Trauma accounts for more than 9000 deaths per annum in the UK. One-third are due to road traffic accidents and chest injuries are responsible for one in four of these deaths. They also play a significant part in 25% of other trauma deaths. Patients admitted with chest injuries often have associated multiple injuries. It is usual for many specialities to share the care of the multi-injured patient. Therefore, good communication is essential. Rapid access to tertiary care surgical services (e.g. cardiothoracic and neurosurgery) are required and access to critical care physicians may improve outcome.

Many patients with chest injuries die after admission to hospital and up to one-third of these may be preventable. In 1992, the major trauma outcome study report found that outcome from blunt trauma varied between hospitals in the UK and was worse than that in the US.

In 2002, a joint report from The Royal College of Surgeons of England and the British Orthopaedic Association produced recommendations aimed at improving outcome from severe trauma. Recommendations included establishing a national trauma research network, geographically-based trauma systems and the auditing of standards. This document recognised that many patients with chest injuries will be treated by non-cardiothoracic specialists. The occult nature of underlying injuries and difficulties in clinical examination may lead to under-diagnosis of potentially life-threatening injuries. Further recommendations included investigations targeted to the mode of injury, early specialist intervention and repeated clinical and radiological investigations.

Pathophysiology

Severe chest trauma results in tissue hypoxia. This may be due to hypovolaemia, pneumothorax, cardiac injury or areas of ventilation/perfusion mismatching in the injured lung. Hypercarbia develops secondary to: (i) associated upper airways obstruction; (ii) reduced respiratory excursion due to pain; and (iii) mechanical causes such as a flail segment. There may be a metabolic acidosis (hypovolaemia or hypoperfusion) or respiratory acidosis (hypoventilation).

Principles of management

The principles of management are as follows:
(i) A primary survey with resuscitation of vital functions, a full secondary survey and a plan for definitive management.
(ii) Hypoxia is the most serious effect of blunt chest trauma. It is important to maintain adequate oxygen delivery.
(iii) Treat rapidly immediate life-threatening injuries (haemorrhage, tension pneumothorax, pericardial tamponade).
(iv) Conduct a full secondary survey, guided by clinical suspicion and examination.
(v) Only 10–15% of patients with blunt thoracic trauma will require surgical intervention for the chest injury.

The primary and secondary survey

The primary survey focuses on the airway, breathing, circulation, disability and exposure (ABCDE) principle. This is conducted simultaneously with resuscitation of vital functions as set out in the Advanced Trauma Life Support Manual.

Life-threatening injuries found in the primary survey

Possible life-threatening injuries found in the primary survey are listed in Table 1. Patients with a seat-belt mark have a 4-fold increased incidence of severe chest trauma and an 8-fold increased incidence of intra-abdominal trauma.

Key points

Blunt chest trauma is a significant preventable cause of death in trauma patients.
It is associated with immediate and late life-threatening injuries.
Helical CT scanning and transoesophageal echocardiography are useful in detecting aortic injury. Aortography remains the gold standard.
Surgical intervention is required in only 10–15% of patients.
Airway obstruction

Airway obstruction is often associated with injuries to the head, face, cervical spine and airway. Meticulous attention to the protection of the spinal cord is vital, especially during manoeuvres to improve airway patency. Airway obstruction presents with noisy upper airway sounds, use of accessory muscles of respiration, hypoxia and hypercarbia. High-flow oxygen is administered. Treatment involves simple airway manoeuvres (such as chin lift or a jaw thrust), the use of airway conduits and formal intubation. A tracheostomy may be required.

Tension pneumothorax

This life-threatening condition presents with hypoxia, tachypnoea, decreased breath sounds with hyper-resonance on the affected side, tracheal deviation away from the affected side and elevated central venous pressure (or bulging neck veins). Clinical signs confirm the diagnosis and chest radiography should not delay treatment which is prompt needle decompression followed by the insertion of a chest drain.

Flail chest

A flail chest occurs when three or more ribs are fractured in two or more areas. Paradoxical chest movement with hypoxia and hypercarbia then occurs. A flail chest is always associated with pulmonary (and often cardiac) contusion. This exacerbates hypoxia. High flow oxygen, analgesia and measures which maintain lung volume and functional residual capacity are the main treatment options. Surgical fixation of ribs is occasionally performed, especially if the injury involves bilateral flail segments.

Epidural analgesia is safe and effective; it is associated with improved oxygenation, although it may not prevent the need for further respiratory support. Continuous positive airways pressure and bi-level positive airways pressure can be administered non-invasively using full-face or nasal masks. This may abolish the need for formal tracheal intubation and positive pressure ventilation if adequate clearance of secretions is also maintained. The patient with a flail chest is best managed in a critical care area.

Massive haemothorax

Massive haemothorax is often due to large vessel damage and presents with shock, hypoxia, absent breath sounds and dullness to percussion on the affected side. It is important to remember that collections of < 200 ml will not be visible on a chest X-ray. Supine chest films show diffuse radiodensity on the affected side. The patient will require volume expansion and chest drain insertion. Surgical intervention is required if the drain output exceeds 1500 ml initially or if drainage exceeds 150–200 ml h⁻¹. There may be a place for interventional radiology to arrest bleeding from isolated vessels.

Cardiac tamponade

The classical signs of cardiac tamponade are hypotension, rising central venous pressure and muffled heart sounds (Beck’s triad). A globular heart may be visible on X-ray. Treatment involves volume expansion and urgent pericardiocentesis, followed by thoracotomy.

Potentially life-threatening injuries found in the secondary survey

Traumatic aortic injury

Traumatic aortic injuries are the second most common cause of death after head injuries in road traffic accidents. These injuries are typically associated with high-speed deceleration. Recently, airbag inflation has also been associated with aortic rupture. Traumatic aortic injury is also strongly associated with multisystem injuries. For example, in a recent series, only 5% of patients were found to have an isolated chest injury. The clinician may be alerted to the possibility of a traumatic aortic injury by a palpable fracture of the sternum or scapula, a ‘steering wheel imprint’, an intrascapular murmur, lower limb paraplegia, decreased femoral pulses, a palpable fracture of the thoracic spine or a left sided flail chest with signs of shock.

Traumatic lesions are commonly located at the isthmus of the aorta (90–98%). There are several degrees of aortic injury, including intimal haemorrhage, intimal haematoma with laceration, medial laceration, complete laceration, false aneurysm and

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**Table 1** Injuries commonly associated with blunt chest trauma

<table>
<thead>
<tr>
<th>Immediate life-threatening injuries</th>
<th>Airway obstruction</th>
<th>Tension pneumothorax</th>
<th>Flail chest</th>
<th>Massive haemothorax</th>
<th>Cardiac tamponade</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potentially life-threatening injuries</td>
<td>Traumatic aortic injury</td>
<td>Pulmonary contusion</td>
<td>Myocardial contusion</td>
<td>Diaphragmatic rupture</td>
<td>Oesophageal rupture</td>
</tr>
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Traumatic lesions are commonly located at the isthmus of the aorta (90–98%). There are several degrees of aortic injury, including intimal haemorrhage, intimal haematoma with laceration, medial laceration, complete laceration, false aneurysm and
free rupture with peri-aortic haemorrhage. Blunt traumatic dissection of the descending aorta is associated with a high risk of death (a mortality figure of 85% has been quoted). Most of these patients die before hospital admission. Of the 15% who arrive at hospital alive, 50% will die within 48 h if left untreated.

Chest X-ray abnormalities usually draw attention to the aortic injury. Abnormalities include a widened mediastinum, abnormal aortic contour, a left-sided haemothorax, lowering of the left main bronchus, tracheal deviation to the right, a left pleural apical cap and right-sided paratracheal widening. Unfortunately, radiographic signs of haemomediastinum have a very poor predictive value for aortic disruption. Angiography is the most commonly performed investigation and is both highly sensitive and specific for traumatic aortic injury. Angiography therefore remains the ‘gold standard’ investigation in aortic injuries (Fig. 1).

Computerised tomography (CT) scanning is often used as a screening test in blunt chest trauma. Helical CT scanning has shown some promising results in confirming the diagnosis of aortic injury; sensitivities approaching 100% with a specificity of 83% have been reported. Transoesophageal echocardiography, in the hands of an experienced operator, has the benefit of prompt bed-side diagnosis.

