population.

## Common types and patterns of injuries and distribution of death

A large and variable array of factors determines the final characteristics of injuries after a traumatic event. Therefore, typical patterns can be identified only in relation to very specific MOIs. For example, within the category of RTAs, a predictable association between the type of vehicle and the pattern of injuries has been demonstrated. However for the same type of vehicle there are confounding variables that will affect the type of injury sustained. These include the direction of collisions, the position of the victim in the car, the use and type of seat belts and deployment of airbags. Another example is that suicidal jumps and accidental falls present substantial differences in injury patterns.

## Sites of injury

If all levels of severity are considered, musculo-skeletal injuries are the most common, followed by head and chest. However the head is the most frequently injured part of the body when considering only the most severe injuries (ISS>15 or fatalities). The next three areas are the chest, limbs and abdomen respectively. These injuries are more often combined than isolated.

Head injuries are also the leading cause of both injury related death and disability among children and young adults and represent the most costly workers' compensation claims in the USA.

#### **Time-related distribution of deaths**

The timing of death after trauma was first described in the USA nearly 25 years ago and described a trimodal distribution; the first peak of deaths (50%) occurred immediately or within minutes, a second peak (30%) occurred within the next 4 hours, usually after arrival at hospital, and the third peak (20%) were those patients who died hours or days later, often from injury related complications. This concept has often been used to justify the efforts toward the organization and improvement of acute trauma-care to try and prevent the deaths that occur after reaching hospital, a period often referred to as the "golden hour". However recent studies, both in the USA and Europe have found that a greater proportion of victims die immediately (approximatey 70%) and subsequent deaths are more uniform with no conspicuous peaks. As these studies come from HIC countries with a presumed better level of health care, it is possible that progressive improvements in trauma care may have contributed to this change through a decrease in unnecessary deaths. For trauma deaths that occur immediately, preventive efforts, particularly legislation, represent the most cost-effective and rational approach to trauma care. However, the relative importance of effective medical care at the scene and in hospital must not be minimized.

#### **Some aspects of the prevention and/or mitigation of injuries**

As already discussed, injuries are such a significant public health problem that they are often described as an "epidemic". Indeed, the burden of injuries is growing quickly when considered on a worldwide scale, posing an extraordinary challenge to health policy makers. Table 19.3 shows the expected increase in the injury burden if the current trend continues.

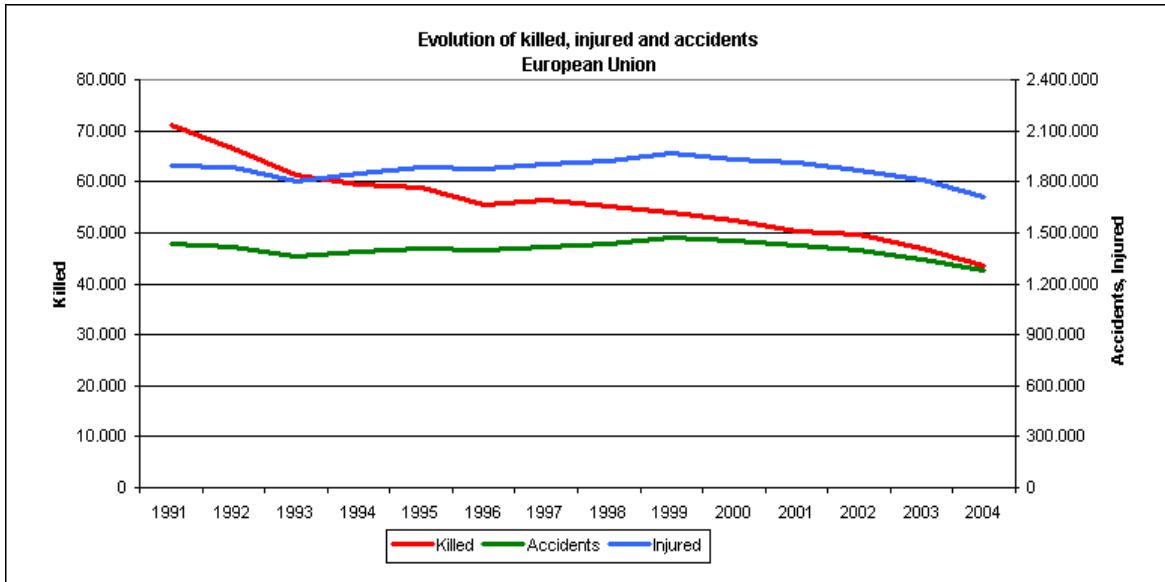
**Table 19.3**

World rankings of injury-related mortality and burden of disease (DALYs lost), 1990 and 2020

	No. of deaths		DALYs lost	
	1990	2020	1990	2020
Road traffic injuries	9	6	9	3
Self-inflicted injuries	12	10	17	14
Interpersonal violence	16	14	19	12
War	20	15	16	8

At the same time, many cost-effective strategies exist to prevent injuries and minimise their consequences. In the past, the implementation of these strategies in HIC has often allowed a reversal of the increasing trend of trauma deaths and disabilities. For example in the European Union, despite increasing traffic volumes, fatalities have been constantly decreasing in the last 15 years (Fig. 20.2). Road safety campaigns, technological progress in motor-vehicles safety and trauma health-care improvements are among the likely factors that have enabled this achievement. Similar decreasing trends have been demonstrated also for injuries in general in the USA. These achievements call for immediate action in L/MIC, where the trauma “epidemic” is increasing. However, in every country regardless of income, every effort should be made to reduce the dismal figures of trauma morbidity and mortality. This will involve tackling the three stages of injury prevention.

**Fig. 20.2: Decline in fatalities in the European Union**



From: SAFETYNET - Annual Statistical Report 2004 based on data from the CARE database. Available at: <http://europa.eu.int/comm/transport/care/studies/doc/safetynet/SN-1-3-ALL-ASR-122004.pdf>

### The three stages of injury prevention

William Haddon described a three-stage process of injury prevention. The underlying idea is that injuries can be reduced by preventing the event from occurring in the first place, so called primary prevention, for example speed limits and limiting alcohol consumption. Secondary prevention aims to prevent or minimise injury even if the injury-producing event occurs, for example safety devices in cars and wearing crash helmets. Finally tertiary prevention aims at minimising disability amongst those who survive by improvements in health care and rehabilitation. The various preventive interventions can be targeted at the host, the agent and the environment level. In Fig. 20.3 the Haddon matrix is applied, for example, to the problem of school violence by firearms.

Fig 20.3:

*Table 2 Haddon matrix applied to the problem of school violence by firearms*

	<i>Host (students at school)</i>	<i>Agent/vehicle (firearms and bullets)</i>	<i>Physical environment (school)</i>	<i>Social environment (school and community norms, policies, rules)</i>
Pre-event (before teen uses weapon)	Educate teens about the dangers of carrying guns to school Educate parents about dangers of allowing teens access to guns Teach students to recognize and report student behaviors indicative of possible violent behavior	Modify guns so they are only openable by the owner	Install metal detectors at entrances to schools Eliminate storage places in schools (for example lockers) where guns might be kept	Adopt school procedures/policies to notify authorities if a student is suspected of having a gun at school Prohibit gun carrying on school grounds Enforce restrictions on the sale or transfer of handguns to teenagers
Event (when gun is taken out to be fired)	Teach students to take cover when they see guns or hear gunfire	Reduce capacity of weapons to fire multiple rounds quickly Modify bullets to be less lethal	Install alarm systems to call law enforcement as soon as weapons are visible	Have law enforcement officers on duty at school to intervene during fights Develop safety plans to help students move to safety in event of violent episode
Post-event (after students are shot)	Teach students first aid skill	Reduce the capacity of the gun to continue firing	Make school grounds readily accessible to ambulances	Ensure well trained emergency medical personnel and access to trauma facilities Provide post-event counseling to students, staff, and families

From: Runyan CW. Using the Haddon matrix: introducing the third dimension. Inj. Prev. 1998;4:302–307

As it can be inferred from the distribution of death and other studies the most effective interventions to decrease the burden of injuries are those of primary and secondary prevention. Nevertheless the improvement of medical care can play an important role. For example high quality health care alone is definitely associated with better outcome after an injury and in several countries has led to notable reductions of mortality from injury. In L/MICs, simple and inexpensive interventions, for example ensuring all accident victims receive appropriate care at scene, can have a great impact. This is the principle underlying the WHO Essential Trauma Care Project that aims at establishing achievable and affordable standards for injury care worldwide.

However, also in HIC where the quality of health care has already lead to a proven reduction of case fatality after injury, there is room for improvement as indirectly demonstrated by several studies that have found an incidence of potentially or definitely preventable deaths among trauma fatalities ranging from 9% to 43%. The same key principles that have so far been demonstrated effective should be pursued in order to attain a further improvement: organization, education, and surveillance

## Summary

Trauma is the fourth commonest cause of death worldwide and sadly the lives it claims are those of the youngest members of society. It is the commonest cause of death between the

ages of 1 and 44 years and for every fatality, 2-4 victims are temporarily or permanently disabled. The greatest contribution to these statistics comes from road traffic accidents. While fatalities have been decreasing despite increases in traffic volumes in more affluent countries, the reverse is true in low-income countries to the extent that trauma is now regarded as an epidemic. Although prevention of trauma is more effective than trying to cure those injured, the delivery of quality health care is an essential step to reduce the thousands of deaths that occur each year.



# **CHAPTER 21**

## **SCORING SYSTEMS FOR TRAUMA PATIENTS**

### **Objectives**

At the end of this chapter the reader should understand:

- The basic principles of anatomical and physiological scoring systems

Trauma care systems deal with patients who have an almost infinite variety of injuries requiring complex treatment. The assessment of such systems is a major challenge in clinical measurement and audit. A common question is “which systems are most effective in delivering best outcomes?” Furthermore, implementing developments in treatment often incurs additional costs – will the expense be worthwhile? Clearly, case mix adjusted outcome analysis must replace anecdote and dogma when making these assessments. Outcome prediction in trauma is a developing science that enables the assessment of trauma system effectiveness.

The effects of injury can be defined in terms of input – an anatomical component and the physiological response – and outcome – mortality and morbidity. These must be coded numerically before we can comment with confidence on treatment or process of care. Elderly people survive trauma less well than others, so age must be taken into account. Most recent work has been concerned with measurement of injury severity and its relation to mortality. Assessment of morbidity has been largely neglected, yet for every person who dies as a result of trauma there are two seriously disabled survivors. However to date outcome prediction has focussed on determining the injured patients average probability of survival (Ps).

### **Input measures**

Injury is assessed through the anatomical component and the physiological response. These two elements are separately scored.

#### **Anatomical scoring system**

The abbreviated injury scale (AIS), first published in 1969, is anatomically based. There is a single AIS score for each injury a patient may sustain. Scores range from 1 (minor) to 6 (incompatible with life). There are more than 1200 injuries listed in the AIS dictionary, which is currently in its fourth edition (AIS90). Intervals between the scores are not always consistent –

for example, the difference between AIS3 and AIS4 is not necessarily that same as that between AIS1 and AIS2 (Box 21.1).

<b>Box 21.1: Examples of injuries scored by the <i>Abbreviated injury scale (AIS90)</i></b>	
<b>Injury</b>	<b>Score</b>
Shoulder pain (no injury specified)	0
Wrist sprain	1 (Minor)
Closed, undisplaced tibial fracture	2 (Moderate)
Head injury – unconscious on admission but for less than 1 hr thereafter, no neurological deficit	3 (Serious)
Incomplete transaction of the thoracic aorta	4 (Severe)
Complex liver laceration	5 (Critical)
Laceration of the brain stem	6 (Incompatible with life)

To overcome the problem of patients with multiple injuries, all the injuries are scored using the AIS. The body is divided into six pre-determined areas, different from the AIS:

- Head or neck, including brain, skull, cervical spine
- Face including mouth, ears, nose and facial bones
- Chest, including all internal organs, diaphragm and thoracic spine
- Abdomen or pelvic contents, including lumbar spine
- Body surface, including burns, lacerations and contusions

The ISS is calculated as the sum of the squares of the **three highest AIS scores** in each of the most severely injured ISS body regions (Box 21.2). Scores of 7 and 15 are unattainable because these figures cannot be obtained from summing squares. The maximum score is 75 (25+25+25). By convention, a patient with an AIS6 in any one region of the body is given an ISS of 75. The injury severity score is non-linear and there is pronounced variation in the frequency of different scores; 9 and 16 are common, 14 and 22 unusual.

### **Case study**

A man is injured in a fall at work. He complains of pain in his neck, jaw, and left wrist and has difficulty breathing. There are abrasions around the left shoulder, left side of the chest, and left knee. Examination of the cervical spine radiologically suggests no abnormality. There is a

displaced fracture of the body of the mandible. There are also fractures of the left wrist, and left ribs (5-9), with a flail segment. The AIS scores for these injuries are shown in Box 21.2.

<b><u>Box 21.2: AIS scores for case study</u></b>	
Injury	AIS score
Fracture of body of mandible	<b>2</b>
Fracture of lower end of radius (not further specified*)	<b>2</b>
Fracture of ribs 5-9 with flail segment	<b>4</b>
Abrasions (all sites)	1
Neck pain†	0

\*If the fracture of the radius was known to be displaced or open the AIS would be 3. If not specified the lower score is used

†Symptoms are not scored if there is no demonstrable anatomical injury

$$\text{Calculated ISS: } 2^2 + 2^2 + 4^2 = 24$$

For the purpose of the analysis described here, the ISS should be calculated only from operative findings, appropriate investigations, or necropsy reports. The ISS has been shown to correlate with outcome and a score of 16 is predictive of a mortality rate of approximately 10%. This value is also used as the definition of major trauma based upon anatomical site of injury. The median value and the range, not the mean value, should identify the overall injury severity score of a group of patients. Non-parametric statistics should be used for analysis.

#### Physiological scoring systems

The revised trauma score (RTS) is used to assess the physiological response of a patient to trauma. After injury, the patient's physiological response is constantly changing but for the purposes of injury scoring, and by convention, the RTS is measured when the patient arrives at hospital. If the patient is intubated before arrival an RTS cannot be measured. The physiological parameters that make up the RTS are respiratory rate, systolic blood pressure and Glasgow Coma Scale score.

### *The Glasgow Coma Scale (GCS)*

This is the accepted international standard for measuring neurological state. The state may be represented as a single figure (for example, GCS=15) or as the responses in each of the three sections of the scale (for example, eye opening = 4, verbal response = 5, and best motor response = 6). Coma is defined as a Glasgow coma score of <9 (Chapter 7). Various modifications of the scale have been suggested for use in small children. Some doctors reduce the maximum score to that which is consistent with neurological maturation. A more useful clinical device, which ensures more accurate communication and simplifies epidemiological research, is to retain the maximum score of 15 but redefine the descriptions.

The RTS combines coded measurements of respiratory rate, systolic blood pressure, and Glasgow Coma Scale score to provide a general assessment of physiological derangement. This scoring system was developed following statistical analysis of a large North American database to determine the most predictive independent outcome variables. The combination of these three variables is independently related to outcome. Selection of variables was also influenced by their ease of measurement and clinical opinion. In practice the RTS is a complex calculation combining coded measurements of the three physiological values from 0 – 4 (Box 21.3). To calculate the RTS, the coded value for each variable is multiplied by a weighting factor derived from regression analysis of the database. This correction reflects the relative value of the variable in determining survival (Appendix 21.1).

<b><u>Box 21.3: Coded values for the Revised Trauma Score</u></b>				
Glasgow Coma Scale score	Systolic blood pressure (mm Hg)	Respiratory rate (Breaths/min)	Coded value	
13-15	> 89	10-29	4	
9-12	76-89	>29	3	
6-8	50-75	6-9	2	
4-5	1-49	1-5	1	
3	0	0	0	

Clearly it would be difficult to calculate the RTS in the field and it has been suggested that assessment of the trauma patient that reveals any coded value of <4, should result in the patient being taken to a centre capable of dealing with major trauma.

## **TRISS methodology**

The degree of physiological derangement and the extent of anatomical injury are measures of the threat to life. Mortality will also be affected by the age and gender of the patient. "TRISS methodology" combines four elements – the revised trauma score (RTS), the injury severity score (ISS), the patient's age, and whether the injury is blunt or penetrating – to provide a measure of the probability of survival (Ps; Appendix 20.2). (The acronym is tortuously developed from Trauma score and Injury severity Score). The Trauma Audit and Research Network (TARN) database ( $n>100,000$ ) has been used to update the methodology to reflect the characteristics of the European trauma population when calculating the survival probability. Gender becomes an important predictor in patients aged  $>55$  years (possibly a reflection of higher comorbidity in males) and as only 2% of European patients are injured by penetrating trauma the blunt/penetrating distinction is no longer required. In the latest European version of TRISS the GCS has been found to be the most valuable physiological predictor.

It is important to realise that Ps is merely a mathematical calculation; it is not an absolute measure of mortality but only an indication of the probability of survival. If a patient with a Ps of 80% dies the outcome is unexpected because four out of five patients with such a Ps would be expected to survive. However, the fifth would be expected to die – and this could be the patient under study. The Ps is used as a filter for highlighting patients for study in multi-disciplinary trauma audit.

## **Comparing systems of care**

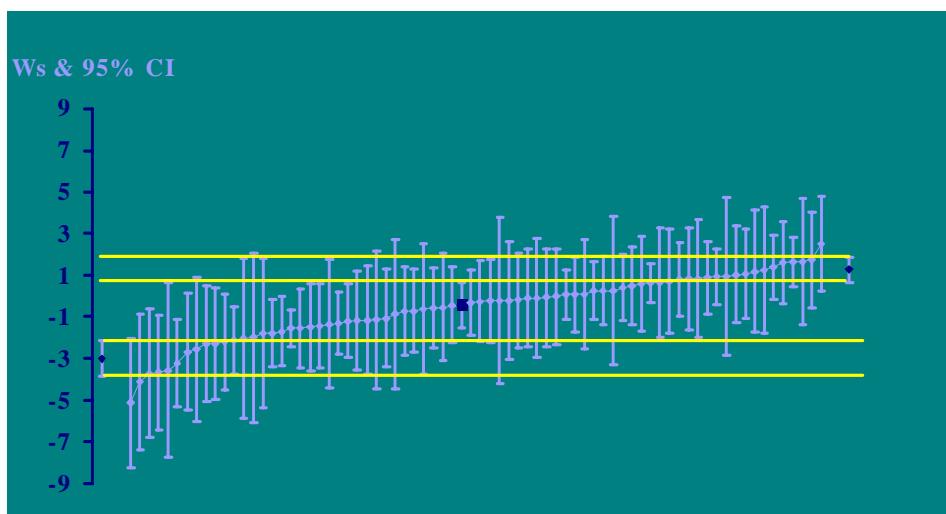
Comparison of the probabilities of survival of all patients seen at a particular hospital with the observed outcome can be used as an index of overall performance. Probabilities of survival are combined in the "standardised W statistic" (Ws) to assess a group of patients.

### *Standardised W statistic*

The Ws provides a measure of the number of additional survivors, or deaths, for every 100 patients treated at each hospital accounting for different mixes of injury severity. The "standardised Z statistic" (Zs) provides a measure of its statistical significance. A high positive Ws is desirable as this indicates that more patients are surviving than would be predicted from the TRISS methodology. Conversely a negative Ws signifies that the system of trauma care has fewer survivors than expected from the TRISS predictions. Comparisons have become more relevant to clinicians after extensive work was undertaken to base the regression analysis on statistics derived from the Trauma Network database. The Ws can be shown graphically with their 95% confidence intervals to illustrate clinical differences between hospitals (see below).

# Hospital comparisons

## Summary Ws Scores 1989-2004



### Trauma Audit and Research Network

First developed in North America, the method used in the Trauma Audit and Research Network (TARN, developed from the Major Trauma Outcome Study) is now used in England and Wales and throughout Europe and Australia to audit the effectiveness of systems of trauma care and the management of individual patients. The Trauma Network provides a valuable method of comparing patterns of care in different parts of the country. It is reliant on careful collection of data in a consistent format to allow collation and comparison of results. Deaths caused by trauma are too varied, too complicated, and too important to be discussed in isolation in individual hospitals. The wider perspective of the Trauma Network is increasingly recognised as the only valid approach to trauma audit and is being taken up by regional and national bodies for this purpose. Identification of deficiencies is valuable only if a mechanism exists to correct them. Local audit meetings and national comparisons must be used to stimulate appropriate changes in systems of trauma care.

The development of the TRISS Ps04 methodology has been a major advance in the measurement of injury severity. The detailed structure of the scales and the method of developing a single number to represent threat to life are, however, under constant review. An alternative method of measuring anatomical injury has been described. The method uses the

root sum squares of the AIS scores for the head and trunk (Anatomic profile). It has been incorporated into a system for the characterisation of trauma (ASCOT), using different weightings for the AIS scores of a patient's most severe injuries regardless of body region. These developments may lead to more accurate scoring systems, but for the present the TRISS methodology has a worldwide reputation for consistency and reasonable prediction of outcome.

European trauma registries are now collaborating within the EuroTARN initiative to compare crude outcomes (% mortality) in similar groups of patients (EuroTARN: <http://eurotarn.man.ac.uk>). As there is greater trauma systems variation across Europe than certain routines it is hoped this collaboration will help identify the true role and benefits of "trauma centres" and other system characteristics that have yet to be determined.

## Summary

Measurement of outcome in terms of survival or death is, however, a crude yardstick. Further progress is required in measuring disability after injury. Most life threatening visceral injuries leave the patient with little disability. In contrast the many more patients who sustain musculoskeletal sequelae are largely ignored in the statistics. Much effort will be required to develop outcomes measures based on disability; these are essential if the treatment of the multiply injured patient is to be based on sound scientific principles.

## **Appendix 21.1: Calculation of the Revised trauma score (RTS)**

	Coded value	$\times$	Weighting factor	=	Score
<i>Respiratory rate (breaths / min):</i>					
10-29	4				
>29	3				
6-9	2		0.2908		
1-5	1				
0	0				
<i>Systolic blood pressure (mm Hg):</i>					
>89	4				
76-89	3				
50-75	2		0.7326		
1-49	1				
0	0				
<i>Glasgow coma scale:</i>					
13-15	4				
9-12	3				
6-8	2		0.9368		
4-5	1				
3	0				

Total = revised trauma score:

## **Appendix 21.2: TRISS methodology**

Probability of survival of individual patient (Ps) =  $1/(1+e^b)$

e = 2.718282, the base of Napierian logarithms

b = b<sub>0</sub> + b<sub>1</sub> (RTS) + b<sub>2</sub> (ISS) + b<sub>3</sub> (Age)

b<sub>0</sub>, b<sub>1</sub>, b<sub>2</sub>, b<sub>3</sub> are coefficients derived from regression analysis applied to data from the Trauma Network. Technically they are different for blunt and penetrating injuries, but this appears less important in European data.

RTS = revised trauma score  
ISS = injury severity score  
Age = 0 if <55 years  
-1.1655 if age = 55-64 years  
-1.8339 if age = 65-74 years  
-2.8182 if age = 75-84 years  
-3.4448 if age >84 years