Overview

Thoracic trauma leads to:

- respiratory and cardiovascular implications
- hypoxia
- hypovolaemia
- reduced cardiac output.

Untreated, it will be fatal.

Trauma in the United Kingdom annually results in 720,000 admissions and over 6 million attendances to emergency departments. There are over 17,000 trauma deaths, with nearly 25% of these directly attributable to thoracic injuries. Many of these patients will die at the scene of the injury, with patients reaching hospital already a self-selected group who have a good chance of survival with early appropriate management. Evidence suggests that a significant proportion of in-hospital deaths from thoracic trauma are preventable, with injuries either not being recognised or being inadequately treated.

Only 10–15% of patients with blunt trauma and 15–30% of those with penetrating trauma ultimately require surgery. The remainder can be treated successfully in the emergency department through the application of fundamental principles of initial trauma management as well as through direct interventions within the scope of practice of emergency physicians. These management principles are especially important as thoracic trauma impacts directly on the heart and lungs, the two organs most integral to the provision of oxygenation and perfusion.
Successful management of thoracic trauma depends ultimately on effective prioritisation of resuscitation through the ABC principles with rapid detection and treatment of life-threatening injuries.

Mechanisms and patterns of chest injury

Chest injuries can be broadly classified as penetrating or blunt, the latter encompassing direct blunt trauma as well as crush, acceleration or deceleration injuries and blast injuries. An understanding of the specific mechanisms involved in individual trauma patients is important as patterns of injury are produced with significant differences in pathophysiology and clinical course (Table 1). Life-threatening injuries without obvious external signs can be missed as attention is paid to more visible but clinically less serious injuries. Diagnosis therefore often depends on maintaining a high index of suspicion of specific injuries which are associated with the underlying mechanism of trauma and tailoring the clinical assessment and investigation to look for and exclude those injuries. Predictors of thoracic injury include head and abdominal injuries, evidence of major haemorrhage in the absence of abdominal swelling or major bony injury, wounds, bruising or seatbelt marks on the chest wall and any degree of respiratory distress.

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Table 1
Patterns of chest injury

Penetrating injuries frequently cause pneumothorax or haemothorax and blood loss with cardiac or vascular injuries may be catastrophic. Patients often deteriorate rapidly but with appropriate management, have the potential to improve rapidly. Surgical intervention is frequently necessary, but special investigations are less commonly required than in blunt trauma.

Blunt trauma typically causes organ damage by compression, acceleration or deceleration and shear forces. In contrast to penetrating trauma, the majority of blunt injuries are managed non-operatively, responding to emergency department interventions such as intubation and ventilation or intercostal drainage. Blunt injuries are typically more difficult to diagnose and additional investigations such as computed tomography (CT) scanning are frequently required. Injuries such as pulmonary contusion may only manifest some time after the initial trauma and anticipation of the potential to develop such problems may allow the emergency physician to tailor management in an effort to reduce the risk of avoidable complications.

Regardless of mechanism, the main consequences of thoracic trauma are the combined effects on both respiratory and cardiovascular function, leading to hypoxia, hypovolaemia and reduced cardiac output, which not only impact directly on the thoracic organs but compound the effects of injuries to other organ systems.

Hypoxia and impairment of gas exchange

Hypoxia is the most common pathophysiological manifestation of moderate to severe chest injury. It may be the direct result of impairment of gas exchange at a pulmonary level or occur at a tissue level through inadequate perfusion despite normal or near-normal pulmonary gas exchange.

Patients with lung injury may experience significant impairment of gas exchange as a result of diffuse interstitial and alveolar haemorrhage, as seen in pulmonary contusion, which is a significant cause of delayed morbidity and mortality associated with chest trauma. Experimental and clinical studies have shown that the condition is progressive. Initial haemorrhage and oedema are followed by interstitial fluid accumulation and decreased alveolar membrane diffusion. These changes produce relative hypoxaemia, increased pulmonary vascular resistance, decreased pulmonary vascular flow and reduced lung compliance.

In patients with impaired respiratory function because of pulmonary injury, the associated mediastinal shift secondary to haemothorax, pneumothorax or both may result in compression of the non-injured lung, further compromising ventilation. This ventilation–perfusion mismatch can lead to an intrapulmonary shunt of more than 30% that contributes significantly to hypoxaemia, especially in the period soon after injury. Later, this hypoxia-induced pulmonary vasoconstriction will divert the blood away from the non-ventilated alveoli, thus reducing the intrapulmonary shunt to about 5%.

Hypovolaemia

All mechanisms of chest injury have the potential to cause major haemorrhage, leading to profound hypovolaemia. It is most often seen with aortic transection, great vessel rupture or laceration, pulmonary hilar injury or penetrating cardiac injuries not producing tamponade. The resulting decrease in cardiac output compounds the effects of hypoxia at a tissue level through reduced perfusion and worsens the outcome for other injuries, both thoracic and elsewhere.
**Reduced cardiac output**

Reduction in cardiac output in the setting of thoracic trauma may result from poor diastolic filling due to hypovolaemia, or from the secondary effects of hypoxia and reduced coronary perfusion on myocardial function, or may be caused by direct cardiac injuries. Such injuries include myocardial contusion in blunt trauma, transection of coronary arteries, blunt or penetrating heart valve injury and cardiac tamponade.

Blunt myocardial injury can reduce cardiac contractility and compliance of the ventricles, resulting in a low cardiac output state. Injury to coronary arteries or smaller vessels, either through direct penetrating trauma or within contused areas, can cause tissue necrosis and infarction leading to heart failure or cardiogenic shock. This is exacerbated by the reduction in coronary perfusion that results from hypovolaemia and hypoxaemia due to multiorgan trauma, and which further compromises global myocardial function.

Traumatic injury to the heart valves may result in acute volume overload of the ventricles, and severe regurgitation may cause acute congestive cardiac failure and death. Acute insufficiency of any valve may go unnoticed in the presence of more obvious injuries, with the result that valve lesions are not often detected during the initial post-trauma survey and resuscitation phase. Their recognition requires a high index of suspicion and further investigation (such as echocardiography).

The ultimate goal of treatment of thoracic trauma is therefore to restore and maintain both oxygenation and tissue perfusion, aiming to restore ventilation to all viable lung tissue and optimise cardiac output, through rapidly identifying and treating those injuries which compromise respiratory and cardiac function.

**Primary survey and resuscitation**

The primary survey is a rapid, focused assessment following the ABC principles (see Chapter 1).

During the course of resuscitation, major life-threatening thoracic injuries will be uncovered during the primary survey (and immediately addressed) whilst other potentially serious thoracic problems will be identified in the secondary survey. The following adjuncts are crucial to the identification of thoracic injuries.

**Diagnostic adjuncts to the primary survey**

**The chest X-ray**

The plain anteroposterior (AP) chest radiograph done in the resuscitation area of the emergency department remains the most important standard initial image in chest trauma. It should be performed within 10 min of the patient's arrival in the emergency department. Clinical signs in thoracic trauma are often subtle or misleading and the chest radiograph is a valuable tool which helps to identify important problems which require intervention as well as guiding decision making on further investigations such as CT scanning.

In blunt trauma, with the risk of spinal injury, the X-rays are performed in the supine position and should be sufficiently penetrated to allow some visualisation of the thoracic spine as well as the outline of the aorta. In penetrating trauma, where possible, the chest X-ray is done with the patient sitting upright to increase its sensitivity in detecting small haemothoraces or pneumothoraces as well as diaphragmatic injuries. It requires 400–500 mL of blood or fluid to obliterate the costophrenic angle on the erect chest X-ray. Widening and shift of the mediastinum and rib fractures may be evident (Figures 1–5).
Figure 1
Widened mediastinum needing urgent further investigation.

Figure 2
Subtle right pneumothorax.
Figure 3
Multiple rib fractures with signs of haemothorax and surgical emphysema.

Figure 4
Right lung contusion on chest X-ray.
The arterial blood gas

Arterial blood gas analyses should be performed on all patients with significant thoracic trauma, in particular those patients who are intubated and ventilated. This will not only give specific measures of oxygen and carbon dioxide, but also indicate acid–base status via pH, base excess and bicarbonate. Many blood gas machines will also provide haemoglobin estimation as well as lactate measurement which is a useful indicator of tissue perfusion and oxygenation.

Focused assessment with sonography for trauma examination

Focused assessment with sonography for trauma (FAST) is a rapid ultrasound assessment tool used in haemodynamically unstable patients with blunt trauma. It is performed in the resuscitation area of the emergency department and, aside from its role in abdominal trauma, may be useful in identifying the presence of pericardial blood or haemothorax, though the latter may only be detectable when >200 mL. It may be similarly useful in certain cases of penetrating thoracic trauma. It requires an operator specifically trained in FAST.

Immediate life-threatening injuries

Six specific thoracic injuries identified on the primary survey may be rapidly fatal if not recognised and treated promptly (Box 1): airway obstruction or rupture (see Chapter 1), tension pneumothorax, open pneumothorax, flail chest, massive haemothorax and cardiac tamponade. During the primary survey, the signs that will aid in diagnosis should be actively sought and treatment should be initiated as the problems are identified (Table 2).

Life-threatening injuries: primary survey

- Airway obstruction or airway rupture.
- Tension pneumothorax.
- Open pneumothorax.
- Flail chest.
- Massive haemothorax.
- Cardiac tamponade.

**Tension pneumothorax**

A traumatic tension pneumothorax is the progressive build-up of air within the pleural space, caused by a one-way leak from lacerations to lung, airway or chest wall. Air enters the pleural space on inspiration but cannot escape during expiration due to the effective formation of a one-way flap valve. The result is progressive accumulation of air with initial collapse of the ipsilateral lung, causing hypoxia, followed by shift of the mediastinum to the opposite side, compressing the contralateral lung and decreasing venous return to the heart. The resulting combination of worsening hypoxia, impaired ventilation and reduced cardiac output leads to traumatic arrest unless the tension is decompressed.

Tension pneumothorax can complicate either blunt or penetrating trauma or be iatrogenically caused through the insertion of central venous lines or the incorrect application of occlusive dressings to penetrating chest wall wounds. It is most commonly seen in patients with a simple pneumothorax or visceral pleural injury who are put on positive pressure ventilation. Such patients may deteriorate rapidly after initiation of ventilation or may present insidiously hours later. Awareness of this potential for deterioration and early recognition and treatment are the key to management. It is a clinical diagnosis and delays in decompression while radiological evidence is obtained may have fatal consequences.

The clinical presentation of tension pneumothorax is that of progressive respiratory distress, chest pain and air hunger. The presentation may mimic airway obstruction as effective ventilation decreases. The classic signs are of tracheal deviation away from the side of the tension, hyperinflation, reduced chest movement with hyper-resonance to percussion and reduced breath sounds on the affected side, elevated jugular venous pressure, tachycardia and hypotension. Untreated, this process will progress to full circulatory collapse and cardiac arrest with pulseless electrical activity.

However, classic signs are often absent, with tachycardia and tachypnoea being the only overt signs. Percussion note and breath sounds are often difficult to hear in the noisy trauma environment, while elevation of venous pressure may not occur with hypovolaemia. In rare cases, tension pneumothorax may be bilateral.

Treatment is the immediate decompression of the affected side, converting tension pneumothorax into simple pneumothorax. This is achieved by the insertion of a large-calibre cannula, around 16 gauge, into the second intercostal space (just above the third rib) in the midclavicular line. The corresponding hiss of air confirms decompression and the cannula should be left open. The chest should be re-examined and an intercostal drain inserted on the same side without delay (see ‘Procedures associated with the primary survey’ below). Needle thoracostomies can easily become kinked or displaced, allowing the tension to recur. It is possible for drains to become blocked and the patient to re-tension, so any deterioration in the patient's condition should be followed by immediate reassessment. In the ventilated patient, open thoracostomy is an effective technique to treat tension and prevent recurrence. Formal drains can be inserted later.

Because of the potential for developing tension pneumothorax, all patients on positive pressure ventilation who have any evidence of pneumothoraces or have multiple rib fractures should be considered for intercostal drain insertion, particularly if they require transfer.

**Open pneumothorax**

An open pneumothorax is a pneumothorax which communicates with the exterior via a chest wall defect. The thickness of chest wall is much less than the length of the trachea with the result that any chest wall defect more than 75% of the size of the trachea provides less resistance to air flow. During inspiration, which generates negative intrathoracic pressure, air is preferentially sucked through the chest wall defect and into the pleural space, rather than down the trachea. This reduces the effectiveness of ventilation and compromises oxygenation. If a flap valve effect is created either by the chest wound itself or dressings applied to it, tension pneumothorax can result.
The diagnostic clinical signs are of a chest wound that appears to be sucking air on inspiration and bubbling on expiration. The patient is typically tachypnoeic with a shallow breathing pattern. Breath sounds on the affected side are reduced with resonance on percussion. However, other than the wound itself, signs may easily be overlooked.

Initial management consists of applying a sterile occlusive dressing taped down on three sides, providing a flutter valve effect which prevents air ingress during inspiration, but lifts on the untaped side during expiration, allowing egress of both air and blood. This may be difficult to apply on large wounds. A commercial dressing, the Asherman chest seal, has a one-way valve that achieves the same objective. Definitive management involves wound closure with insertion of an intercostal drain.

**Massive haemothorax**

Haemothorax is an accumulation of blood in the pleural space following blunt or penetrating trauma and is typically caused by rib fractures, lung parenchymal injuries or vascular injuries. The majority of haemothoraces are small and self-limiting, but following penetrating injuries to heart, great vessels and hilar structures or less commonly following blunt trauma, bleeding into the pleural space may be severe.

A massive haemothorax is defined as loss of more than 1500 mL of blood or more than one-third of blood volume into either the pleural space or out via an intercostal drain. Examination may reveal evidence of a penetrating wound or bruising to the chest wall with crepitus from rib fractures.

The cardinal signs of massive haemothorax are of hypovolaemic shock with dullness to percussion and reduced or absent breath sounds on the affected side. Neck veins are typically collapsed secondary to hypovolaemia, but may be distended if there is associated tension pneumothorax or cardiac tamponade. It is unusual for the haemothorax itself to shift the mediastinum sufficiently to cause visible distension of neck veins.

The key to management of massive haemothorax is the restoration of circulating blood volume together with drainage of the haemothorax via a wide-bore intercostal chest tube (28 French or larger) inserted in the fourth or fifth intercostal space just anterior to the midaxillary line. (See Procedures associated with the primary survey, below.) If decompression of the haemothorax takes place before venous access is obtained and volume resuscitation commenced, haemodynamic decompensation can rapidly occur, particularly if the source of bleeding is uncontrolled. Volume resuscitation is initially with crystalloid, but the need for blood should be recognised early and transfusion commenced with type-specific blood until fully cross-matched blood becomes available. If there is significant drainage of blood via an intercostal drain, autotransfusion can be considered.

During resuscitation, systemic blood pressure should not be allowed to rise uncontrollably through overvigorous fluid administration if cardiac or vascular injury is suspected as this may precipitate further bleeding (permissive hypotension). In addition, the initiation of positive pressure ventilation during resuscitation can also dramatically increase bleeding from pulmonary vessels through mechanical clot displacement. The potential for sudden haemodynamic deterioration following intubation and ventilation should therefore be anticipated and recognised by the emergency physician, though this does not change the indications for ventilation in trauma patients.

Thoracotomy in theatre is likely to be required if initial drainage of blood is more than 1500 mL or ongoing drainage exceeds 200 mL/h for 2 or more hours. Initial volume drained is typically less important than ongoing drainage and the need is based on the patient's clinical status as well as ongoing requirement for blood transfusion. Early cardiothoracic opinion should therefore be obtained.

**Flail chest**

Injuries to the chest wall are extremely common following blunt trauma and vary from minor bruising to severe bilateral crush injuries with multiple rib fractures which compromise ventilation. Rib fractures may be associated with pulmonary contusion and fractures of the first rib should raise the suspicion of underlying vascular injury.

When a segment of chest wall loses bony continuity with the thoracic cage, it becomes flail and will move paradoxically on respiration, reducing tidal volume and compromising ventilation. In addition, rib fractures may be accompanied by significant blood loss. The principal cause of hypoxia after flail chest is the development of severe pulmonary contusion.

Diagnosis is usually clinical, by observation of abnormal chest wall movement and the palpation of crepitus, although splinting of the chest wall may initially make it difficult to detect. The chest radiograph will not always reveal rib fractures or costochondral separation, although arterial blood gas analysis may reveal hypoxia and/or hypercapnia.

Treatment is initiated with high-flow oxygen and lung expansion must be restored by intermittent positive pressure ventilation if required and any haemopneumothoraces must be drained. Intercostal chest drains are almost always required, particularly if positive pressure ventilation is needed. The aim of further management is to preserve respiratory function.
Pain reduces the tidal volume and inadequate ventilation of the basal lung segments results in atelectasis. Pain also inhibits coughing, allowing secretions to obstruct bronchi and cause acute respiratory failure. Effective pain relief is required so that regular physiotherapy can be carried out with the patient’s full co-operation. Epidural anaesthesia may be extremely effective in the management of patients with flail chest.

Careful fluid management is essential because the injured lung, with its increased capillary leakage, is sensitive both to inadequate perfusion and also to fluid overload.

A flail segment in itself does not justify mechanical ventilation, although elective intubation and ventilation are often appropriate. The degree of respiratory distress and hypoxia determines the need for ventilation (Box 2) and it is important to be aware that pulmonary contusion may develop insidiously over hours to days. Functional, not physical, integrity of the ribcage is required and adequate analgesia and careful fluid management are essential. Operative stabilisation of fractures is rarely indicated.

**Box 2**

**Indications for ventilation in flail chest**

- Significant impedance to ventilation due to the flail segment.
- Large pulmonary contusion.
- Unco-operative patient, e.g. head injuries.
- Development of respiratory failure: hypoxia/hypercapnia.
- General anaesthesia for another indication.

Cardiac tamponade

Bleeding into the pericardium is usually the result of penetrating trauma to the heart or great vessels, although blunt trauma may also damage these structures. Many patients with lacerations to the heart will exsanguinate quickly but in cases that are not rapidly fatal, bleeding may be contained within the pericardium. In the acute setting, the pericardial space has a fixed volume and only a small amount of blood accumulating is sufficient to exert pressure that restricts cardiac filling and compromises cardiac output.

Many of the signs of cardiac tamponade overlap with tension pneumothorax and this should first be excluded. The classic Beck’s triad of elevated central venous pressure, hypotension and muffled heart sounds is not commonly seen in the emergency department. Neck veins may not be distended if there is significant hypovolaemia and heart sounds can be difficult to assess in a noisy environment. Other signs of tamponade include Kussmaul's sign of paradoxical elevation of central venous pressure during inspiration and exaggeration of the physiological phenomenon of pulsus paradoxus. Systolic blood pressure decreases during spontaneous (rather than assisted) inspiration and a systolic drop of >10 mmHg is considered abnormal. Both Kussmaul's sign and pulsus paradoxus may be difficult to detect in the trauma setting.

The diagnosis of tamponade should be suspected in any patient with penetrating injury and shock, or with blunt trauma and shock that does not respond to fluid resuscitation and in whom tension pneumothorax has been ruled out. X-rays are typically normal, although access to FAST may aid in the diagnosis.

Immediate pericardiocentesis is indicated in patients with suspected cardiac tamponade who have failed to respond to initial resuscitative measures. (See ‘Procedures associated with the primary survey’, below.) Removing as little as 10–20 mL of blood from the pericardial space can considerably improve the condition of the patient with cardiac tamponade. Although pericardiocentesis may be life saving in certain circumstances, it has also caused death through cardiac laceration and should be performed with caution. In 25% of cases the blood within the pericardium has clotted and aspiration will not be possible. A fine balance between internal and external cardiac pressures may prevent exsanguination in cardiac tamponade, and aspiration of blood from the pericardium may contribute to fatal haemorrhage by reducing the external cardiac pressure and allowing a rise in intracardiac pressures.

In the arrested patient, pericardiocentesis should not be allowed to delay immediate thoracotomy. Patients with a positive pericardiocentesis should always undergo formal surgical exploration in the operating theatre.
Procedures associated with the primary survey

Intercostal drain insertion

Drainage of typically air or blood from the pleural space via intercostal drain insertion is the most common intervention in thoracic trauma. Despite being a simple procedure within the skills of the emergency physician, it has a complication rate of up to 10%. Adherence to proper technique will prevent many of the serious complications. Indications for intercostal drain insertion are shown in Box 3.

**Box 3**

**Indications for intercostal drain insertion**

- Pneumothorax: simple, open or tension (decompress first).
- Haemothorax.
- Traumatic arrest: typically bilateral drain insertion.
- Rib fractures in patients requiring positive pressure ventilation (relative indication).

Drains are typically inserted after plain chest radiographs are taken, but in certain circumstances such as the arrested patient or patients who have had tension pneumothoraces decompressed, it may be appropriate to insert an intercostal drain without first obtaining a chest X-ray. The technique for inserting an intercostal drain is shown in Box 4. Acute and chronic complications of this procedure are shown in Boxes 5 and 6 respectively.

**Box 4**

**Inserting an intercostal drain: technique**

1. Approach is typically in the fourth or fifth intercostal space just anterior to the midaxillary line, over the rib below the chosen space. In practical terms, this is usually the highest space in the axilla that can be easily accessed.

2. The area is cleaned and draped appropriately.

3. In the conscious patient, 10–20 mL of 1% lidocaine should be infiltrated into the skin and periosteum as well as down to the pleura, with the needle passing above the rib to avoid the neurovascular bundle. Aspiration of air will confirm position.

4. The skin is incised down to the rib. The track is then established above the rib by blunt dissection down to and through the pleura. Trocars should NOT be used.

5. Once the pleura is punctured, a gloved finger is inserted into the pleural cavity and a finger sweep performed to clear pleura or clots and to prevent damage to lung and other organs when the drain is inserted.

6. A large-bore (>28) drain is then introduced along the track with a surgical forceps. It should slide easily into position through the track. It is essential that the drain is inserted far enough for all sideports to lie within the chest cavity, but not so far as to abut the mediastinum.

7. The tube is connected to an underwater drain system which should always be positioned below the level of the patient. Bubbling or swinging of the drain helps to confirm placement.

8. Once positioned, the drain should be secured with 0 gauge sutures. Purse-string sutures are no longer advised.
9. The chest must be re-examined and position and efficacy of the drain confirmed on X-ray.

10. The tube should not be clamped after insertion.

Acute complications (usually related to poor technique)

- Laceration of intercostal vessels (cause—approach under the rib instead of over).
- Penetration of diaphragm or abdominal cavity (cause—drain placed too low).
- Laceration of lung (cause—pleural adhesions not cleared).
- Injury to stomach or bowel (cause—failure to identify diaphragmatic hernia).
- Subcutaneous tube placement.
- Tube in too far, causing pain.
- Tube displacement (cause—not adequately secured).

Chronic complications

- Infection or empyema.
- Clotted haemothorax.
- Blocked tube (usually due to clot).

Pericardiocentesis

Pericardiocentesis is indicated in suspected or confirmed cardiac tamponade. The diagnosis should be suspected in patients with penetrating injury and shock, or with blunt trauma and shock that does not respond to fluid resuscitation. The aspiration of as little as 10 mL of blood may improve blood pressure. The technique for pericardiocentesis is described in Box 7. The complications of this technique are shown in Box 8.

Pericardiocentesis: technique

1. Patient's vital signs and ECG must be monitored throughout and following the procedure.

2. The skin is prepared and landmarks identified.
3. In conscious patients, the puncture site should be anaesthetised with 1% lidocaine.

4. A 16–18 gauge over-the-needle catheter at least 15 cm in length attached to a 50 mL syringe is required. Consider a three-way tap on the syringe.

5. The skin is punctured 1–2 cm inferior to the left xiphochondral junction with the needle at 45° to the skin, aimed at the tip of the left scapula.

6. As the needle is advanced, aspirate until the syringe fills with blood, while observing the monitor for ECG changes. Aspirate as much blood as possible.

7. Electrocardiogram changes such as widening/enlarging of the QRS complex, marked ST changes or frequent ectopics suggest that the needle is too far advanced and must be withdrawn until the changes resolve. ECG changes may also occur during aspiration as the pericardial sac is drained. Again, slight withdrawal of the needle is indicated.

8. After aspiration is complete, remove the needle, leaving the catheter in place, attached to a three-way tap.

9. Secure the catheter in place.

10. Aspiration via the catheter can be repeated if the signs of tamponade recur.

11. Surgical exploration is usually required following positive pericardiocentesis.

Box 8

Complications of pericardiocentesis

- Aspiration of ventricular blood.
- Laceration of myocardium.
- Coronary vessel injury: may cause new haemopericardium and tamponade.
- Arrhythmias, typically ventricular fibrillation.
- Pneumothorax.
- Infection.

Emergency department thoracotomy

This remains a controversial topic and is a procedure that carries a high mortality. By no means can indiscriminate emergency room thoracotomy by the untrained be advocated, in particular when the potential exists for the procedure to be done by an experienced surgeon in the more controlled environment of an operating theatre. However, knowledge of the indications and principles of thoracotomy is important for any doctor dealing with major trauma and in a select group of patients, it can be a life-saving procedure. The exact criteria
defining that selection remain controversial. With a recent increase in the number of patients presenting to many emergency departments with penetrating chest trauma, in particular stab wounds, this knowledge may become increasingly relevant.

Overall survival rates for patients undergoing emergency department (ED) thoracotomy are 4–33% across numerous studies, with mechanism and location of injury as well as the presence or absence of vital signs being the most important determinants of survival. Stab wounds have a better prognosis than gunshot wounds and survival may approach 70% in isolated stab wounds with cardiac tamponade. Outcome in blunt trauma is dismal, with studies showing survival rates of 0–2.5%. However, the patient with blunt trauma who is exsanguinating via an intercostal drain due to a ruptured pulmonary vessel may be salvageable with thoracotomy and immediate cross-clamping of the bleeding vessel. Survival rates in both blunt and penetrating trauma patients who have not exhibited signs of life at any point are extremely poor.

Box 9 shows the indications for ED thoracotomy. Contraindications are shown in Box 10.

Box 9

**Indications for ED thoracotomy**

* Penetrating thoracic injury
  * Traumatic arrest with previously witnessed cardiac activity (prehospital or in-hospital).
  * Hypotension unresponsive to treatment (BP <70 mmHg).
  * Rapid exsanguination from intercostal drain or into airways.

* Blunt thoracic injury
  * Unresponsive hypotension (BP <70 mmHg).
  * Rapid exsanguination from intercostal drain or into airways.

Box 10

**Contraindications to ED thoracotomy**

* Blunt thoracic injuries with no witnessed cardiac activity.

* Multiple blunt trauma.

Box 11

**Aims of ED thoracotomy**

* Relief of cardiac tamponade.

* Control of haemorrhage.
Access for internal cardiac massage.

A left lateral thoracotomy approach can also occasionally be used to provide access for aortic cross-clamping in uncontrolled exsanguinating haemorrhage distal to the thoracic aorta. Following initial stabilisation, the patient should be immediately transferred to an operating theatre for definitive management.

Whatever the indication, if the opportunity exists, thoracotomy should always take place in an operating theatre in the hands of an experienced trauma surgeon. Survival rates are hugely improved when thoracotomy takes place in the theatre environment.

Secondary survey

Once immediately life-threatening conditions have been excluded or diagnosed and treated, the patient can be more thoroughly assessed. This assessment includes a detailed history, full examination, an upright chest radiograph if clinically possible, measurement of arterial blood gases, ECG and other diagnostic tests relevant to the clinical setting.

Several potentially life-threatening injuries may not be identified on the primary survey and require more detailed examination and investigation to diagnose (Box 12). A high index of suspicion based on the mechanism of injury and knowledge of common injuries associated with certain mechanisms (see Table 1) may aid considerably in reaching a diagnosis. Identification of certain injuries should prompt a thorough search for other associated injuries, for example great vessel injuries associated with first rib or scapular fractures. Missed injuries contribute to significantly increased mortality in the post-traumatic period. As in the primary survey, a structured, organised approach is required.

**Box 12**

Potentially life-threatening injuries identified on the secondary survey

- Tracheobronchial injury.
- Simple pneumothorax.
- Haemothorax.
- Pulmonary contusion.
- Blunt myocardial injury.
- