Severe and Multiple trauma

Clinical problems

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LEARNING OBJECTIVES
After studying this module on Severe and Multiple trauma, you should have the information to assist you in:

1. Performing the initial evaluation of a trauma patient utilising primary, secondary and tertiary assessment
2. Determining and effecting appropriate immediate management of life-threatening injuries
3. Outlining and addressing the acute issues which are part of ongoing care once in ICU
4. Evaluating trauma care outcomes including the recognition and facilitation of the role of rehabilitation.

FACULTY DISCLOSURES
The authors of this module have not reported any disclosures.

DURATION
7 hours

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# Contents

**Introduction** ........................................................................................................................................................................... 5

1/ Characteristics of trauma .......................................................................................................................................................... 6

   Mechanisms of injury .................................................................................................................................................................... 6
   Blunt injuries .................................................................................................................................................................................. 6
   Penetrating injuries ....................................................................................................................................................................... 6
   Blast injury .................................................................................................................................................................................. 6
   Thermal injuries ........................................................................................................................................................................... 6

   Assessing injury severity ............................................................................................................................................................... 6

   Scoring systems ............................................................................................................................................................................... 9

2/ Acute trauma care including primary assessment/ interventions ............................................................................................. 11

   Organisational aspects ..................................................................................................................................................................... 11
   Pre-hospital setting ......................................................................................................................................................................... 11
   Emergency department ...................................................................................................................................................................... 11
   Self- and patient-protection ......................................................................................................................................................... 12

   Primary assessment ....................................................................................................................................................................... 12
   A – Airway (with C-spine control) ............................................................................................................................................... 14
   B – Breathing ................................................................................................................................................................................ 14
   C – Circulation ............................................................................................................................................................................... 15
   D – Disability and CNS emergencies .......................................................................................................................................... 16

   Primary interventions ...................................................................................................................................................................... 18
   A, B – Airway/Breathing: ............................................................................................................................................................ 18
   C - Circulation: ................................................................................................................................................................................ 20
   D – Disability and CNS emergencies .......................................................................................................................................... 23

3/ Secondary assessment and interventions .................................................................................................................................... 23

   Assessment .................................................................................................................................................................................... 23
   Imaging techniques ......................................................................................................................................................................... 25

   Interventions ................................................................................................................................................................................ 27
   Monitoring the patient .................................................................................................................................................................... 27
   Haemorrhage control .................................................................................................................................................................... 28

   Trauma-induced coagulopathy and transfusion management .................................................................................................. 30

   Damage control resuscitation and surgery .................................................................................................................................. 38

   Neurosurgical interventions .......................................................................................................................................................... 40

   Pain relief ...................................................................................................................................................................................... 40
<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>4/ Post-acute trauma care including organ-specific management</td>
<td>41</td>
</tr>
<tr>
<td>Shock resuscitation</td>
<td>41</td>
</tr>
<tr>
<td>Transfusion and coagulation management</td>
<td>42</td>
</tr>
<tr>
<td>The systemic inflammatory response syndrome and host response</td>
<td>43</td>
</tr>
<tr>
<td>Management of specific injury patterns in the ICU</td>
<td>44</td>
</tr>
<tr>
<td>Head and neck</td>
<td>44</td>
</tr>
<tr>
<td>Chest</td>
<td>44</td>
</tr>
<tr>
<td>Abdomen</td>
<td>48</td>
</tr>
<tr>
<td>Pelvis</td>
<td>49</td>
</tr>
<tr>
<td>Extremities</td>
<td>50</td>
</tr>
<tr>
<td>Spine and spinal cord</td>
<td>52</td>
</tr>
<tr>
<td>Special consideration: pregnancy</td>
<td>53</td>
</tr>
<tr>
<td>5/ General ICU care of the trauma victim and Rehabilitation</td>
<td>54</td>
</tr>
<tr>
<td>Nutrition</td>
<td>54</td>
</tr>
<tr>
<td>Thrombo-embolism prophylaxis</td>
<td>55</td>
</tr>
<tr>
<td>Antibiotic prophylaxis</td>
<td>55</td>
</tr>
<tr>
<td>Pain relief, sedation and delirium management</td>
<td>55</td>
</tr>
<tr>
<td>Tertiary surgical interventions</td>
<td>56</td>
</tr>
<tr>
<td>Rehabilitation</td>
<td>56</td>
</tr>
<tr>
<td>Physical and psychological rehabilitation</td>
<td>57</td>
</tr>
<tr>
<td>Conclusion</td>
<td>58</td>
</tr>
<tr>
<td>Patient challenges</td>
<td>59</td>
</tr>
</tbody>
</table>
INTRODUCTION

Trauma, which has been termed the neglected disease of modern society, is among the leading causes of death in all age groups. Each year it is estimated that around 5.8 million people worldwide die as a result of trauma, with 90% of these deaths occurring in middle- and low-income countries. This represents around 10% of the world’s deaths, more than the deaths from malaria, tuberculosis and HIV/AIDS combined. Below the age of 45 years, trauma is the leading cause of death worldwide. Classically, it affects productive members of society, often leading to long-term disability and dependency in longer term survivors.

Changing epidemiology
While some decades ago the typical trauma patient admitted to the intensive care units of industrialised countries was young (15-45 years) and a victim of high velocity trauma e.g. a road traffic accident, today’s critically ill trauma victim is older with low-impact injury mechanisms (e.g. ground level falls) making up a larger proportion of injuries. An important risk factor for trauma death in elderly patients is concurrent use of anti-thrombotic drugs, which makes them particularly vulnerable to bleeding e.g. intracranial haematomas.
Mechanisms of injury

The mechanisms of injury can be divided into four main types. Combinations are common.

**Blunt injuries**

This is the most frequent mechanism of injury, commonly resulting from road traffic collisions, falls and sport-related incidents. In settings where blunt trauma predominates, severe head injuries are the most common cause of death and long-term disability. The other leading cause of death is exsanguination, mostly from injuries to the great vessels.

**Penetrating injuries**

Penetrating injuries are often associated with knife and firearm use, but may also be seen in farming, traffic or other accidents. Together with blast and burn trauma, these are the usual injuries occurring in combat situations. In Europe, penetrating trauma cases admitted to hospitals account for less than 5% of severe trauma, although rates are higher in metropolitan areas. In some US and South African cities the equivalent figure is as high as 50%. In penetrating trauma to large cavities, the penetrating object is left in place until it can be removed under surgical control, since uncontrolled bleeding may result from premature extraction.

**Blast injury**

Together with penetrating trauma, blast injuries are most frequently seen in war zones and during civil (terrorist attack) bombing events. At close range, explosions may cause burns. Further away, injuries may originate from flying objects or from the victim being accelerated into solid objects. The blast wave typically produces damage at the interface of media of differing density, e.g. air and tissue, such as lung (pneumothoraces), bowel (perforation) and tympanic membrane (perforations). The blast wave may even cause limb fractures and direct lung trauma.

**Thermal injuries**

For information on burns including electrical injuries see the PACT module on Burns Injury.

**Assessing injury severity**

Assessment of injury severity is important clinically to the correct triage of patients to a trauma centre, to selecting the adequate intensity of care and to prognosticating on short-/long-term patient outcome; it is also important to the comparison of trauma centres. Using indicators to adequately assess injury severity is particularly useful in some patients who on superficial assessment seem to be stable and have only minor injury. Trauma severity in such patients may be underestimated and lead to inadequate care provision e.g. transfer to a non-trauma centre or
admission to a ward other than an ICU. This is particularly relevant for vulnerable populations (e.g. the chronically ill, elderly) or those with good compensatory mechanisms (e.g. young adults).

Injury severity can be assessed with the use of selected indicators as well as specific scoring systems (see scoring systems below). Clinical, anatomical, mechanical and special patient or system considerations are summarised in the 2011 Guidelines for Field Triage of Injured Patients, an initiative launched by the American College of Surgeons Committee on Trauma and the Centres for Disease Control and Prevention. According to these guidelines, transfer to a trauma centre is indicated by the presence of any one of the following criteria:

**Clinical indicators:**
- Glasgow Coma Scale ≤13
- Systolic arterial blood pressure <90 mmHg
- Respiratory rate <10 or >29 bpm or the need for ventilatory support

**Anatomical indicators:**
- Penetrating injuries to head, neck, torso and extremities proximal to elbow or knee
- Chest wall instability or deformity
- Two or more proximal long bone fractures
- Crushed, degloved, mangled or pulseless extremity
- Amputation proximal to wrist or ankle
- Pelvic fractures
- Open or depressed skull fracture
- Paralysis

**Mechanical indicators:**
- Falls >6 m (>20 ft) in adults and >3 m (>10 ft) in children
- High-risk automobile crash
  - Car deformation with intrusion of the car body (including the roof) of >30 cm (>12 inch) at the occupant site or >45 cm (>18 inch) at any site
  - Ejection (partial or complete) from automobile
  - Death in same passenger compartment
  - Vehicle telemetry data consistent with a high risk of injury
- Auto vs. pedestrian/bicyclist thrown, run over or with significant (>30 km/h; >20 mph) impact
- Motorcycle crash >30 km/h (>20 mph)

**Special patient or system considerations:**
- Older adults
  - Risk of injury/death increases >55 years
  - Systolic blood pressure <110 mmHg may represent shock in patients aged >65 years
  - Low-impact mechanisms (e.g. ground level falls) may result in severe injury
- Children should be triaged to trauma centres with high level paediatric facilities. This may not be possible in all health systems due to distance to paediatric centres
- Anticoagulants and bleeding disorders
  - Patients with head injury are at high risk of rapid deterioration
- Burns
- Without other trauma mechanism: triage to burn facility
- With trauma mechanism: triage to trauma centre
- Pregnancy >20 weeks
- Emergency medical service provider judgment


In some situations, it may be difficult to distinguish the effects of injury from pre-existing disease. For example, atrial fibrillation is a common cardiac pathology in the general population, but may also result from a direct chest injury or the effects of cardiovascular stress in a patient with pre-existing heart disease.

A 50-year-old driver (restrained with a seat belt in a head-on collision at 30 km/h) presents with mild head injury [Glasgow Coma Scale (GCS) score of 13: eye opening 3, verbal response 4, motor response 6] and fractures of his zygomatic arch, causing a large haematoma around his left eye. On examining him you note a fracture-dislocation of his left forearm, a glyceryl trinitrate (GTN) patch on his chest, and atrial fibrillation with a heart rate of 60/min. Interpret the cardiovascular findings and make a differential diagnosis in accordance with the individual questions below:

**Q. Is the heart rate of significance?**

A. The slow heart rate of 60 despite the obviously painful injuries is unexpected and concerning - see below.

**Q. Outline the significance of the GTN patch and its possible relationship to the bradycardia.**

A. It suggests the patient suffers from coronary artery disease. Therefore the heart rate may be due to chronic medication, e.g. beta blocker, calcium channel blocker (or even digoxin). Or it may be a sign of myocardial infarction or contusion.

**Q. Might the haematoma around the eye be contributory to the bradycardia?**

A. Yes. It may be causing pressure on the eyeball or traction on the eye muscles (oculo-cardiac reflex). The head injury also raises the possibility of elevated intracranial pressure or high spinal injury (although both unlikely given the clinical history).

The mechanism of injury may also provide clues to the type of injury sustained (Table 1).
Table 1.

<table>
<thead>
<tr>
<th>Trauma Mechanism</th>
<th>Common Injuries</th>
</tr>
</thead>
<tbody>
<tr>
<td>Windshield Damage (spider’s web pattern)</td>
<td>- Head injury&lt;br&gt;- Soft tissue injuries of scalp, face or neck&lt;br&gt;- Cervical spine injuries</td>
</tr>
<tr>
<td>Steering Wheel Damage</td>
<td>- Soft tissue injuries to the neck&lt;br&gt;- Sternum fracture&lt;br&gt;- Myocardial injury&lt;br&gt;- Pneumo/haemothorax&lt;br&gt;- Flail chest&lt;br&gt;- Abdominal trauma</td>
</tr>
<tr>
<td>Dashboard Damage</td>
<td>- Pelvic and lower extremity trauma&lt;br&gt;- Head injury&lt;br&gt;- Cervical spine injuries</td>
</tr>
<tr>
<td>Lateral Impact Car Collision</td>
<td>- Head injury&lt;br&gt;- Cervical spine injury&lt;br&gt;- Torso injuries&lt;br&gt;- Pelvic injury&lt;br&gt;- Proximal (lower and upper) extremity injury</td>
</tr>
<tr>
<td>Rear Impact Car Collision</td>
<td>- Cervical spine injuries</td>
</tr>
<tr>
<td>Motorcycle Accident</td>
<td>- Helmets protect from head but not cervical spine trauma&lt;br&gt;- Other injuries similar to victims ejected from vehicles with likely injury to head, neck and extremities</td>
</tr>
<tr>
<td>Auto vs. Pedestrian Collision</td>
<td><strong>Adults:</strong>&lt;br&gt;- Lower leg injuries (bumper/fender impact)&lt;br&gt;- Upper leg injuries (bonnet/hood impact)&lt;br&gt;- Head injuries (fall from/over the car)&lt;br&gt;&lt;br&gt;<strong>Children:</strong>&lt;br&gt;- Upper leg or pelvic injuries (bumper/fender impact)&lt;br&gt;- Torso injuries (bonnet/hood impact)&lt;br&gt;- Head injuries (fall from/over the car)</td>
</tr>
<tr>
<td>Falls</td>
<td><strong>Adults:</strong>&lt;br&gt;- Feet or leg fractures&lt;br&gt;- Hip or pelvic fractures&lt;br&gt;- Spine fractures&lt;br&gt;- Wrist fractures&lt;br&gt;&lt;br&gt;<strong>Children:</strong>&lt;br&gt;- Head injuries (fall from/over the car)</td>
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</table>

**Scoring systems**

Following clinical, radiological and laboratory evaluation, scoring systems are typically applied in the early clinical setting to guide clinical management and prognosticate the risk of death. Some scoring systems also estimate morbidity and hospital length of stay. Common injury scoring systems are the Injury Severity Score (ISS), Revised Trauma Score and Trauma Score. The ISS is the most commonly used; it is based on the Abbreviated Trauma Scale and defines major trauma as an ISS score >15.


http://www.trauma.org/archive/scores/ais.html
http://www.trauma.org/archive/scores/iss.html
http://www.trauma.org/archive/scores/rtls.html
2/ ACUTE TRAUMA CARE INCLUDING PRIMARY ASSESSMENT/INTERVENTIONS

During the acute phase, trauma care is particularly challenging and best follows a structured approach. This has been established by several initiatives, of which the Advanced Trauma Life Support (ATLS) programme is the most widely used. The structured approach is based on a primary assessment to recognise immediate life-threatening problems (requiring immediate intervention) and a subsequent secondary assessment. The timing of radiological interventions may be determined by the nature of injury.

Organisational aspects

Pre-hospital setting

Pre-hospital trauma care may be provided by physician and/or paramedic-staffed emergency medical services depending on the country or region. Several modes of transportation may be employed, including ground (ambulance) or air (helicopter) transport. Although no survival benefit has been shown for physician staffing of emergency medical services, the theoretical advantage of these systems is that adequate resuscitation starts earlier and the time to surgical control of haemorrhage or admission to high level (usually termed level three) care may be shortened by physicians performing primary and secondary assessments in the pre-hospital setting. Physician-staffed emergency medical services have often been criticised for the ‘stay and play’ (rather than ‘scoop and run’) approach when caring for trauma patients. Nowadays, most physician-staffed systems have reduced their interventions at the scene to a vital minimum, particularly in patients with ongoing haemorrhage.

Make sure you have excluded or treated a pneumothorax before transportation by air ambulance, as the air in the pleural space will expand at increasing altitude. Intracranial (or any other collections of air) will also expand with altitude and must be considered when choosing the mode of transport.

When transporting a trauma patient from the accident site, it is crucial to choose the most appropriate destination healthcare facility.

NOTE Primary transfer to a trauma centre is associated with a mortality reduction in patients with severe injury, probably due to factors such as the presence of all the facilities and expertise required to care for the trauma patient and a volume of work which allows maintenance of clinical skills.

Emergency department

Major trauma management requires a team approach - one person alone cannot manage it optimally. The core team treating a severely injured patient in the emergency department requires five to eight members (doctors, nurses and other health-care professional staff) for maximal effectiveness. The tasks undertaken by each team member should be identified in advance (e.g. by agreed protocols) and carried out in parallel, while still maintaining priorities.
Effective teamwork in trauma care may be life- or limb-saving. It requires leadership, a structured approach and good communication. The team leader should have no competing clinical responsibilities but coordinates the team efforts and supervises patient care. The role of the team leader is generic in nature and does not depend on a particular specialty background.

Find out how a major trauma case is managed in your hospital. Who is notified in your hospital’s trauma team call-out? Who acts as the team leader? When does the team leader relinquish overall control? Who is responsible for ongoing coordination of care?

See the PACT module on Communication

**Self- and patient-protection**

Self- and patient-protection is an important aspect of trauma care. This is particularly relevant for the pre-hospital setting, where the accident scene may pose a danger to both the patient and the emergency medical personnel. Safety measures set out by specially trained staff (e.g. fire fighters, police, alpine or water rescue teams) are occasionally required and may delay access and thus mean a delay in initiating treatment.

Self-protection of emergency medical and other healthcare workers from blood-borne infections by wearing gloves and other safety apparel as indicated (e.g. protective glasses, gowns/suits and X-ray protection) is another relevant aspect of trauma care.

See PACT module on Clinical Imaging

**Primary assessment**

The primary assessment is a strictly clinical survey aiming to identify acute life-threatening injuries/complications.

Any such problem uncovered in the primary survey is managed immediately by one or more of the primary interventions outlined below. This rapid clinical assessment should be done systematically (following the ‘A-B-C-D’ approach - see below) and take no more than a few minutes (Figure 1).
1. Check for airway patency and respiration using the volar aspect of the forearm held over mouth and nostrils; check for elevation of the chest during inspiration.
2. Check for a carotid pulse; a palpable carotid pulse indicates a systolic arterial blood pressure >40-50 mmHg.
3. Check for external bleeding by visual inspection of the body. Note that thick clothing may disguise relevant haemorrhage.

4. Check for presence of shock by assessing capillary refill, peripheral temperature, venous filling and diaphoresis.

5. Check for central cyanosis by inspecting the colour of the lips. In patients with dark complexion, assess the oral mucosa. Remember that in severe anaemia cyanosis may be absent despite deep hypoxia.

6. Auscultate the lungs to detect pneumothorax and cutaneous emphysema.

7. Check for external jugular vein filling and venous congestion of the head and neck to recognise obstructive shock.

8. Check the pupils to assess isocoria and their reaction to light. You may also check for the filling of conjunctival vessels - if the conjunctivae are white and no vessels visible, haemoglobin is usually <6 g/dL.

A - Airway (with C-spine control)

Inspect the upper airways to detect obstruction e.g. by blood clots, loose teeth or dental prosthesis. Listen for snoring, gurgling or stridor as this may reflect partial airway obstruction. Remember that total airway obstruction can be silent. Watching chest movements and feeling for airflow through the nose or mouth (e.g. with the volar aspect of the wrist) are further ways to check for airway patency. A pathognomonic clinical sign for airway obstruction is inward movement of the chest during inspiration with simultaneous outward movement of the abdomen known as ‘seesaw respiration’ or ‘paradoxical chest movement’. In patients who have been intubated in the pre-hospital setting, it is important to confirm the tracheal tube placement on arrival at the emergency department - usually by a quick check of chest expansion and of the capnograph trace.

While checking for airway patency or when intervening to control the airway, C-spine control by immobilisation using manual in-line stabilisation, external fixation of the head and neck by sandbags or a semi-rigid collar should be performed.

B - Breathing

Assurance of airway patency (above) also includes some elements of assessment of breathing; the presence of breathing is checked by observing chest movements and detecting airflow through the nose and/or mouth. If breathing is present, look for signs of respiratory distress and/or central cyanosis. Remember that in severely anaemic patients central cyanosis may not be present. Further, check for symmetry of chest movements. In patients with pneumothorax, a massive haemothorax or bronchial obstruction (e.g. by foreign body, blood clot, tooth, endotracheal tube), the affected chest wall side does not follow respiratory movements of the contralateral side or lags behind them. In tension pneumothorax, the affected chest wall side may be elevated showing little or no movement. Auscultation is another method to assist the assessment of symmetry of breathing sounds. In practice, the use of percussion is only of limited help in differentiating between pneumo- and haemothorax (hyper-resonant vs muffled or dull sounds), particularly where ambient noise often interferes with the clinical examination. Palpation of the chest can detect subcutaneous emphysema as a highly specific sign of pneumothorax (Figure 2) or pneumomediastinum.
Figure 2. Massive subcutaneous emphysema evident on CT scan in a patient with severe chest trauma and a left sided pneumothorax.

Clinically, subcutaneous emphysema is diagnosed by digital palpation detecting the characteristic ‘snowball crepitus’. In case of an open pneumothorax (sucking chest wound or communicating pneumothorax), there is a defect in the chest wall. If the defect diameter is two-thirds that of the trachea or greater, air is drawn preferentially through the defect rather than the trachea. Insertion of a chest tube at a separate site and covering the wound with an occlusive dressing is the most reliable solution and has probably supplanted the use of an occlusive dressing, sealed on three sides to act like a flutter valve.

C - Circulation

Evaluate the circulation by palpating for central (e.g. carotid or femoral) pulses first. Presence of central pulses indicates a circulation with a minimum systolic arterial blood pressure of approximately 40-50 mmHg. Next, check for obvious external haemorrhage and peripheral pulses (e.g. radial) to assess pulse quality. Rapid and faint pulses reflect low systemic blood flow, usually due to hypovolaemia. Further assess peripheral perfusion by capillary refill time, skin temperature and mottling. Prolonged capillary refill time (>3-4 seconds), cold extremities and skin mottling (Figure 3) are signs of inadequate systemic perfusion which typically occur earlier than arterial hypotension. Accordingly, physiological definitions of shock do not require the presence of arterial hypotension but focus on clinical signs of peripheral hypoperfusion, tachycardia and the level of consciousness to recognise shock. When laboratory measurements can be taken, e.g. in the hospital setting, elevated lactate concentrations, an increased base deficit and/or reduced central/mixed venous oxygen saturation are indicators of inadequate systemic tissue perfusion/shock.
Figure 3. Extensive skin mottling of the lower extremities in a patient with severe haemorrhagic shock. Remember that skin mottling often starts over the knee caps.

Extended jugular veins with venous congestion of the head and neck (Figure 4) are clinical indicators of thoracic inflow obstruction (obstructive shock) as observed in tension pneumothorax or traumatic cardiac tamponade.

Figure 4. A patient with cardiac tamponade and clinical signs of obstructive shock. Venous congestion of the head is a typical and alerting sign.

D - Disability and CNS emergencies

Neurologic disability is best evaluated by the Glasgow Coma Scale score (see below). In addition however, the presence of any lateralising signs and the status of the brain stem reflexes require assessment. For brain stem assessment, the pupillary size, form and reaction to light are most readily assessed. Unilateral dilatation without a light response strongly suggests ipsilateral transtentorial herniation. Bilateral...
pupillary dilatation with no response to light is a sign of impending brain-stem compression but may also reflect intoxication or severe hypothermia. Irregularly shaped pupils result from irritation to the oculomotor nerve and characteristically precede transtentorial herniation.

**Glasgow Coma Scale**
The Glasgow Coma Scale (GCS) was developed by two neurosurgeons from Glasgow to assess the level of consciousness in patients with traumatic brain injury during the acute phase. It assesses the eye, verbal and motor response to verbal commands or painful stimuli. The maximum score is 15, the minimum 3.

<table>
<thead>
<tr>
<th>Glasgow Coma Scale</th>
<th>Score</th>
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<tbody>
<tr>
<td><strong>Eye opening (E)</strong></td>
<td></td>
</tr>
<tr>
<td>Spontaneous</td>
<td>4</td>
</tr>
<tr>
<td>To voice</td>
<td>3</td>
</tr>
<tr>
<td>To pain</td>
<td>2</td>
</tr>
<tr>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td><strong>Verbal response (V)</strong></td>
<td></td>
</tr>
<tr>
<td>Oriented</td>
<td>5</td>
</tr>
<tr>
<td>Confused</td>
<td>4</td>
</tr>
<tr>
<td>Inappropriate words</td>
<td>3</td>
</tr>
<tr>
<td>Incomprehensible sounds</td>
<td>2</td>
</tr>
<tr>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td><strong>Best motor response (M)</strong></td>
<td></td>
</tr>
<tr>
<td>in the upper limbs</td>
<td></td>
</tr>
<tr>
<td>Follows commands</td>
<td>6</td>
</tr>
<tr>
<td>Localises pain</td>
<td>5</td>
</tr>
<tr>
<td>Withdraws from pain</td>
<td>4</td>
</tr>
<tr>
<td>Abnormal flexion</td>
<td>3</td>
</tr>
<tr>
<td>Abnormal extension</td>
<td>2</td>
</tr>
<tr>
<td>None</td>
<td>1</td>
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</table>

\[(E+M+V)\].
Score 3-8 points = severe head injury  
Score 9-12 points = moderate head injury  
Score 13-15 points = mild head injury

Spontaneous flexor posturing or flexion to painful stimuli indicates injury to the upper midbrain (decortication); unilateral flexion is a sign of contralateral transtentorial herniation. Spontaneous extensor posturing or extension to painful stimuli reflects lower midbrain or pontine injury (decerebration).

![Warning](https://via.placeholder.com/150)

Although a GCS score of 8 or less is a widely accepted, clinically useful indication for tracheal intubation to protect the airway, some patients require airway protection with higher scores (e.g. those with moderate brain injury particularly if the neurological status is deteriorating; also those with maxillary trauma or upper airway burns injury).

See PACT module on Burns Injury
Cervical spine management

The patient with severe trauma is at risk of concomitant injury to the cervical spine. Traumatic brain injury patients are at particular risk with up to 8% sustaining C-spine trauma. Trauma patients, who do NOT fulfil all of the assessment criteria listed below should receive C-spine control:

- No pain or tenderness around the vertebral column
- Awake, cooperative, and not under the influence of drugs or alcohol
- No distracting pain from other injuries
- No neurological deficit.

In practice, these criteria are fulfilled if the patient is awake and can flex his neck so that the chin touches the chest without pain. The same criteria can be used to withdraw C-spine control measures in the emergency department.

In comatose patients, a normal computerised tomography scan of the cervical spine may equally render a high sensitivity (>99%) to exclude relevant trauma to the cervical spine. In more complex situations, an MRI scan may be needed to exclude cervical spine fractures or soft tissue injury.

See PACT module on Clinical imaging (Approach to cervical spine assessment and ‘SCIWORA’ - spinal cord injury without radiographic abnormality)

Primary interventions

Whenever an A, B, C or D pathology is identified, a corresponding intervention is carried out without delay. When working in a team (e.g. in the emergency department), primary assessment and interventions can often be performed simultaneously.

Some life-saving interventions may supervene over the completion of all but the most rapid of primary surveys, for example the arrest of catastrophic haemorrhage.

A, B - Airway/Breathing:

Oxygen therapy and airway management

Application of oxygen, preferentially through a face mask with a high oxygen flow (e.g. 10 L/min), is the first measure to treat hypoxaemia and increase systemic oxygen supply.

The common causes of airway obstruction in trauma are:
- Decreased level of consciousness (LOC) - often due to brain injury
- Direct trauma (e.g. major facial injuries or stab wounds to the neck)
- Evolving neck swelling e.g. haematoma in the neck
Airway obstruction needs immediate action, starting with simple airway manoeuvres, such as the jaw thrust or careful insertion of a naso- or oro-pharyngeal airway, progressing to mask ventilation and advanced airway procedures when needed.

Any suspicion of basal skull fracture is a contraindication to the insertion of a naso-pharyngeal airway.

Tracheal intubation is often necessary and is typically achieved, in expert and controlled circumstances, using a rapid sequence intubation utilising anaesthesia. Laryngoscopy may be technically difficult even without facial or neck injury. Manual in-line immobilisation, which is necessary in most trauma patients requiring tracheal intubation, impairs the view at laryngoscopy - resulting in a grade III (out of IV) view in 45% of patients.


Intubation is even more difficult if upper airway bleeding occurs or the airway is distorted by direct injury. These and other factors (e.g. light conditions, suboptimal positioning) are reasons why endotracheal intubation is a technically challenging and high-risk procedure in trauma patients, particularly in the pre-hospital setting. It should be performed or supervised by an experienced operator. Alternative airway devices need to be available to try to avoid hypoxaemia if conventional tracheal intubation fails. A surgical airway may be required.

While patients in cardiac arrest are intubated without sedation, drugs are usually required when intubating trauma patients. It is essential to avoid cardiovascular compromise by using appropriate doses of these drugs. Careful titration (remembering prolonged circulation times in patients with shock) of sedative agents (e.g. ketamine and/or benzodiazepines) is important. In expert hands, other agents may be chosen but the use of etomidate should be avoided to prevent secondary adrenocortical insufficiency which is a cause of increased morbidity.


For further information on airway management including the structured approach to the difficult airway, see the PACT module on Airway management.
**Respiratory support**
Dyspnoea following trauma must always be taken seriously. Indications for tracheal intubation and mechanical ventilation other than to protect the airway are usually to manage abnormalities of respiratory drive or mechanics and problems of gas exchange e.g.:
- Severe traumatic brain injury
- Cervical spinal cord injury with related severe hypoventilation
- Severe disruption of the thorax (fractured ribs, flail segment)
- Hypoxaemia despite application of high-flow oxygen (lung contusion, pulmonary haemorrhage, aspiration).

**C - Circulation:**

**Cardiac arrest management**
Cardiac arrest from trauma has a particularly grave prognosis and has often been considered irreversible. Recent studies showed that long-term outcome may be better than commonly assumed with ~2% of patients surviving one year or longer. When performing cardiopulmonary resuscitation in a trauma patient, it is essential to identify and treat reversible causes of cardiac arrest. These typically include severe blood loss and obstructive shock due to tension pneumothorax or cardiac tamponade. Other causes of cardiac arrest (e.g. severe brain trauma, haemorrhage from injuries to the great vessels) are largely untreatable in the acute setting.

**Intravenous cannulation and monitoring**
Establishment of intravenous access is a key intervention. Short but large-bore intravenous cannulae promote rapid fluid infusion (Hagen–Poiseuille law). In patients with severe trauma, early insertion of at least two such cannulae may be life-saving since venous filling can dramatically reduce as haemorrhage progresses. Simultaneously, before invasive monitoring is considered, non-invasive monitoring (ECG, non-invasive blood pressure, pulse oximetry, end-tidal CO₂ measurement - if mechanical ventilation has been started) is commenced.

See the PACT module on Haemodynamic monitoring and management

**Emergency haemorrhage control**
Haemorrhage control is crucial to treat the underlying cause of hypovolaemic shock. In the acute setting, this can mostly be achieved by external compression only (e.g. by local compression of superficial bleeding sites). Recent experience from military medicine suggests that haemostatic compression dressings can enhance local clot formation and increase the chances of haemorrhage control by external compression. If external compression cannot control haemorrhage from extremities, a tourniquet may be used. However, tourniquet use should be minimised due to ischaemia of distal tissues and neuropathic injury. It should preferentially be used only by those
experienced in its application. In patients with pelvic fractures, a pelvic compression belt or an external fixator (if in hospital emergency department) minimises motion at the fracture sites and reduces venous haemorrhage.

Principles of haemorrhagic shock resuscitation
Fluid replacement constitutes the mainstay of haemorrhagic shock resuscitation. Even if fluid loading results in anaemia, adequate tissue perfusion is of higher relevance than maintenance of normal haemoglobin concentrations. The reason for this is that the body has more physiologic mechanisms to compensate for anaemia than hypovolaemia-induced tissue hypoperfusion. Although synthetic colloid solutions have a higher and longer-lasting volume effect than crystalloid solutions, colloids, particularly starches, have been associated with a higher incidence of coagulopathy, transfusion and acute kidney injury and poorer outcomes in certain critically ill patient populations. Although, crystalloid solutions are now considered the first choice in resuscitation, recent evidence, from a study undertaken specifically in human resuscitation (hypovolaemic shock) circumstances, suggested that both crystalloid and colloid fluids were equally efficacious but that 90-day survival rates favoured colloids. Therefore, the importance of adequate fluid resuscitation remains the key message and ongoing research is required for further clarification of the optimum approach in differing clinical scenarios. Normal saline is best avoided as the unphysiologically high chloride concentrations in normal saline aggravate acute kidney injury and metabolic acidosis, and may thus contribute to the ‘triad of death’ in trauma.


When haemorrhage remains uncontrolled, however, liberal fluid resuscitation may be detrimental because any elevation of blood pressure at the bleeding site will prevent or disrupt clot formation. In these cases, a strategy of restrictive fluid resuscitation and acceptance of low arterial blood pressures (also known as permissive hypotension or damage control resuscitation) may be superior.

In severe arterial hypotension or concomitant traumatic brain injury, vasopressors may be used. While most reports describe the use of vasopressin or one of its derivatives, norepinephrine is the most widely used vasopressor for this indication in clinical practice.


In case of severe shock, uncontrolled haemorrhage and/or severe anaemia, transfusion of O negative or O positive packed red cells can be necessary. No absolute haemoglobin value can be stated as a definite indication for uncrossmatched transfusion. The decision always needs to be made on clinical judgment.

**Relief of obstructive shock**

In trauma, obstructive shock results from tension pneumothorax, cardiac tamponade and in extremely rare cases tension pneumomediastinum. Tension pneumothorax results from a one-way valve effect in which air is forced into the pleural cavity during inspiration but is unable to escape during expiration. This pushes the trachea, and mediastinum to the contralateral side, compressing the opposite lung but most significantly, kinking the great veins and reducing venous return dramatically. Clinical features include arterial hypotension, tachycardia, respiratory distress, tachypnoea, ipsilateral absence of breath sounds, tracheal deviation to the opposite side and jugular venous distension from mediastinal distortion.

**NOTE**  Tension pneumothorax is diagnosed clinically and should be decompressed immediately (without waiting for X-ray confirmation) by placing a large-bore intravenous cannula into the second or third intercostal space (just ABOVE the rib) in the mid-clavicular line. Remember that the first rib cannot be palpated under the clavicle and that a standard intravenous cannula may be too short to reach the pleural space in morbidly obese patients. Preparations should then be made to insert a chest drain as soon as possible since drainage of air through such a cannula is only possible over a short period of time (due to kinking or clotting within the lumen).

In the pre-hospital setting, the diagnosis of cardiac tamponade in blunt trauma is extremely difficult to make. In the emergency department, the clinical signs may be strongly suggestive but the definitive diagnosis is now by urgent (transthoracic) echocardiography. Although often suggested, needle pericardiotomy is impractical in trauma patients and rarely drains blood clots from the pericardium. Emergency clamshell thoracotomy is indicated in patients with penetrating chest trauma and
observed cardiac arrest in order to treat cardiac tamponade or stop central haemorrhage and perform open cardiac massage.

**D - Disability and CNS emergencies**

**Management of cerebral herniation**
If the primary assessment indicates signs of impending cerebral herniation, hypertonic osmotic solutions (e.g. hypertonic saline, mannitol) may be administered urgently. In addition, tracheal intubation and mechanical ventilation are commenced to protect the airway, avoid hypoxaemia and to avoid hypercarbia - usually by instituting mild hyperventilation ($\text{PaCO}_2$ 35 mmHg / 4.7 kPa approximately). Deep sedation is desirable while avoiding arterial hypotension. To prevent cerebral hypoperfusion and secondary brain injury, arterial blood pressure is maintained at normal to upper normal values (e.g. mean arterial pressure of 90 mmHg or higher) - often achieved with infusion of a vasopressor. In the situation where these urgent measures are being taken to treat impending cerebral herniation, urgent consultation with neurosurgery with a view to possible specific e.g. surgical intervention is indicated.

See the PACT module on Traumatic brain injury.

**3/ Secondary assessment and interventions**

**Assessment**

The secondary assessment follows the primary assessment and associated interventions and constitutes a systematic head-to-toe examination aiming to clinically recognise all sites of injury (Figure 5). It is important to expose the patient before the head-to-toe examination. If the victim is alert and cooperative or if a relative is present, a rapid and structured history should be taken to identify additional medical problems. The ATLS course suggests using the AMPLE mnemonic.

AMPLE:

A: allergies, M: medication, P: past history, L: last meal, E: events and environment
1. Expose the patient
2. Head: Glasgow Coma Scale to assess level of consciousness; note that scalp haemorrhage may be severe and lead to shock (especially in young children); check for periorbital and retroauricular haematoma as indicators of basal skull fractures;
bleeding from ear and nose may be associated with cerebrospinal fluid leak; test for maxillofacial stability to detect fractures
3. Neurology: assess sensory-motor function of the trunk and extremities
4. Neck: palpate to detect cutaneous emphysema; palpate the cervical spine for pain, swelling and dislocation
5. Chest: inspect the skin to recognise injuries (‘seat belt’ sign); palpate the sternum to detect sternal fractures; gently compress chest to assess for massive instability and locate rib fractures (in the awake patient); inspect and palpate the chest wall to detect flail chest
6. Abdomen: inspect the skin for signs of injury; palpate the abdomen and assess abdominal wall tension; tap and palpate the flanks to recognise renal injury and extensive retroperitoneal haemorrhage
7. Pelvis: horizontally and vertically compress the pelvis to test for stability
8. Perineum and orifices: check for lesions and bleeding from orifices
9. Extremities: inspect, palpate and move all joints of the upper and lower extremities to detect fractures and displacements; look for swelling and palpate for instability/crepitus to detect bone fractures; palpate the muscles to recognise early compartment syndrome
10. Back: log-roll the patient; inspect, palpate and tap the spine to detect pain, swelling and dislocations

In the emergency department, a basic set of laboratory analyses (e.g. blood count, coagulation, kidney/liver function, muscle enzymes) is ordered. Varying from centre to centre, this set may also include further measurements such as cardiac biomarkers, toxicology including ethanol as well as HIV/hepatitis serology.

The secondary survey and related interventions, while vitally important, must never take priority over primary survey concerns. Sometimes, obvious injuries may need to be temporarily overlooked to focus on life-threatening problems. This may mean a patient having emergency surgery before the secondary assessment is completed - or even begun e.g. the need for laparotomy to stop major intra-abdominal bleeding.

**Imaging techniques**

Although X-ray and sonography are important in trauma management, a standardised, protocol-based head, neck, chest, abdomen and pelvis CT scan (so-called trauma scan) is the central radiologic technique when managing the multiple trauma patient. Using modern high-resolution CT scanners, such an examination can be performed within ~5 minutes and renders a reliable overview of most relevant injuries. In addition, the use of contrast medium allows for detection of relevant ongoing haemorrhage (requires minimum bleeding rate 0.5 mL/min).

Timing of the CT scan during the acute phase is crucial. Over the last years, many emergency departments have adopted protocols to perform the CT scan as early as possible, e.g. immediately following the primary assessment. In pre-hospital, physician-based, emergency medical systems, the CT scan can even be performed directly upon hospital admission assuming that primary assessment and interventions have already been addressed during the pre-hospital phase.

In haemodynamically unstable patients, the CT scan may be delayed and identification of the bleeding site achieved using sonography. Using a structured approach, the extended focused abdominal sonography in trauma (eFAST) (Figure 6), has a high sensitivity in detecting intra-abdominal bleeding sites. In addition, it may
be used to exclude/diagnose cardiac tamponade. In experienced hands, sonography may further be used to examine the chest and lungs following trauma. Lung sonography shows a higher or at least equivalent sensitivity to X-ray for the diagnosis of pneumothorax, haemothorax and/or lung contusions. Despite the benefits of lung sonography, a chest X-ray is mostly performed during the acute phase in haemodynamically unstable trauma patients.

Figure 6. Extended Focused Assessment with Sonography for Trauma (eFAST). See legend below for text.
1. Hepatorenal space (Morison pouch)
2. Splenorenal space (Koller pouch)
3. Pouch of Douglas
4. Pericardium
5. Pleural cavity left and right

Based on the results of the secondary assessment of the extremities, X-rays of the sites of suspected injuries are usually performed following the CT scan, when there are no other urgent interventions needed.

Rarely, fiberoptic techniques such as bronchoscopy and oesophagoscopy are indicated during the acute phase of trauma care e.g. when injuries to the oesophagus or tracheobronchial tree are suspected in patients with pneumomediastinum. Angiography is only rarely used as a sole diagnostic procedure but has become a highly effective method to control haemorrhage from various bleeding sites (see below). Transcranial Doppler sonography, although not common in resuscitation situations, can be used to detect elevated intracranial pressure non-invasively.

**Interventions**

**Monitoring the patient**

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**NOTE** It is important to remember that neither an arterial nor a central venous catheter is life-saving - a large-bore peripheral IV cannula is. If haemorrhage is ongoing, insertion of an arterial and/or central venous line must not delay resuscitative, haemorrhage control.

Additional cannulations and comprehensive monitoring of the patient is typically completed following the CT scan or while other imaging techniques are performed. Whenever indicated and if time allows, this includes insertion of an arterial and central venous catheter as well as placement of a urinary catheter and/or nasogastric tube. Further, invasive monitoring (e.g. intracranial pressure, cardiac output measurements) is typically initiated at a later stage (when resuscitation and stabilisation is reasonably complete) either in the operating room or the intensive care unit.

See PACT module on Haemodynamic monitoring and management

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**Warning** Before inserting a nasogastric tube, it is important to exclude basal skull fracture (Figure 7) - which may be difficult. If in doubt, consider an oro-gastric tube.
**Figure 7.** Intracranial position of supposed naso-'gastric' tube. Arrow: tube misplaced in the brain.

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**Haemorrhage control**

Timely control of ongoing haemorrhage and prevention of further blood loss are essential steps in caring for the trauma patient. Haemorrhage control can be achieved by various methods as outlined below. Optimally, the most effective, most rapid and least invasive approach should be chosen.

**Haemorrhage control using surgery**

Control of external or internal haemorrhage by surgery is a common strategy. Surgical interventions during the acute phase are often necessary for various indications e.g. craniotomy, reposition of fractures/dislocations, spinal stabilisation/spinal cord decompression, wound debridements, laparotomy for hollow viscus injuries, abdominal packing or surgical repair of open eye injuries. Further surgical intervention may be required at a later time for definitive treatment.

**Haemorrhage control using interventional radiology**

In this technique, bleeding arterial vessels are occluded by the endovascular approach by insertion/injection/embolisation. Stents (Figure 8), coils, particles, foam, plugs or microspheres are used. Although angiography can be used to identify the site of haemorrhage, this has often been already achieved by other imaging techniques (e.g. sonography, CT scan). Common bleeding sites suitable for interventional embolisation are the pelvis, retroperitoneum, chest and/or abdominal wall. Most interventional radiologists leave the introducer sheath for the angiography catheter in place for 24 hours following the intervention to have immediate vascular access available in case re-bleeding occurs.
**Figure 8.** Pelvic haemorrhage before (left) and after (right) interventional placement of a stent graft

**Conservative management of haemorrhage**
Some sites of haemorrhage stop spontaneously or by (self-)tamponade. This is often the case for extremity bleeding, haemothorax or visceral organ haemorrhage (e.g. liver, spleen, kidney). Splenic (Figure 9) and renal haemorrhage is often better managed by a conservative approach, since surgical technique commonly involves organ removal, which may have far-reaching consequences for the patient, particularly the young. A conservative approach to haemorrhage management can only be considered if the patient is cardiovascularly stable (following fluid resuscitation).
When pursuing a conservative strategy for haemorrhage management, the patient needs close monitored in a high-dependency or intensive care unit. Such monitoring for any signs of blood loss (tissue perfusion, heart rate, haemoglobin concentrations) is key to the detection of ongoing or new bleeding, which would be an indication for immediate surgical intervention. Repeated imaging of the bleeding site (e.g. liver, spleen or kidney) may be useful but requires that comparable imaging techniques are used during the initial and subsequent examinations.

Depending on the extent and location of haemorrhage (e.g. central vs subcapsular bleeding in the liver), these patients should be closely observed and monitored for 3-5 days since this is the most common time period when re-bleeding occurs. During this period, bed rest is commonly ordered; during the first 24-48 hours oral intake is minimised to small amounts of fluids or nothing in case surgery or emergency airway control becomes necessary.

**Trauma-induced coagulopathy and transfusion management**

See also PACT module on Bleeding and Thrombosis

Trauma-induced coagulopathy (TIC) or Acute Traumatic Coagulopathy describes the impairment of physiological clot formation and stability in trauma patients. Although the terms have been used interchangeably, TIC is not the same as disseminated intravascular coagulation (DIC). While TIC occurs early in the course of trauma, disseminated intravascular coagulation is often a late phenomenon often only arising in response to infection and sepsis during post-resuscitation critical illness.

Varying degrees of TIC affect up to 25% of patients admitted to a trauma centre and are observed in nearly all patients with severe injuries. TIC may precipitate blood
loss and may even interfere with definite surgical haemorrhage control. Presence of TIC is a factor adversely affecting trauma outcome e.g. transfusion needs, organ failures, ICU admission, length of stay, infection, functional outcome and mortality.

TIC is currently assumed to result from several pathophysiological mechanisms:
- Shock and endothelial dysfunction
- (Local and systemic) hyperfibrinolysis
- Loss of coagulation factors and platelets
- Dilution of coagulation factors - primarily fibrinogen (Factor I)
- Hypothermia and acidosis

The combination of hypothermia, metabolic acidosis and coagulation abnormality has been termed the ‘triad of death’.

**Recognising TIC**
Standard coagulation tests (e.g. prothrombin time, activated partial thromboplastin time) are of limited value to diagnose TIC and guide coagulation therapy. Whole-blood viscoelastic tests, such as rotational thromboelastometry/-graphy, can more sensitively and reliably assess the complex nature of TIC (Figure 10). These laboratory methods describe initiation, formation and stability of the blood clot and can give more comprehensive insight into the pathologies of the coagulation system during acute haemorrhage (Figure 11).

**Figure 10. Normal thromboelastography.**

<table>
<thead>
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<th>1</th>
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<th>3</th>
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<td><strong>Fingeramputation APTM</strong></td>
<td><strong>Fingeramputation FIBTEM</strong></td>
<td><strong>Fingeramputation</strong></td>
</tr>
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<td>St.: 19h13</td>
<td>St.: 19h13</td>
<td>St.: 19h13</td>
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<tr>
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<tr>
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<td>CI: 83s</td>
<td>CI: 62s</td>
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<tr>
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<td>CFT: 82s</td>
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<td>alp: 69°</td>
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</table>
Thromboelastography in a trauma patient with severe haemorrhage showing delayed clot formation (prolonged clotting and clot formation time) as well as reduced clot stability due to dilution/loss of fibrinogen. Different protocols based on the results of thromboelastometry/graphy have been proposed to guide coagulation management in trauma. A strength of thromboelastometry/graphy is its high sensitivity to recognise systemic hyperfibrinolysis (Figure 12). Limitations are the inability to integrate temperature effects (measurements are performed at 37 °C) or assess local coagulation pathologies (e.g. local hyperfibrinolysis). Platelet dysfunction and the effects of various anticoagulant drugs (e.g. vitamin K-antagonists) are also difficult to evaluate. At the time of writing, no study has proven a relevant outcome benefit related to thromboelastometry/graphy-guided coagulation therapy when compared to treatment guided by conventional coagulation tests.
Thromboelastography in a multiple trauma patient, with haemorrhage and traumatic brain injury, revealing systemic hyperfibrinolysis. Given the importance of TIC for trauma outcome, early and aggressive coagulation management is crucial when caring for bleeding patients.


General coagulation therapy
Aggressive maintenance of normothermia is a technically simple but practically challenging task in the trauma patient e.g. in the emergency department when the patient is unclad and examined while other procedures are conducted. It is important that the entire trauma team is aware of the detrimental effects of hypothermia for trauma outcome.
Correction of acidosis is only meaningful if it occurs as a consequence of shock reversal. Infusion of sodium bicarbonate to correct metabolic acidosis does not improve coagulation. Use of normal saline as a resuscitation fluid can aggravate acidosis by causing hyperchlaeraemia and should be avoided.

**Transfusion of red blood cells**

Crossmatching of red blood cells should be requested when the patient fulfils any of the following criteria:

- Reduced haemoglobin concentration (see below)
- Systolic blood pressure <90 mmHg (adults)
- Heart rate >120/min (adults)
- Depressed Glasgow Coma Scale score <9
- Hypothermia (<35°C)
- Base deficit >2 mmol/L
- Elevated lactate levels (>2 mmol/L)
- Abnormal thromboelastometry values
- Injury Severity Score >22 points or other score count indicative of severe injury
- Planned surgery with a relevant risk of haemorrhage
- Visible haemorrhage

The haemoglobin trigger to transfuse red blood cells in acute haemorrhage is not different from other conditions viz. it ranges between 7 and 9 g/dL. No distinct injury pattern (e.g. traumatic brain injury, spinal cord injury, chest trauma) has been found to benefit from higher transfusion triggers. Practically however, it needs to be kept in mind that, particularly the first haemoglobin concentration taken in trauma patients, is often falsely high because fluid resuscitation has not yet been started. Similarly, during fluid resuscitation, the situation is often complex - there may be haemoglobin being lost (bleeding), haemoglobin being gained (transfusion) and dilution of haemoglobin levels (fluids). Red blood cell transfusion is usually based on clinical estimation of the relative contributions of the above.

**Tranexamic acid (and mortality reduction)**

Adult trauma patients with significant haemorrhage (systolic arterial blood pressure <90 mmHg or heart rate >110 bpm or both) or who are considered to be at high risk of significant haemorrhage should receive tranexamic acid. A first bolus dose of 1 g over 10 minutes should be given as early as possible, but no later than 8 hours following injury. A second dose of 1 g is administered over 8 hours started immediately following the initial bolus. This approach reduces the risk of overall mortality and in particular the risk of death from bleeding.


**Specific coagulation therapy**

Once TIC occurs, specific coagulation therapy is indicated (see also next section on massive haemorrhage/transfusion protocols). Fresh frozen plasma is recommended when the prothrombin and/or activated partial thromboplastin time exceeds 1.5 times the normal value and/or fibrinogen concentrations drop <100-150 mg/dL. When transfusing fresh frozen plasma, it is important to administer adequate quantities e.g. 10-15 mL/kg. Fresh frozen plasma is transfused as soon as possible following rewarming since most coagulation factors rapidly lose their activity at room temperature.

Platelet transfusion of 4-8 bags of platelet concentrate (equivalent to 1 apheresis unit) is recommended when the platelet count falls to <75,000/µL (in the presence of ongoing haemorrhage), or to <100,000/µL (in traumatic brain injury or massive haemorrhage - see below).

A major disadvantage of fresh frozen plasma is the time delay associated with administration. It takes time for warming, transport from the blood bank to the patient and for transfusion (e.g. when 10-15 mL/kg are administered). In addition, fibrinogen concentrations in fresh frozen plasma are typically low (150-350 mg/dL). Considering that fibrinogen concentrations in TIC are the first to drop due to loss and dilution, particularly in young trauma patients, fibrinogen concentrates or cryoprecipitates are often indicated to treat hypofibrinogenemia (<100-150 mg/dL) or reduced clot stability (e.g. maximum clot firmness in the fibrinogen measurement of the thromboelastometry <10-12 mm).

Prothrombin complex concentrates contain coagulation factors II, VII, IX and X (as well as protein C and S) and are typically indicated to reverse therapeutic anticoagulation with vitamin K-antagonists. For other indications in acute haemorrhage they are rarely indicated (e.g. prolonged prothrombin times or clot formation times in thromboelastometry, despite fresh frozen plasma transfusion). In addition, coagulation factor concentrates have a relevant risk of inducing venous and arterial thromboembolic complications.

There is no current evidence that the use of recombinant factor VIIa can improve morbidity and mortality in acutely bleeding trauma patients. However, its use has been associated with a significant risk of arterial thrombosis. Similar to other coagulation factor products (e.g. factor XIII, IX or XII), recombinant factor VIIa is only indicated in trauma patients with chronic coagulation disorders, but may be considered in patients with ongoing haemorrhage and persisting coagulopathy.


**Massive haemorrhage/transfusion protocols**
Massive transfusion has been defined as either 10 or 20 units of red blood cells transfused within 24 hours. Such definitions can only be applied in retrospect and are therefore not very useful when resuscitating a bleeding trauma victim.


In massive haemorrhage, it is unwise to withhold coagulation therapy until TIC can be detected since in certain populations the risk of TIC is excessively high and almost inevitably present. Empiric initiation of coagulation therapy in these patients should start at the beginning of resuscitation in the expectation that severe forms of TIC can be prevented and haemorrhage control facilitated. Fulfilling two or more of the following four criteria predicts a high risk of massive haemorrhage and should trigger the local massive transfusion protocol:

- Penetrating injury mechanism
- Positive FAST or CT scan for free intra-abdominal fluid
- Admission systolic arterial blood pressure <90 mm Hg
- Admission heart rate >120 bpm


When massive transfusion protocols are activated, a pre-defined set of red blood cells, fresh frozen plasma and platelet concentrates should be sent from the blood bank to the emergency department. The ratio between red blood cells, fresh frozen plasma and platelet concentrates varies with local protocols. Evidence in 2013 suggests a ratio of these blood components of 1:1:1 may be beneficial in massive
haemorrhage (Zink KA reference, below). Depending on coagulation test results, specific therapies can be administered. Clinical experience is that when using such massive transfusion protocols, fibrinogen concentrates or cryoprecipitate are the most commonly required additional coagulation products.


In such clinical scenarios, it is important to administer blood products through high-performance warming systems to avoid hypothermia and use filters to remove air bubbles which often form during warming of cold blood products.

Complications of massive transfusion (in addition to the typical transfusion-related risks - see below) should be kept in mind:
- Transfusion-associated circulatory overload (TACO)
- Transfusion-related acute lung injury (TRALI)
- Dilutional coagulopathy (fibrinogen being the first affected)
- Hypocalcaemia
- Hyperkalaemia
- Hypothermia

**Reversal of chronic anticoagulation therapy**
The changing epidemiology of trauma means an increasing proportion of injured patients will be older and receiving chronic anticoagulation therapy. Vitamin K-antagonists therapy is a significant risk factor for death in these patients. In addition to vitamin K-antagonists, several other anticoagulation therapies are in current use. The table below summarises half-life times and antagonists for commonly used drugs.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Mechanism of Action</th>
<th>Duration of Action</th>
<th>Antagonist</th>
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<tbody>
<tr>
<td>Acetylsalicylic Acid</td>
<td>COX inhibition</td>
<td>3-5 days</td>
<td>Platelets</td>
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<td>Clopidogrel</td>
<td>ADP antagonist</td>
<td>5-7 days</td>
<td>Platelets</td>
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<td>Rivaroxaban</td>
<td>direct factor Xa inhibitor</td>
<td>24 hours</td>
<td>None (PCC, FVIIa?)</td>
</tr>
<tr>
<td>Dabigatran</td>
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<td>24–36 hours</td>
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<td>48-96 hours</td>
<td>FFP or PCC + vitamin K</td>
</tr>
<tr>
<td>Phenprocoumon</td>
<td>vitamin K antagonist</td>
<td>5-6 days</td>
<td>FFP or PCC + vitamin K</td>
</tr>
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COX, cyclooxygenase; ADP, adenosine diphosphate; PCC, prothrombin complex concentrate; FVIIa, activated Factor VIIa; FFP, fresh frozen plasma.
Although fears are often expressed about normalising coagulation in the patient with a mechanical heart valve, the risk of bleeding is the immediate concern and the risk of a mechanical heart valve clotting is about 8% per year.

Damage control resuscitation and surgery

Damage control surgery involves rapid surgery to control bleeding and decontaminate wounds using simple procedures. Definitive surgical repair is deliberately postponed to minimise surgical trauma and avoid aggravation of hypothermia and metabolic acidosis. Definitive anatomical repair is completed once physiological stabilisation has been achieved.

This strategy is applied in the severely injured who sustain uncontrolled bleeding. Characteristic injury patterns which may require the damage control approach are penetrating torso injuries, severe pelvic and/or abdominal trauma with uncontrolled bleeding from the liver or spleen or any great arterial vessel. The need for a damage control strategy is usually identified by clinical indicators of massive haemorrhage (e.g. gross abdominal expansion) or a failing response to standard resuscitation (e.g. when systolic arterial blood pressure drops <90 mmHg despite initial resuscitation). The damage control strategy requires exceptionally good and ongoing communication between the surgeon, anaesthesiologist, intensivist and other team members.

Figure 13: Tip of knife in descending aorta following stab in the back.

White arrow: tip of knife,
Blue arrow: descending aorta cut open for controlled removal of the knife.

Damage control resuscitation includes restrictive fluid resuscitation and permissive hypotension (systolic arterial blood pressure >80 mmHg or mean arterial blood
pressure >50 mmHg) to reduce further blood loss.


Permissive hypotension is contraindicated in patients with central nervous injury (e.g. traumatic brain injury, spinal cord injury). A vasopressor (either norepinephrine or vasopressin) is a better choice to maintain minimum blood pressure for heart and brain perfusion than fluid loading since fluid infusion causes haemodilution, increases blood flow in all tissues and thus enhances blood loss from uncontrolled bleeding sites. The concept of vasopressor use, on the other hand, is to maintain blood flow to the heart and brain while leaving other tissues vasoconstricted and (in this case - deliberately) hypoperfused. Correction of hypovolaemia occurs only following surgical haemorrhage control. Massive transfusion protocols with aggressive coagulation therapy (see above) are often initiated in these patients. Prevention of hypothermia and avoidance of normal saline (not to aggravate metabolic acidosis) is central.

NOTE The vast majority of major trauma cases will not need damage control. In a stable patient, the aim should be for 'early definitive surgical care'. Clinical judgment is needed to identify the rare patients whose survival depends on a radically different approach.

The four phases of damage control

1. Initial assessment and change to damage control resuscitation: The need for damage control is recognised, the resuscitation strategy adopted and the patient transferred immediately to the operating room.

2. Immediate, limited surgical intervention: Haemorrhage and contamination are controlled using temporary methods, such as packing of visceral organs and exteriorisation (by stoma creation) of the bowel, if necessary. Sometimes, the wound may need to be left open to prevent abdominal compartment syndrome (e.g. using a vacuum dressing – Figure 14). A damage control laparotomy should not take longer than 90 minutes. Another common damage control technique is external stabilisation of extremity fractures.
Figure 14. Management of a trauma patient with an open abdomen.

Note the (vacuum dressing) following damage control laparotomy.

3. Continuing stabilisation on the ICU: This phase includes physiological system control (restoration of tissue perfusion, transfusion, specific coagulation management), monitoring for wound complications (e.g. abdominal compartment syndrome, if the abdomen has been closed) and continuing vigilance for missed injuries.

4. Re-operation: Definitive repair can take place during this phase, which typically follows after 12-48 hours of stabilisation. If the wound was left open at the initial operation, wound closure and definitive repair may be possible now.

**Anecdote** A motorcyclist required a damage control laparotomy to stop intra- and retroperitoneal bleeding and to repair perforations of the stomach and bowel. The abdomen was packed and left open. The patient was becoming cold and acidotic at the end of surgery but still had a comminuted, unstable fracture of the distal femur and tibial plateau. The trauma surgeon abandoned the usual, careful but lengthy approach to realignment. In 15 minutes, a temporary external fixator across the knee joint was applied, accepting an imperfect position.

Neurosurgical interventions

For further information see the PACT module on Traumatic Brain Injury.

Pain relief

Pain relief is an essential part of trauma care and must be administered as soon as the situation allows when caring for the injured. In severely/multiply injured patients, pain relief is best achieved by titrated, intravenous administration of opiates. Non-opiates often fail to adequately relieve pain when used alone but may be useful supplemental agents. While bone and joint pain responds well to non-steroidal anti-inflammatory drugs such as diclofenac, their use in severely and
multiply injured patients is restricted in view of their potential to induce kidney injury and transient thrombopathy.

For short and painful interventions (e.g. repositioning of fractures or dislocations), consultation with an anaesthesiologist may be very useful. Ketamine is an attractive agent as it is not only an analgesic but also an anaesthetic drug but adequate monitoring and facilities and expertise in airway management are required. Use of ketamine is usually combined with small doses of benzodiazepines to prevent hallucinations and nightmares. Regional and peripheral anaesthetic techniques may be considered in selected patients.

**NOTE** Remember that analgesia may deplete the victim of his own catecholamines thus unmasking previously undiagnosed hypovolaemia and leading to haemodynamic collapse.

**4/ POST-ACUTE TRAUMA CARE INCLUDING ORGAN-SPECIFIC MANAGEMENT**

The post-acute phase of trauma care commences in the intensive care unit. Typically, bleeding has been stopped during the acute phase.

**The goals of the post-acute phase are:**
1. Completion of resuscitation and restoration of tissue perfusion
2. Completion of history taking and diagnostics (missing injuries)
3. Re-establishment of homeostasis and physiologic functions
4. Organ- and injury-specific management
5. Scheduling of tertiary interventions and surgeries
6. Initiation of early physical therapy and planning of rehabilitation

One important link between the acute and post-acute phase is the hand-over. All team members contributing to the care of the patient need to take part in this hand-over.

**NOTE** When handing over information to another team, there is a recognised risk for miscommunication. However, it is a useful opportunity to summarise information. Follow a structured (e.g. checklist) approach.

See the PACT module on Communication.

**Shock resuscitation**

Following haemorrhage control, the goals of shock resuscitation focus on restoration of tissue perfusion. It is important to remember that macrocirculatory parameters such as arterial or central venous blood pressure as well as cardiac output are only poor indicators of microcirculatory perfusion. Indirect parameters of tissue perfusion
such as arterial or venous lactate concentrations and its changes over time (lactate clearance), peripheral perfusion (skin mottling, capillary refill time), central/mixed venous oxygen saturation and urine output are more relevant resuscitation endpoints. Depending on the clinical situation, fluids, blood, vasodilators, inotropes and/or vasopressors are used to restore tissue perfusion. Resuscitation endpoints must be adjusted in patients with traumatic brain injury where arterial blood pressure is relevant to avoid cerebral hypoperfusion and secondary brain injury.

**Transfusion and coagulation management**

During the post-acute phase of trauma, a restrictive transfusion protocol targeting haemoglobin concentrations between 7-9 g/dL has been shown to minimise the adverse effects of blood transfusion on morbidity and mortality. The most common and serious complication of transfusion in trauma patients is immunomodulation, imposing an increased risk of secondary sepsis with each unit of transfused blood.


Coagulation management is continued also during the post-acute phase of trauma although, following haemorrhage control, it is usually clinical signs of bleeding (e.g. drainage content - Figure 15) that determine the need and intensity of coagulation management. It is important to remember that standard coagulation parameters such as the prothrombin or partial thromboplastin time do not correlate with the risk of bleeding. Pro-coagulants should only be administered in case of ongoing or renewed bleeding - the arterial and venous thromboembolic risk should always be kept in mind.
Figure 15. Clotted blood in thoracic drainage.

Clot formation is indicative of an adequate coagulation response in a patient following chest trauma.

See PACT module on Bleeding and Thrombosis

The systemic inflammatory response syndrome and host response

Trauma, tissue injury and shock are among the strongest non-infective stimuli of the immune system. This explains the pronounced pro-inflammatory response most patients exhibit during the first days following trauma. This pro-inflammatory response depends on individual factors (e.g. genetic immune pattern) and is further aggravated by therapeutic interventions such as surgery, blood transfusion, (non-protective) mechanical ventilation and extracorporeal therapies. Therefore, interventions which are known to increase inflammation (e.g. prolonged surgery) are likely best avoided or postponed until inflammation attenuates. Clinically, stimulation of the immune system is recognised by non-specific signs such as tachycardia, tachypnoea, fever, leukocytosis, elevated C-reactive protein serum concentrations as well as organ dysfunctions.

The first days of pro-inflammation are followed by a combined pro- and anti-inflammatory response of variable length. This phase is again followed by a phase of compensatory anti-inflammation during which infection and sepsis are common and lethal complications can occur in severely injured patients.
**Management of specific injury patterns in the ICU**

*Head and neck*

**Traumatic brain injury**
See the PACT module on Traumatic Brain Injury.

**Maxillary and facial trauma**
Maxillary trauma, particularly when dislocated fractures are involved, often requires surgery. Some surgical interventions can be done during the acute phase depending on the patient’s overall condition but can often wait days or longer without detriment. Swelling is a typical problem following maxillary/facial trauma and usually lasts three to five days following trauma. Secure the airway well during this time. Sometimes early tracheostomy can even be indicated in these patients.

Completion of the diagnostic work-up often requires involvement of other specialists (e.g. maxillary surgeons, ENT surgeons, ophthalmologists) and an interdisciplinary approach to determine the optimum care for the patient.

In all patients with fractures of the frontobasal bones, it is important to detect leakage of cerebrospinal fluid (CSF). This can be done by laboratory detection of the beta-trace-protein, a protein found at higher concentrations only in CSF. An undetected leak from the dura (and therefore an open brain injury), puts the patient at increased risk of intracranial infective complications (e.g. subdural empyema, intracerebral abscess). Timely closure of the leak is important to reducing the infection risk. There is no strong evidence on whether the use of prophylactic antibiotics in this situation is advisable or not.

Maxillary trauma is one of the rare indications for antibiotic prophylaxis in trauma patients. The reason for this is the frequent association with open brain trauma as well as the high physiological bacterial load of the sinuses, nose and mouth. An amino-penicillin antibiotic (e.g. ampicillin) is a good choice to cover the upper respiratory tract flora of most trauma patients.

**Neck trauma**
Trauma to the neck is rare. Frequent causes are penetrating injuries, e.g. due to stabbing or injury from strangulation. Whenever trauma to the neck occurs, injury to the airways (including the larynx) as well as carotid and vertebral arteries needs to be excluded. Swelling and/or fracture of the larynx may be fatal complications of neck trauma. As in maxillary and facial trauma, early tracheostomy may be considered as an option to protect the airway and bridge the time until swelling is reduced and spontaneous breathing is again possible.

**Chest**

**Lung contusion and early post-traumatic acute respiratory distress syndrome**
Pulmonary contusions with or without rib fractures are a relevant source of morbidity and mortality. Alveolar structures may be disrupted with bleeding into the airway. Pulmonary capillary permeability increases, leading to alveolar oedema that further impairs gas exchange. It may resemble and cause the acute respiratory distress
syndrome (ARDS) within the first 24-48 hours following trauma. In mild cases, there is a minor disturbance in oxygenation but resolution normally occurs within 72 hours. In severe cases, its presentation may be dramatic with fulminant pulmonary oedema during the first 24 hours following trauma (Figure 16). In patients with traumatic chest compression (e.g. high velocity injuries), high pressure lung oedema may develop. High pressure oedema may be accompanied by subconjunctival haemorrhage and petechiae of the face and neck due to acute increases of venous pressure resulting from high intrathoracic pressures.

**Figure 16. Severe ARDS after high velocity chest trauma**

This patient required veno-venous extracorporeal membrane oxygenation.

Non-invasive ventilation may be a therapeutic option during the early phase. However, in severe cases progressing to ARDS, intubation and invasive ventilation is required. Lung protective ventilation (optimum positive end-expiratory pressure, tidal volumes of 6 mL/kg and controlled plateau pressures) is an important management principle to reduce ventilator-associated lung injury. Prone positioning improves oxygenation and reduces mortality in severe ARDS (PaO$_2$/FiO$_2$ <150mmHg / 20kPa). Veno-venous extracorporeal membrane oxygenation may be a life-saving therapy in patients with the severest forms of lung injury - as in the case of Fig 16.

See the PACT module on Respiratory failure.

Rib fractures
The clinical significance of most rib fractures result from secondary injuries caused by the fracture segments (e.g. pneumothorax, lung contusion, haemorrhage from an intercostal artery, laceration and bleeding from liver or spleen). Clinically, pain and chest wall instability are the main complications directly related to rib fractures. Adequate pain management is essential to facilitate non-shallow spontaneous breathing and adequate chest physiotherapy (mobilisation of secretions). In patients with lower rib fractures, continuous thoracic peridural or paravertebral analgesia is a highly effective method.

Chest wall instability (flail chest) is a complication of serial rib fractures. In flail chest, at least two ribs must be fractured in at least two places to create a chest wall segment that moves paradoxically with respiration. (See reference below for video of flail chest). Although this makes ventilation less efficient, it is the underlying pulmonary contusion and severe pain that cause the associated respiratory problems. It is important to recognise patients with a flail chest (clinical diagnosis) early, since these patients usually require mechanical ventilation for several days. Early extubation carries a high risk of extubation failure and hypoxaemia. Rarely (in carefully selected patients) surgical rib stabilisation may be necessary.

Callaghan MA, Phelan D. Flail chest from blunt thoracic trauma. BMJ case reports 2011 (Aug 8). PMCID: PMC31166588

Pneumo- and haemothorax
Pneumothorax results from injury to the lung either by excessive increases of the transpulmonary pressure gradient or by external laceration (e.g. through fracture segments of ribs). Chest drain insertion is indicated in the majority of pneumothoraces. Small pneumothoraces which can only be seen on a chest computed tomography for example, may be treated by oxygen inhalation alone. Most pneumothoraces resolve spontaneously following drainage using adequately-sized chest drains. If lung injury is severe, bronchopleural fistula may occur. These fistulae often present as a persistent pneumothorax with continuous bubbling in the chest drain and failure of the lung to expand. In these patients, early spontaneous breathing needs to be achieved to keep transpulmonary airway pressure as low as possible to allow for fistula closure. Surgery with or without lung resection is indicated only for large fistulae or those persisting for several days or weeks.

Most cases of haemothorax are caused by lung lacerations or tears to intercostal or internal mammary vessels (Figure 17). These are often self-limiting. A haemothorax may also result from great vessel injuries (often rapidly fatal) and fracture-dislocations of the thoracic spine. The first-line therapy of haemothorax is insertion of a chest drain to evacuate the blood from the pleural cavity and allow for re-expansion of the lung. Compression of the bleeding site is typically achieved with this approach and is sufficient to stop further haemorrhage. Thoracotomy should be considered only if chest drain amounts exceed 1.5-2 litres within short periods of time.
Figure 17. Large left-sided haemothorax in a chest trauma patient.

Note the shift of the mediastinum to the contralateral side indicating the space-occupying effect of the blood in the left pleural cavity.

Aortic injury

Traumatic rupture of the thoracic aorta is a rare but potentially fatal injury, 80-90% of patients with an aortic injury being known to die at the scene. The anatomical site of the rupture (or traumatic dissection) is most frequently at the aortic isthmus. Those who survive to reach the hospital usually have an incomplete, contained rupture. The risk of rupture is relatively small within the first two weeks following trauma. Therefore, aortic repair which is mostly achieved by stenting and only rarely by open aortic surgery (nowadays) should be performed urgently but not emergently during this time window. In the acute setting, other injuries such as traumatic brain injury or abdominal injuries may have a higher priority for emergency surgery.

Until definitive repair of the aortic injury is achieved shear stress at the site of aortic injury should be kept at a minimum. Practically, this is achieved by limiting heart rate (<80 bpm) and systolic arterial blood pressure (<120-140 mmHg) using beta-blockers. This strategy may likely be in conflict with the management of traumatic brain injury but it is difficult not to give priority to managing the brain injury. Some compromise may be agreed between the parties involved including the neurosurgeons. Theoretically, drugs which reduce arterial blood pressure by reducing afterload may increase torque at the site of injury and increase the risk of bleeding.

Clinically, symptoms may be subtle ranging from hoarseness and stridor (as a sign of damage to the recurrent laryngeal nerve or compression of the tracheobronchial tree) to rare intercapular pain. Cardiovascular signs include hypotension (from haemorrhage), hypertension (from afferent nerves responding to a stretch stimulus), a systolic murmur, a pulse deficit between left and right arm or between arms and legs, and distal ischaemia. Paraplegia from spinal cord ischaemia may follow thoracic aortic rupture (or its surgical management). Radiologic signs are the everyday pointer to this pathology and need to be in mind from the evaluation of the first CXR taken.
They include a widened mediastinum, rightwards deviation of the oesophagus, obliteration of the aortic knuckle/knob, a left apical cap and a left-sided haemothorax.

**NOTE** If traumatic dissection or rupture of the aorta is suspected clinically and radiologically, the diagnosis needs rapid confirmation / exclusion by an (ECG-triggered) CT angiogram or a transoesophageal echocardiography.

**Rare thoracic injuries**
Tracheobronchial trauma is a rare but potentially fatal injury. In almost all cases surgery is indicated. Until this is achieved, transient bridging of the site of injury using an tracheal tube or stent may be possible. Similarly, oesophageal trauma is managed surgically. It is essential to perform surgery early to avoid mediastinitis which could limit the possibility of surgical closure of the oesophageal site of injury.

Similarly, clinically relevant cardiac trauma is rare. Most injuries to the heart are asymptomatic and recognised by supra/ventricular extrasystoles, tachyarrhythmias and mildly elevated cardiac biomarkers e.g. troponins. Injuries to the ventricular wall or valves require early surgical repair. Rarely direct trauma to a coronary artery may cause occlusion and myocardial infarction.

Diaphragmatic rupture is often missed initially since the classical features - bowel sounds heard in the chest, nasogastric tube seen in the chest on X-ray and referred pain to the shoulder - are commonly absent. Diaphragmatic rupture occurs in about 5% of patients hospitalised after chest injury. It occurs mostly on the left side, is more common in penetrating than blunt trauma and surgical repair is necessary.

**Abdomen**

**Visceral organ injuries**
The most relevant complication of visceral organ injuries in the ICU is re-bleeding. This may occur not only in conservatively managed injuries, but also following open or interventional haemorrhage control. Organ dysfunction as a direct consequence of trauma is rare but may occur in severe liver trauma. Following nephrectomy a transient increase in serum creatinine and urea concentrations is often observed. Antibiotic prophylaxis is only indicated in renal trauma.

Following splenectomy, leukocytosis and thrombocytosis commonly occurs. When the platelet count exceeds 1,000,000/µL administration of acetylsalicylic acid is indicated to prevent thromboembolic complications. Furthermore, antibiotic prophylaxis is standard and vaccinations (pneumococci, haemophilus influenzae, meningococci) must not be forgotten before patients following splenectomy are discharged from the hospital. In addition, patients need to be well instructed on their increased risk of infection, the overwhelming postsplenectomy infection syndrome and their increased risk of acquiring tropical infections. An asplenia card should be handed out to these patients.

**Hollow viscus injury**
Hollow viscus injury is rare but carries a high morbidity and mortality if not diagnosed during the acute phase. Early recognition is crucial to allow for timely
surgical repair. In these cases, peritonitis is not common and antibiotic therapy is only indicated for short intervals e.g. 24-48 hours. A common postoperative complication is gastrointestinal paralysis (ileus).

Complications are serious only if hollow viscus injury is detected late (>24 hours following injury). While surgical repair may be difficult and require secondary interventions, delayed recognition of duodenal perforation can preclude surgical repair and make drainage of the perforation site the only therapeutic option to facilitate healing of the perforation site. Hollow viscus injury is seldom a contraindication to early enteral nutrition.

See PACT module on Nutrition

In oligo/anuric patients with enlarging ascites, bladder rupture must always be considered. Surgical repair is the treatment of choice. In these patients, serum urea and creatinine concentrations are typically elevated resulting from absorption of these substances and other urinary solutes over the large peritoneal surface area.

![NOTE]

Even on a high-quality CT scan, organ perforation (especially of the duodenum) may not be visible. A high index of suspicion and repeated clinical and radiological examinations are required.

Pancreatic injury
Relevant trauma to the pancreas is uncommon. In most cases, it is caused by a direct blow to the upper abdomen e.g. from the steering wheel in sudden deceleration. Although it may clinically be asymptomatic in the beginning, it can later cause diffuse upper abdominal pain and tenderness. Bleeding from pancreatic injury is a rare complication which typically needs surgical repair. Pancreatic injury may also lead to pancreatitis with chemical peritonitis and third space fluid loss.

See PACT module on Pancreatitis.

Abdominal compartment syndrome
Any increase of volume in the abdominal cavity can increase the intra-abdominal pressure. In abdominal trauma, this can occur due to packs, swelling, intestinal paralysis (ileus) and/or haematoma. When intra-abdominal pressure exceeds 15 mmHg, intra-abdominal venous congestion and organ dysfunction is likely to occur. Any increase of intra-abdominal pressure >20 mmHg together with the occurrence of new organ dysfunctions represents an abdominal compartment syndrome and requires decompression. In trauma patients, this can most often only be achieved by surgical intervention.

See also the PACT module on Abdomen in acute/critical care medicine.

**Pelvis**

Typical ICU complications of pelvic trauma are intestinal paralysis (ileus) and the abdominal compartment syndrome. Extensive haematomas are a frequent cause of increased intra-abdominal pressures. In acute pelvic haemorrhage, surgical decompression of increased intra-abdominal pressure should occur only following
haemorrhage control to avoid precipitation of bleeding.

Pelvic fractures, particularly those involving the hip joint, often require complex surgical interventions for stabilisation. These interventions are usually not performed during the acute period. Until surgical repair is performed, extension of one or both lower extremities may be necessary. Prolonged immobilisation of these patients even after surgical repair carries thromboembolic risks and negatively affects early rehabilitation and physical therapy.

Fractures of the anterior pelvic ring often involve urogenital injuries including the bladder (see above) or urethra. Urologic expertise is needed to diagnose urogenital trauma and/or place the urinary catheter.

**Extremities**

Extremity injuries (fractures, soft tissue or muscular trauma, amputations) are frequent. During the post-acute phase, the two most relevant complications observed in trauma involving the extremities are the compartment syndrome and rhabdomyolysis with acute kidney injury. Fat embolism syndrome is rare, but should be considered if its clinical signs occur. When managing extremity trauma, note the following:

- In case of vascular injuries requiring surgical or interventional revascularisation, therapeutic heparinisation may be indicated.
- Repeated clinical assessment of arterial perfusion of the extremity is crucial. In some patients, infusion of vasodilators (e.g. prostaglandins) may be indicated to improve arterial perfusion.
- Antibiotic prophylaxis is indicated in patients with open fractures.
- Clinicians should have a high index of suspicion to diagnose nerve lesions which are often only diagnosed during the post-acute phase in the intensive care unit and reversal may be possible by surgical decompression of the nerve.

**Compartment syndrome**

Direct or indirect trauma (e.g. by fractures) causes haematoma and secondary oedema which increases pressure within fascial compartments. In some cases, compartment syndrome can also result from tight casts, dressings or compression devices. Although the lower leg is the most common location where compartment syndrome occurs, it can develop in other parts of the extremities as well (lower arm, upper arm, thigh, buttocks). With increasing pressure, venous return is compromised and subsequently arterial perfusion of the muscles.

In awake patients, one of the earliest signs of compartment syndrome is burning pain (typically out of proportion to the clinical appearance) and in injuries involving the lower leg numbness between the first two toes and the inability to elevate the big toe (compression of the superficial perineal nerve). Loss of two-point discrimination may be another early sign. In sedated or comatose patients, palpation or direct measurement of compartmental pressures are appropriate to detect compartment syndrome. Although frequently used, pulse oximetry measurements at the affected extremity are of only limited value since they will only detect a fall in arterial perfusion which is a late sign of compartment syndrome. When compartmental pressures rise to 30 mmHg in normotensive or to 20 mmHg or higher in hypotensive
subjects, surgical fasciotomy is indicated to reduce compartmental pressure, optimally within six hours. Wound dressing can be performed using a vacuum dressing with staged wound closure following reduction of muscular swelling and oedema.

**Rhabdomyolysis with acute kidney injury**
Damage to muscle cells release myoglobin into the plasma which is filtered through the glomeruli into the renal tubules. There, precipitation of myoglobin with obstruction of the tubules and direct injury to the proximal tubular cells can occur at high myoglobin concentrations. This precipitation is most marked at low urinary pH. When serum myoglobin concentrations have reached >1000 mg/dL, acute kidney injury may occur. In clinical practice, serum creatine kinase concentrations are often measured instead of myoglobin, but it is important to remember that creatine kinase has no nephrotoxic potential and is only measured as an indicator of muscle injury. To prevent acute kidney injury from myoglobinuria (so-called crush injury), adequate renal perfusion and systemic hydration is crucial. Optimally, urine output should reach 1.5 mL/kg/h to increase tubular flow and avoid obstruction. Diuretics are not helpful and may even impose further nephrotoxicity. Although not evidence-based, making the urine more alkaline (to a pH >6.5 if possible) by infusion of sodium bicarbonate solutions is another frequently used, theoretically useful therapeutic option to avoid or attenuate myoglobin-induced acute kidney injury.

See also the PACT modules on Oliguria and anuria (Acute kidney injury Part I) and Acute kidney injury Part II: renal replacement therapy.

**Fat embolism syndrome**
Fat embolism syndrome (FES) describes a constellation of neurologic and pulmonary dysfunction combined with thrombocytopenia, petechiae and fever. It typically occurs following long bone and pelvic injuries but may develop in response to severe shock states as well. The pathophysiology is unclear and may involve physical (particulate) and/or chemical mechanisms. Neurologic features, including agitation or coma, usually occur within 24 to 48 hours following injury. Pulmonary dysfunction / ARDS develops simultaneously or a few hours later. Thrombocytopenia and a petechial skin rash (typically in the axilla (Figure 18) or the groin) is last to develop at 72 to 96 hours after the trauma. Although the diagnosis is primarily a clinical one, detection of retinal changes or fat globules in the urine (a non-specific sign) may help. Other than fixation of long bone fractures, no definitive therapy for fat embolism syndrome is required, just support of any organ dysfunctions - usually poor gas exchange and confusion or delirium. Clinical signs usually resolve within a few days. Visual impairment may be a disabling long-term complication of fat embolism syndrome and is due to microvascular fat embolism of retinal arteries.
Figure 18. Characteristic (axillary) petechial rash of the fat embolism syndrome. The FES in this patient followed a fracture of the femur. The rash evolved only after the patient had become agitated and comatose, developed respiratory failure and finally became thrombocytopenic (67,000/µL).

Spine and spinal cord

While no pharmacological intervention has been shown to improve functional outcome following spinal cord injury, early surgical decompression and avoidance of tissue hypoxia and spinal cord hypoperfusion may be beneficial for spinal cord regeneration. Incomplete lesions may have a higher chance of regeneration than complete lesions.

Injuries to the spine and particularly the spinal cord are associated with several potential complications during the post-acute phase. Transsection of sympathetic spinal cord fibres compromises cardiovascular function. In spinal cord lesions higher than thoracic vertebrae 4-6, sympathetic stimulation of the heart is lost resulting in relative bradycardia, reduced pump function and arterial hypotension.

Haemodynamic instability (neurogenic shock) is often precipitated by decreased peripheral vascular tone resulting from loss of sympathetic nervous stimulation. Volume repletion together with inotropic and/or vasopressor therapy may be necessary to maintain arterial blood pressure at high enough levels to adequately perfuse the injured spinal cord (e.g. mean arterial blood pressure >65-70 mmHg). Autonomic dysfunction (imbalance between sympathetic and parasympathetic tone) can result in bradycardia and even asystole particularly in upper thoracic and cervical spinal cord injuries. These arrhythmias are often triggered by vagal stimulation (e.g. suctioning, coughing, etc.). Ten percent of patients with cervical spinal cord injuries exhibit bradyarrhythmias requiring transient cardiac pacing. Permanent cardiac pacing is required in less than 1% of all patients with cervical spinal cord injuries.

Any lesion of the spinal cord compromises respiratory function. The higher the lesion the more pronounced are its effects on the respiratory system. While lesions of the
Lumbar spinal cord affect the innervation of abdominal and pelvic auxillary muscles and can compromise coughing, lesions of the thoracic and lower cervical spinal cord affect intercostal muscles and increase chest wall compliance by eliminating muscular tone. Lesions above C3-5 affect innervation of the diaphragm. Depending on the chronic health status of the patient before trauma, mechanical ventilation is typically required in cervical spinal cord lesions at C6 or higher. In these patients tracheostomy may facilitate weaning and should be considered following the first week of invasive mechanical ventilation. Patients with spinal cord lesions at or higher than C3-5 have little chance of being permanently free of mechanical ventilation and often have a tracheostomy fashioned early.

Further complications of patients with spinal cord injuries include infective complications (pneumonia), gastrointestinal paralysis (ileus), thromboembolic complications (Figure 19) and pressure ulcers. In patients with cervical spinal cord injuries, hypothermia, hyponatraemia and anaemia are further potential complications arising during the post-acute phase.

**Figure 19.** Bilateral pulmonary embolism in a patient with spinal cord injury (left).

When pulmonary embolism leads to shock in trauma patients, systemic thrombolysis is often contraindicated due to a high bleeding risk. In these cases, regional thrombolysis (e.g. 5 mg bolus injection of recombinant tissue plasminogen activator (rtPA) followed by continuous infusion of 10 mg over 15 hours) using a catheter (right) is an effective alternative therapy.

**NOTE** Ten per cent of patients with a cervical spine fracture have a second non-contiguous vertebral column fracture.

**Special consideration: pregnancy**

Although physiologic maternal changes increase the pregnant woman’s capacity to compensate for blood loss, the fetus is at particular risk of morbidity and mortality following trauma. While direct trauma to the enlarged and highly vascularised uterus can result in severe haemorrhage and fetal demise, indirect changes may compromise fetal oxygen delivery and cause death at any gestational age. The most common cause of fetal mortality is hypoxia due to maternal anaemia, hypoxaemia and/or uterine hypoperfusion. The most frequent need for preterm delivery following trauma is as a result of abruptio placentae. Considering the adverse side effects of beta2-mimetics (e.g. tachycardia, vasodilatation) in patients with shock, tocolysis is not routinely indicated for preterm labour following trauma. When maternal haemorrhage has been controlled and cardiovascular function stabilised, labour may
be inhibited using magnesium sulphate. Repeated sonographic examinations of the fetus should be performed. Continuous cardiotocographic monitoring is indicated if obstetricians consider it useful and are available to interpret and respond to new anomalies. Immunoprophylaxis with Rh immunoglobulin is indicated in every Rh negative mother to prevent alloimmunisation following trauma. If transfusion is required, strict attention is paid to transfuse Rh negative blood only.

See the PACT module on Obstetric critical care.

5/ General ICU Care of the Trauma Victim and Rehabilitation

Nutrition

Enteral nutrition should start early following trauma, ideally within 24 hours. Contraindications to early enteral nutrition are rare and include severe shock, abdominal compartment syndrome and hollow viscus perforation (mostly only during the first 24 hours). Gastroparesis and intestinal paralysis (ileus) can prevent reaching full enteral nutrition within the first 3-4 days following trauma. Pharmacological stimulation using dopamine-antagonists (e.g. metoclopramide, domperidone), motilin-agonists (e.g. erythromycin) or cholinesterase-inhibitors (e.g. prostigmine) can reduce the time of gastrointestinal paralysis and facilitate enteral nutrition uptake.

The optimum time point to supplement with parenteral nutrition is unknown, but may not be before day 5 or 7. Blood sugar control in trauma patients does not differ relevantly from other critically ill patients and should target a blood sugar level of 85-180 mg/dL (4.7-10 mmol/L). In patients with traumatic brain injury insulin-induced reductions of blood sugar levels <110 mg/dL (6.1 mmol/L) may precipitate cerebral energy failure and should be avoided.


Nutritional demand is difficult to assess. Initially, patients are usually catabolic but during recovery, patients become anabolic and have increased nutritional demand.

See the PACT module on Nutrition.
Thrombo-embolism prophylaxis

The risk of venous thromboembolism and pulmonary embolism is high in patients after severe trauma. Patients with pelvic and/or lower extremity fractures as well as those with the abdominal compartment syndrome are at exceptionally high risk to develop deep vein thrombosis. Therefore, thrombo-embolism prophylaxis is a key part of the general management of the injured patient. However, as long as haemorrhage persists or the risk of re-bleeding is high, prophylactic anticoagulation using heparins may not be possible. Mechanical thrombosis prophylaxis devices (compression stockings, intermittent pneumatic compression stockings) should be used to bridge the time when no pharmacological anticoagulation can be administered. Remember that intermittent pneumatic compression stockings have the potential to increase or re-induce haemorrhage from pelvic injuries and should be avoided in these patients. In some patients with complex fractures of the lower extremity and an increased risk of haemorrhage, thromboembolic prophylaxis may be impossible. Retrievable inferior vena cava filters may be an alternative method for thromboembolic prophylaxis in these patients. As soon as possible, ideally on the day of trauma, thrombo-embolism prophylaxis with heparins, preferentially low-molecular weight heparins, should be initiated.

Antibiotic prophylaxis

Antibiotic prophylaxis is rarely indicated in severely injured patients but should be considered (see Task 4 on organ-specific trauma care) in the following:

- Open traumatic brain injury
- Maxillary fractures involving sinuses
- Open fractures
- Hollow viscus injury
- Renal trauma leading to haematuria

It is important to remember that the severity of trauma alone is no indication for antibiotic prophylaxis. Neither is insertion of an intracranial pressure device or drainage device an indication for antibiotic prophylaxis. Unrestricted use of antibiotic agents increases secondary infectious complications by selecting resistant bacteria and fungi. Depending on the vaccination status of the patient, refreshing of tetanus vaccination is important.

Pain relief, sedation and delirium management

Analgesia should be provided according to the injuries sustained and the patient’s response to its administration. While opioid infusions are convenient and easily adjusted, pain from some injuries does not respond well to them e.g. neuropathic pain from neural injury. Regional anaesthetic techniques can provide optimum analgesia in patients with chest, abdominal, pelvic and/or extremity injuries.

Propofol in conjunction with opioid analgesics is a common means of sedating a mechanically ventilated trauma patient in the ICU. In resistant patients, benzodiazepine use may also be required, possibly with an adjunct such as clonidine. Note that sedation resistance may be a sign of inadequate analgesia.

Unless indicated for traumatic brain injury, severe shock or respiratory failure, the intention in ICU is that patients are awake and cooperative or easily rousable.
Repetitive small bolus injections of sedative and/or analgesic agents can help to achieve this goal and avoid over-sedation. Sedation scales should be used to describe the sedation target and document sedation depth. Over-sedation is a common but easily avoidable complication of sedation therapy in critically ill trauma patients. Prolonged mechanical ventilation and lengths of stay in the ICU as well as increased rates of cranial computed tomography scans, delirium and secondary infections are serious complications of over-sedation.

See PACT module on Sedation and Analgesia.

Delirium is a common complication following trauma, particularly if the brain has been injured. While hyperactive delirium is easily recognised, hypoactive delirium is a frequently overlooked and serious condition which adversely affects (cognitive) patient outcome.


Non-pharmacological measures (e.g. adequate analgesia, environmental noise reduction, re-establishment of a day-night rhythm, communication, etc.) should be used both to prevent and manage delirium. Antipsychotic drugs such as haloperidol but also newer anti-psychotics such as quetiapin or risperidone are often used to mitigate hyperactive delirium. Remember that none of these drugs has been proven to effectively treat delirium and might actually aggravate agitation and confusion. Their use should be restricted to patients who cannot be managed with non-pharmacological measures alone. The avoidance of benzodiazepines may also be an important step to avoiding or managing delirium.

See the PACT module on Sedation and Analgesia.

**Tertiary surgical interventions**

During the post-acute phase, further operative interventions are often necessary to achieve definite surgical repair of fractures or other injuries (e.g. aortic injury). The timing of these interventions should depend on the clinical course of the patient in ICU or beyond. When cardiopulmonary function has been stabilised and homeostasis re-established, tertiary surgical interventions can be performed. The correct time point must be determined by discussion with all the senior clinicians involved (most often Critical Care and Surgical) taking into account potential advantages (e.g. improvement of pain control, possibility of early mobilisation) and disadvantages (e.g. inflammatory stimulus, haemorrhagic complications, aggravation or renewed organ dysfunction) of the surgical procedure.

**Rehabilitation**

Rehabilitation has been defined as the process of returning the patient as completely as possible to physical, social and mental well-being. Rehabilitation is a crucial
component of trauma care which should start early in the ICU and continue for as long as it is beneficial after ICU discharge. Rehabilitation centres often have long waiting lists and issues such as confirmation of cost coverage by insurance companies (where applicable) may take longer than expected. Therefore planning the rehabilitation process after hospital discharge should start in the ICU. In tracheostomy patients for example, the intensivist may have a particular role in facilitating early progress to cuff deflation, speaking valve speech and to decannulation.

**Physical and psychological rehabilitation**

The rehabilitation team, including specialists in rehabilitation medicine, physiotherapists, occupational and speech therapists, and others, set realistic goals for each patient, aiming to optimise function and to facilitate independence and reintegration into social life and work whenever possible.


The psychological effects of trauma may be complex and long-lasting. Besides grieving the loss of their personal health and inability to function normally, patients may express feelings for others who died or were disabled in the incident. Pain, fatigue and financial consequences also cause distress and demotivation. Depression and feelings of guilt over others who have not survived need to be recognised and managed.

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**ANECDOSE** A 35-year-old trauma victim made a reasonable recovery and was suitable for mobilisation in wheelchair. Unfortunately, he had developed a plantar flexion and therefore could not use the wheelchair properly. Weeks of delay in the Rehabilitation Unit might have been avoided by earlier recognition and expert management e.g. by injection of botulinum toxin into the calf muscles while in ICU. Rehabilitation is expensive but less so than disability!
CONCLUSION

There is no single correct treatment for a trauma victim. There is a common generic approach that underpins optimal care: it involves early emphasis on the ABCD approach to save lives and on the secondary survey to assess the pattern and severity of injuries and avoid missed injury. Early operative treatment may be in the form of immediate damage control surgery in unstable patients (rare) or definitive surgical repair in stable patients (common). Standard Intensive Care management is fundamental to overall care and metabolic and host defence considerations assume increasing importance during ICU stay. Further surgery may be needed for reconstructive procedures. Rehabilitation, which should start in the intensive care unit, is the essential ‘finishing school’ that allows each trauma victim to reach their full potential.
PATIENT CHALLENGES

A 30-year-old woman was the unrestrained driver of a car travelling at high speed on a motorway. She was involved in a road traffic collision which forced her car into a ditch. Her car rolled over and she was ejected, landing on a fence post, which penetrated her trunk. From the initial details relayed by the paramedics at the scene, it was not clear whether the entry point was in the abdomen or the chest. The post is not now embedded in the wound.

Epidemiology of major trauma - ‘accidents’ are the fifth most common cause of death in the USA.


Mechanisms of injury

Q. When you arrive at the scene, what are your priorities in terms of site safety?

A. You want to confirm that the accident site safe? Is there a danger of other vehicles crashing into the victim's car? Is the road slippery due to oil or ice? Is there an explosion hazard due to a fuel leak? You check with the fire or police services that the scene has been declared safe.

Q. What is your approach on reaching the victim?

A. You follow an ABCD algorithm - as outlined in the ATLS (Advanced Trauma Life Support) manual.

Precautions before approaching the trauma patient

The ABCD approach to initial clinical management

http://www.facs.org/trauma/atls/index.html
The woman’s airway and breathing are intact but she has a blood pressure of 92/64 mmHg and a Glasgow Coma Scale (GCS) score of 11 at the scene (eyes 3, verbal 3, motor 5). She seems to have severe pain in her neck.

Q. Given that her Airway and Breathing are OK (at least for the present), how do you respond to the documented hypotension?

A. Place a large bore (14G) IV cannula (two if possible) and start fluid therapy.

Q. What is the most likely cause of the hypotension?

A. Hypovolaemia is the most likely cause in trauma victims.

Q. What is your priority in relation to blood pressure management?

A. It is essential to avoid profound hypotension, especially if there is cerebral injury.

PACT module on Hypotension

**Learning Issues**

Generic approach to trauma care - Task 2.

Q. Given that transport to a hospital will be necessary shortly, what are the treatment goals for the hypotension in this initial phase?

A. It is useful to set appropriate goals for vital parameters during the transport for each patient. Accepting a systolic blood pressure of 70-80 mmHg is recommended in the pre-hospital care of penetrating trunk injuries and other situations in which uncontrolled bleeding is suspected.

Q. Might the blood pressure goal vary depending on the patient?

A. Yes, a mean blood pressure of 65 mmHg may be sufficient for a young skier with a broken femur while the same person with a head injury may require a mean pressure of 90 mmHg to maintain cerebral perfusion.

Q. What intravenous fluids are preferred, and how rapidly should they be administered?
A. It is the amount of circulating blood volume, rather than the type of replacement fluid (colloid or crystalloid), that is important. If hypovolaemia is thought to be immediately life-threatening, warmed fluid (crystalloid as Ringers Lactate is used most frequently) should be given rapidly while the haemodynamic goals above are being attained.

Q. Why is serial frequent re-assessment of the vital sign trend in response to therapy important?
A. Further treatment will be guided by the response to therapy. If there is a poor response to volume, no time must be lost on the scene and a rapid transfer to the appropriate hospital is particularly imperative.

**NOTE** Repeat assessment is a key issue in trauma care.

Q. Considering that correcting hypotension in the face of uncontrolled bleeding may cause the bleeding to increase, is full resuscitation to euvolaemia and normal blood pressure appropriate now.
A. No. Normalising the blood pressure may be inappropriate at this stage.

Q. Given that blood pressure may be maintained due to cardiac reserve and increased endogenous catecholamines (stimulated by pain or anxiety), is there any hazard in treating the patients pain?
A. Haemodynamic decompensation may occur after sedation or analgesia (since this reduces catecholamine levels), especially if hypovolaemia co-exists. A cautious, monitored approach is required.

**NOTE** Even though blood pressure is often regarded as the key determinant of organ perfusion, it is flow (not pressure) that matters most.

Intensive care management must be started before the patient physically reaches the ICU. Extended ICU care may be invaluable in the treatment process.

Q. Other than the well-recognised risk of fluid overload and pulmonary oedema, are there other possible disadvantages of overzealous fluid administration?

A. Other disadvantages might include dilution of clotting factors, hypothermia (which may worsen coagulation abnormalities and increase the risk of infection), and an increase in perfusion pressure which might conceivably cause dislodgement of clots and further bleeding.


En route to hospital, the patient is pale, her pulse rate is 65/min and her blood pressure is 75/45 mmHg. She is becoming harder to rouse.


Q. Given the ‘lucid interval’ and the likely brain injury, what is the main differential diagnosis for the decreased responsiveness?

A. A delayed fall in level of consciousness (LOC), after a ‘lucid interval’, in these circumstances is typical of an epidural (extradural) haematoma.

Q. What non-traumatic causes might you consider for a decrease in LOC?

A. These include the use of drugs (including alcohol), abnormal blood glucose levels, and abnormalities of cerebral perfusion.

www.facs.org/trauma/optimalcare.pdf

PACT module on Traumatic brain injury
PACT module on Coma and altered consciousness
The patient's point-of-care blood glucose level is 6.5 mmol/L (116.6 mg/dL) 30 minutes after the accident. Her GCS score is 10 (eyes 2, verbal 3, motor 5). There is no evidence of drug abuse. She is still moaning and seems to be in severe though vaguely localised pain. The nearest hospital is five kilometres away, while the nearest trauma centre is 50 km away (30 km of motorway, approximately 30–45 minutes by car).

Q. Should this victim be taken directly to the trauma centre, or should she first be transferred to the smaller unit for stabilisation?

A. The literature suggests that outcome in severely injured patients is better if they are taken directly to a specialist trauma unit. Centralised expert care is preferred, provided that the extra journey time does not incur unacceptable risks.

Q. Do you need to consider alternatives to the above two options? If yes, please give examples.

A. Options may have to be considered. For example, a helicopter emergency medical service (HEMS) may be available (in addition to the road ambulance) for advanced medical treatment on site. Helicopter transfer may be considerably faster, depending on weather, road and traffic conditions. An alternative might be to meet the helicopter at the nearby rural hospital to facilitate rapid secondary transfer after immediate stabilisation there. However, such an approach should not delay the transfer.

Learning Issues

Trauma team response
Role / indications for referral to a trauma centre
Conflicting priorities

**NOTE** It is prudent to ‘expect the worst’ and prepare for it rather than to be overwhelmed by the severity of injury.

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PACT module on Transportation

It was decided to take the victim to the trauma centre. Members of the hospital trauma team check drugs and equipment prior to the patient’s arrival.

Q. Given that the team’s success is greatly enhanced by proactive decision making, what else can the trauma team do to prepare optimally for receipt of the patient?

A. They members assess what resources (personnel, equipment and drugs) will be needed and what is available. The plan is shared by all so that everyone follows the same strategy. This includes the pre-emptive assignment of roles, e.g. team leadership, responsibility for ventilation or vascular access. Communication with radiology and with surgical /operating theatre team may be indicated as urgent abdominal or thoracic surgery may be needed.

Learning Issues

Trauma team response

NOTE Trauma care is a team rather than an individual activity. Teamwork depends on competence, cooperation, coordination and continuity.

Learning Issues

Assessment of trauma care resources

PACT module on Communication

On the basis of the pre-hospital report suggesting a penetrating chest injury, a cardiothoracic surgeon is called. When the patient arrives, it is evident that the fence post has penetrated her lower abdomen rather than her chest. She is now drowsy and confused, with a respiratory rate of 35/min.


Q. How should the patient be assessed now?

A. Once again, evaluation follows the classic approach, with a primary and secondary assessment. This does not differ in principle from the pre-hospital evaluation, and is repeated as necessary during the stabilisation phase.
The woman is in obvious respiratory distress, with severe tachypnoea and expiratory wheezing. No obvious 'A' (airway) problem can be found. However, there is a severe 'B' (breathing) problem: respiratory distress with absent breath sounds in the right side of the chest. Other signs at this time include congested neck veins, subcutaneous emphysema and arterial hypotension. An intern on your team wonders if immediate intubation of this patient with severe respiratory distress would be appropriate.

Q. You consider the intern’s thought to be ill-advised. Give reasons.

A. There are signs of a right-sided pneumothorax, probably already under tension, which first requires immediate correction.

An adverse outcome (serious hypotension and shock) might result from intubation and positive pressure ventilation. What might the mechanisms, of such a complication, be?

Q. From the intubation?

A. Presuming it is performed with the assistance of sedative / anaesthetic drugs, the hypotension (due to the removal of the endogenous catecholamine effect) may be severe particularly if hypovolaemia is present.

Q. From the positive pressure ventilation?

A. The positive pressure ventilation may force even more air into the pleural cavity, worsen mediastinal shift and further compromise cardiac filling, potentially leading to a catastrophic fall in cardiac output and even a fatal outcome.

Communication with the pre-hospital team is crucial for successful planning and to avoid misunderstandings: e.g. an ‘injury to the knee’ may be conveyed onwards as an amputation at the knee!

Needle decompression is performed in the second intercostal space, in the mid-clavicular line, without waiting for a chest X-ray. There is an evident hiss of air, and the breathing pattern improves immediately.

Q. The subcutaneous emphysema suggested a pneumothorax as the underlying diagnosis in this instance. If it had not been present, what other diagnoses would you have considered given the other clinical signs?
A. Severe asthma may present with these clinical features and the presence of wheeze particularly raises this possibility. A pericardial effusion with cardiac tamponade and diaphragmatic rupture may also present as dyspnoea, congested neck veins and hypotension.

The expiratory wheezing in this instance disappears after release of the tension pneumothorax and you consider this to have been probably due to tracheal shift and expiratory obstruction caused by the tension pneumothorax. There is no ‘A’ problem and the patient’s respiratory status has improved and the neck vein distension has resolved. However, needle thoracostomy has provided only a preliminary solution for the pneumothorax. A chest drain needs to be inserted for definitive treatment.

Q. What size chest drain should be inserted?
A. In a traumatic pneumothorax, anticipate draining blood as well as air from the pleural cavity, even if the clinical signs point to a pure pneumothorax. While a 24 FG drain may be sufficient to drain air, a larger drain size (28-32 FG) is used for blood.

Q. If the needle thoracostomy has relieved the respiratory distress, how urgent is the chest drain after needle decompression?
A. Moderately urgent as the needle may become displaced.

Q. Might the completion of the one-minute primary assessment (i.e. the remaining C and D) be more important than beginning this procedure, which will take several minutes to complete.
A. Yes

Learning Issues

Management of life-threatening injuries
Trauma team response

Q. Taking into account the pre-arrangements that have been made, outline how the task (of chest drain insertion) might be allocated so that team members can provide optimal care?
A. One member should insert the drain while another continues with the assessment - in conformity with the prearranged roles or on instructions from the team leader.

Q. What should the team leader be doing now? Should s/he get involved in clinical procedures?
A. The team leader stands back and observes the situation - coordinating information, assigning tasks and only intervening personally in critical situations. A leader who is directly involved in clinical manoeuvres may lose the overview.

**Learning Issues**

Trauma team approach
Role of the trauma team leader

While the team proceeds with the initial evaluation, a lack of motor activity in the lower limbs is noted. The patient remains hypotensive, with a blood pressure of 75/52 mmHg and heart rate 60/min). Although the tension pneumothorax has been treated, her respiratory pattern is still abnormal - there is rocking movement of her chest and abdomen which is not due to airway obstruction or a flail segment.

Q. You are concerned that there may be a spinal cord injury. Would it be a possible cause of the hypotension and respiratory dysfunction that is evident now?

A. Spinal cord injury explains many of the findings and the lack of spontaneous limb movement is a telling sign. Loss of intercostal muscle action leads to a diaphragmatic breathing pattern, sometimes with a rocking movement. It also explains the lower than expected pulse rate and the neck pain.

Q. How might you achieve extra information to support your diagnostic suspicion?

A. Patients who are able to communicate can be asked about numbness and whether they can move their limbs and can be tested clinically for motor and sensory levels. Hypotension, particularly if not otherwise readily explained, and slow pulse rate are consistent with the diagnosis.

Q. How will the secondary survey help?

A. The spine will be examined for swelling, tenderness and deformity, while maintaining spinal precautions.

Q. Will spinal imaging be necessary?

A. Yes, spinal imaging will be mandatory with this mechanism of injury, regardless of the clinical findings.

PACT module on Clinical imaging

Q. What other causes could explain persistent hypotension in this patient?
A. The primary differential diagnosis is bleeding (external or internal) and remains the working diagnosis until ruled out. Other causes, such as overwhelming head injury (interfering with brain-stem activity), myocardial contusion/infarction, sepsis or allergy are unlikely but are considered during the secondary survey.

Q. Is tachycardia invariable in the bleeding patient?

A. Although tachycardia is a characteristic finding, severe haemorrhage can occasionally be associated with a bradycardia and must be considered an ominous sign which may precede complete circulatory collapse.

NOTE: Severe head injuries may be found in patients with few symptoms. A high index of suspicion needs to be present when evaluating so-called 'minor head injury'. A guideline for when a cerebral CT scan is indicated can be found in the following reference.


As one resident inserts the chest drain and another the arterial cannula, the patient’s GCS score improves to 13 (eyes 3, verbal 4, motor 6). The radiologist wants to know which investigation to prioritise. The neurosurgeon is insistent that an immediate head CT scan is required for assessment.

Q. What imaging is required during the resuscitation phase in the emergency department? How will prioritisation be done now?

A. The trauma team leader is still in charge, but must consider suggestions from other specialists. Clinical examination during the primary assessment has yielded information on immediate life-threatening injuries and guided the immediate interventions, such as thoracocentesis and tracheal intubation. These manoeuvres have priority over precise anatomical diagnosis and definitive care of specific organs.

Q. Which imaging has priority as adjuncts to the primary survey?

A. Plain X-rays of the chest (CXR) and pelvis, combined with ultrasonography of the trunk, provide invaluable information immediately.

Q. Emergency CT scans of the head and abdomen provide more detailed information. When should they be done?
A. They will be appropriate as soon as the secondary clinical assessment has been completed. Other imaging to identify fractures and dislocations can be performed later.

As the ABCD order determines the priorities and this unstable patient may not yet be safe in the CT scan, the urgent CT is deferred until after the CXR.

Learning Issues
Conflicting priorities
Other potentially life-threatening injuries

NOTE: If a film does not affect immediate management, it can usually be deferred until the end of the secondary assessment. Review the chest and (perhaps the pelvic) X-ray. Focus on how they influence immediate management and treatment - seek evidence of further injury (e.g. bony, aortic or pulmonary) and check the position of tracheal and intercostal tubes etc. A chest X-ray may be unnecessary if a chest CT scan is being performed.

PACT module on Clinical imaging

Q. The clinical assessment of the abdomen is worthwhile but diagnostically unreliable. What are the advantages of computed tomography in this case? Is contrast administration useful?

A. Fast, reliable computed tomography has made other interventions, such as peritoneal lavage, obsolete. In an obtunded polytrauma victim, a complete head, neck and trunk CT scan is justified and can be performed in a few minutes and 3-D reconstructions allow the vertebral column to be assessed. The administration of contrast shows the great vessels.

Q. In this instance, there is a penetrating wound to the abdomen and emergency laparotomy is necessary. Is there a role for abdominal CT?

A. While a CT scan may help identify injuries to solid organs and to the spine, immediate surgery may be a better option if there is any further cardiorespiratory instability.

Despite the diaphragmatic breathing pattern, the patient continues to breathe spontaneously and reasonably comfortably and her oxygenation remains stable (saturation of 97% on 40% oxygen). Her airway seems secure and she can swallow. She has been given 2000 mL of crystalloid infusion and her mean arterial blood pressure has increased to 65 mmHg but she is still bradycardic (52/min) and she has well-perfused skin.

Loops of bowel are protruding from the abdominal wound with no evidence of active
bleeding from this source or any other. Abdominal ultrasound reveals a moderate quantity of intra-abdominal free fluid. The wound is covered with a saline-soaked temporary dressing. Excluding the bradycardia, there does not seem to be an active ‘C’ problem.

However, while accepting the limitation of the diminished GCS score, the victim seems uneasy and has considerable pain in her neck.

\[\text{NOTE}\] Beware of haemodynamic parameters that contradict the clinical scenario, e.g. bradycardia with severe pain or following abundant blood loss.

**Learning Issues**

PACT module on Arrhythmia


The intern reports that she has spoken to a family member and that the patient does not have a cardiac history and is not taking medications - such as β-blockers, digitalis or amiodarone. She is also not an athlete with a low baseline heart rate.

Q. Given the above information, outline three injuries that might cause bradycardia acutely in this patient?

A. Cardiac contusion or ischaemia is possible.

A spinal lesion above the upper thoracic (T4-6) level may cause bradycardia - due to loss of the sympathetic supply to the heart.

Injuries to the eye or extraocular muscles may (very occasionally) lead to reflex bradycardia.

Q. As a supplement to clinical examination, how might myocardial injury be excluded or confirmed?
A. By a 12-lead ECG, a biomarker (troponin) level and by echocardiography.

Q. Whatever the cause, is the bradycardia important to her management?
A. Yes, it will compromise the compensatory mechanisms for shock.

**Learning Issues**

**Spinal injury**

On painful stimuli at her upper limbs, the victim reacts with flexion of her arms. With further evaluation, you note that stimulation below the shoulders produces no motor response. Her respiratory rate is still 25/min, and paradoxical ‘rocking’ movements of her chest and abdomen are still present.

On balance, it is agreed to proceed to CT scan of the head, cervical spine, chest and abdomen, with reconstructions of the thoracic and lumbar spine. Intubation and ventilation have been considered, but deferred in view of the improving physiological state.

The radiologist reports the findings of the scans immediately: a fracture-dislocation at the cervicothoracic junction; a small contusion in the right frontal lobe, but no intracranial haemorrhage or significant mass effect; a fractured fifth rib with no other evidence of thoracic injury; a penetrating injury to the abdomen; free intra-abdominal air and fluid and a small hepatic contusion. There is a tiny residual right pneumothorax on the CT scan.

Q. Now that the primary survey is done (together with early imaging), what needs to be done now?
A. A secondary assessment - including complete undressing of the patient and head-to-toe examination - is performed to look for signs of other injuries.

You will also consider the necessary additional investigations, which are likely to include arterial blood gas analysis, drug testing and a pregnancy test. More invasive monitoring, including a central venous catheter, is considered as it may provide important information. It is now also imperative that the appropriate specialists become actively involved. A relatively senior staff member should inform the family about the patient’s condition and collect any further, additional relevant information from them.

**NOTE** Strict aseptic technique must be followed to minimise subsequent infection risk.
Secondary assessment

PACT module on Clinical examination

PACT module on Haemodynamic monitoring and management

During the secondary survey, a fracture-dislocation of the left ankle and poor capillary refill in the foot are noted.

Q. As the ankle injury may cause acute ischaemia of the foot, what needs to be done next? Does the ankle fracture need definite repair now?
A. The trauma/orthopaedic surgeon will want to reduce the ankle fracture promptly and confirm satisfactory circulatory supply to the foot. Following this, the fracture will have a low priority and can be splinted with a temporary cast.

Q. Is checking for a compartment syndrome warranted?
A. Unlikely with an ankle fracture but nonetheless worthwhile.

Q. What should be done about the small residual pneumothorax?
A. The chest should be re-examined and the drain position and function reviewed. Oxygen saturation and blood gas profile rechecked. A small residual pneumothorax may well disappear, but a high index of suspicion will be maintained.

Q. If the pneumothorax worsens, what may need to be done?
A. The drain may need to be adjusted, suction applied or a further drain inserted.

Management of potentially incapacitating injuries (ankle)

The patient has now been in the emergency department for almost an hour. The GCS score has remained stable at 13 (eyes 3, verbal 4, motor 6). The pain in the patient’s neck seems to have diminished somewhat due to the judicious administration of small increments of intravenous fentanyl. The patient’s blood pressure is 91/65 mmHg after another litre of intravenous fluid. Oxygen saturation has remained stable at 97%, but the patient has become more tachypnoeic, with diaphragmatic breathing still evident. There is some oozing from the abdominal wound and significantly more bloodstaining.
of the dressings covering the abdomen. As you cross the hallway, the young woman’s boyfriend approaches you and wishes to know if she will survive. You check that he is the next of kin and whether there is any other family member present that should be included in the conversation.

NOTE When informing next of kin, a good way to start is to ask them what they have learned so far from police, bystanders or other medical personnel - such as the intern’s earlier interaction with them in this case. This will allow you to check if the family has understood previous information and to avoid giving redundant or possibly contradictory information.

Learning Issues

PACT module on Communication and on Ethics

Q. The young woman has an Injury Severity Score of 29, and using the TRISS methodology has a survival probability of approximately 80%. What do you tell him? Does the quoted probability of survival apply to this patient?

A. At this point, it is too early to make any specific prognosis. Although you know survival probability (for a cohort of victims with similar injuries), you know not extrapolate this to individual patients.

You tell the boyfriend that at this point you don't know whether she will survive, but you and the rest of the hospital team are doing everything possible to help her and will keep him informed. You tell him that she will likely need to go to the operating room for surgery and that there will be a need to talk to next of kin at that stage to provide information on what is planned and to achieve ‘consent’.

Q. Because the patient is obtunded, you take the opportunity of this conversation to enquire further into the patient’s background medical status and you utilise the structured ‘AMPLE’ approach. Please, outline its components.

A. A: allergies, M: medications, P: past history, L: last meal; E: events and environment.

Learning Issues

Risk of death using a variety of trauma scores

http://www.trauma.org/

When you return, the orthopaedic surgeon has reviewed the images and wants to take the patient to the operating room immediately for stabilisation of the spinal column. Although the surgeon has not had the benefit of an MRI scan, he is confident that there is likely cord transection and the need for surgery is primarily to stabilise the spine with a view to facilitating mobilisation and rehabilitation.
Q. What else needs to be done, and how do you set your priorities?

A. Even though stabilisation of the vertebral column is important, it is not (for this indication) an emergency and laparotomy and exploration of the abdominal cavity has a higher priority as (on the basis of the clinical information and CT scan) continuing blood loss or bowel perforation cannot be excluded with certainty.

Q. What complications might occur if the spinal operation were undertaken first?

A. Control of abdominal bleeding, if it restarts, would be impossible in a patient who is in the prone position for repair of the spinal column. Delay in repair of intra-abdominal injuries may lead to peritonitis and sepsis resulting in additional life-threatening conditions.

Q. What about further radiographic evaluation of the ankle fracture?

A. It can wait until after the laparotomy and stabilisation of the vertebral column.

NOTE Documentation can follow the same approach as diagnosis, utilising the ABCD of the primary survey (with corresponding acute vital threats) and then incorporating the secondary survey with the ‘AMPLE’ history.

Learning Issues

Damage control surgery


Q. What prophylactic treatments are considered at this stage?

A. Tetanus prophylaxis and prophylactic antibiotics. The latter should provide cover for abdominal organisms and skin flora.

Q. The anaesthesiologist says that the proposed procedure (laparotomy and stabilisation of the spinal column) is likely to last at least four hours and he is concerned about the effect of this on the (likely) head injury - an abnormal GCS score with a risk of diffuse axonal injury (although not be evident on the early CT scan). He points out that, while under anaesthesia, clinical assessment of brain function (as a monitor) will not be possible. What do you advise?
A. Even though the CT scan has shown no intracranial mass effect, the insertion of an intracranial pressure monitoring probe may be considered to monitor for increasing ICP as a sign of cerebral oedema. You advise consulting neurosurgery.

The neurosurgeon is sufficiently confident, on the basis of the CT scan and the improving trend in the GCS score, to omit this intervention.


Learning Issues

PACT module on Traumatic brain injury

At laparotomy, one litre of free intra-abdominal blood is found. The colon had been perforated by the fence post. A hemicolecctomy is performed and a stoma created from the transverse colon. The small hepatic contusion is confirmed and no further bleeding or injury is identified. Following laparotomy and surgical stabilisation of the spine, the patient is admitted to the ICU.

Q. The intensivist would normally like to extubate the patient early but has concerns that this may not be possible in this patient. What are the concerns?

A. A number of factors may slow the patient’s progress and ability to wean from ventilation particularly the high spinal cord injury (causing ventilatory impairment), the likelihood of pulmonary contusion (causing hypoxaemia) and perhaps impairment of consciousness due to brain injury (thus limiting airway control when attempting extubation).

NOTE Ensure that in a patient following head trauma that a careful evaluation of the neurological status is performed and documented before induction of general anaesthesia. In case of doubt do not hesitate to ask for a cerebral CT scan.

Learning Issues

Weaning from mechanical ventilation
Continued care in the ICU and beyond, host defence, metabolic control, discharge

PACT modules on Airway management and Mechanical Ventilation

Q. What place is there for inotropes or vasoconstrictors in the early management of this major trauma patient?

A. Hypotension in trauma victims is usually due to hypovolaemia and should be treated with adequate fluid and blood transfusion. Vasoconstrictors / Inotropes may be useful in reducing the degree of hypotension and hypoperfusion while normovolaemia is attained.
Q. If the blood pressure remains low despite achieving normovolaemia (due to neurogenic shock), is noradrenaline (norepinephrine) warranted in this patient?

A. In spinal cord injury, as in head injury, arterial hypotension is a risk factor for poor outcome. Administration of noradrenaline (norepinephrine) to restore normal blood pressure is indicated presuming euvolaemia and an adequate cardiac output has been achieved with fluid therapy.

See PACT module on Haemodynamic monitoring and management

Q. Regarding early extubation, what are the advantages and disadvantages?

A. Advantages: the conscious patient is her own best neurological monitor. If successful, a shorter and less complicated ICU (and hospital) stay can be anticipated.

Disadvantages: Prolonged intubation entails prolonged sedation which may delay neurological assessment. Delaying extubation also increases the risk of pneumonia, and sedation may increase the need for inotropic or vasopressor infusions.

Q. What are the disadvantages of premature extubation?

A. If extubation is performed when the patient’s respiratory drive, respiratory muscle power, chest wall mechanical function or pulmonary shunt are unfavourable, the patient may become hypoxic or hypercarbic and require re-intubation. Sputum retention and pneumonia may occur, due to respiratory muscle fatigue, poor cough and sputum retention.

Q. What assessment is required when the patient arrives in the ICU?

A. A further global examination (sometimes called a tertiary survey), repeating all the elements of the primary and secondary surveys, is required. Continuing efforts should be made to identify previously unrecognised injuries and to recognise deterioration in previously identified problems e.g. development of a compartment syndrome associated with the ankle fracture).

Learning Issues

‘Tertiary’ assessment

PACT module on Clinical examination

Q. What complications of the spinal injury may appear now?
A. Complications include paralytic ileus, autonomic hyper-reflexia, prolonged spinal shock (flaccid paralysis), loss of temperature control (loss of vasoconstriction when cold and absence of sweating when pyrexial) and pressure ulcers.

Q. List some other complications which might become evident in this patient now?
A. These might include fat embolism, ARDS, sepsis, acute renal failure and liver dysfunction.

Q. Do these and other complications have a cumulative adverse effect on the patient?
A. They increase the overall risk (to survival) and of morbidity - in their own right and by causing a likely delay in discharge from the ICU.

**Learning Issues**

PACT module on Sepsis and MODS

PACT module on Acute Kidney Injury - parts 1 and 2.
PACT module on Acute hepatic failure

Over the following days, the patient receives intensive physiotherapy and is weaned from ventilatory support after a period of non-invasive ventilation. Repeated vomiting (due to paralytic ileus) is a concern, but no signs of aspiration pneumonia appear. Her paralysis has not improved but early fixation has allowed her to embark on her rehabilitation programme quickly. The ankle fracture is operated on five days later, after achieving cardiorespiratory stability and discontinuing vasopressors. The patient is awake and cooperative, but remembers nothing about the accident. She is now awaiting transfer to the ward and to a rehabilitation unit for intensive physiotherapy.

Q. As you talk with the young woman before her transfer to the rehabilitation unit, she suggests that it might have been better had she died. How do you respond and inform her of her prospects and time course for her rehabilitation?
A. Your response to her must be truthful - optimistic if there is a reasonable chance of improvement and realistic if expectations are inappropriate. Severe polytrauma patients who have significant disability on discharge from the acute hospital setting often make remarkable improvement over the following months as they undergo intensive rehabilitation. The final result will often not be evident for 12 months or more, as many organ systems (e.g. lung function after ARDS or neurological function following head trauma) are slow to reach full recovery. Given her current situation, there is a good chance that over the coming year, this woman's quality of life will improve significantly.

See PACT modules on Ethics and on Communication.

On reflection, good outcome and quality of life depend on an efficient chain of rescue and continuing care. A protocolised approach to early management, as in the ATLS guidelines, facilitates efficient, coordinated and goal directed acute trauma care. The Intensive Care physician and team have a central role in the acute and ongoing Critical Care and are key to the teamwork that, with good rehabilitation, can achieve the optimum outcomes for very seriously injured patients.