Early diagnosis and surgical intervention with aortic repair (commonly with the aid of cardiopulmonary bypass) is imperative in assuring a good outcome. Craniotomy for an intracranial lesion or a laparotomy for active bleeding should be performed before aortic repair.

**Pulmonary contusion**

Pulmonary contusion is the most common potentially life-threatening injury in blunt thoracic trauma. Respiratory failure develops over time as the inflammatory process in the injured lung parenchyma develops. Initially, the mechanical parenchymal injury is followed by oedema and bleeding. Later, the inflammatory, hormonal and metabolic responses predominate. This process is compounded by pain and abnormal chest wall movements, which contribute to hypoxic respiratory failure.

Predictors of severe pulmonary contusion include radiological evidence of contusion on the admission chest film, the presence of three or more rib fractures, the presence of a chest drain and hypoxia at admission.

Pulmonary contusion is best managed by oxygen therapy, aided by mechanisms that will improve functional residual capacity such as CPAP or a mechanical ventilation mode with PEEP. Pain control is essential and meticulous attention to fluid balance is necessary. If invasive haemodynamic monitoring is required, devices that allow an estimation of filling volumes (such as those which measure intrathoracic blood volume/extravascular lung water or right-ventricular end-diastolic volumes) may yet prove to be beneficial. A recent ARDS Network paper examined the relationship between extravascular lung water and mortality in a cohort of 850 critically ill patients with PaO2/FiO2 ratios < 300 mmHg. A strong relationship was found between increasing extra-vascular lung water and mortality.

**Myocardial contusion**

Myocardial contusion occurs more commonly than suspected; its presentation may be subtle. A fracture of the sternum or a seat-belt mark should always alert the physician to possible underlying cardiac contusion. Symptoms are often masked by underlying chest wall and lung injuries. The diagnosis is confirmed by ECG abnormalities, raised troponin T, serial elevation of the CK-MB iso-enzyme concentrations or echocardiographic
abnormalities. ECG abnormalities include tachycardia, premature ventricular contractions, atrial fibrillation, bundle branch block and ST segment changes. The treatment is supportive and particular attention should be focused on the prevention of myocardial ischaemia and the management of pain.

**Diaphragmatic rupture**

Diaphragmatic rupture is more commonly diagnosed on the left as the diaphragm is closely associated with the liver on the right. Symptoms depend on the amount of intestinal contents herniating into the thoracic cavity. The clinician needs to be vigilant when confronted with a patient with air in the left hemithorax or a raised left hemidiaphragm. CT scan of the chest may confirm diaphragmatic rupture and surgical correction is usually indicated.

**Oesophageal injury**

This condition presents with severe chest pain, signs of air and fluid in the mediastinum and pleural cavity, and signs of shock secondary to mediastinitis. Gastrograffin swallow may confirm the diagnosis. Treatment consists of thoracotomy, repair of the defect and supportive intensive care management. Sepsis is a common consequence of oesophageal rupture.

**Tracheobronchial injury**

Tracheobronchial injury presents with a pneumothorax and persistent air leak. It is follows high-energy impacts and is thus often associated with other vital organ injury (e.g. aortic, hepatic). The diagnosis may be confirmed with flexible bronchoscopy. The mainstay of treatment involves isolating the damaged airway. This may be achieved initially by double-lumen endotracheal intubation with ventilation to the healthy lung only. This may also facilitate surgery if thoracotomy is indicated. The need for surgical reconstruction of the damaged airway is dependent on the size of the defect and associated respiratory status. Lobectomy, or even pneumonectomy, may be necessary.

**Further intensive care management**

Patients who receive ventilatory support for blunt thoracic trauma are usually severely injured. These patients are at risk from ventilator-associated pneumonia, persistent air leaks and infection of undrained pleural collections. Acute respiratory distress syndrome often complicates the injury and weaning from respiratory support may be prolonged. Specific lung protective strategies include low tidal ventilation (6–8 ml kg⁻¹) with higher positive end-expiratory pressure and high frequency oscillating ventilation. Meticulous attention to fluid balance and nutrition is important. Prolonged use of muscle relaxants to facilitate ventilation is less common nowadays as there is an association with polyneuropathies in critically ill patients.

**Key references**


See multiple choice questions 122 and 123.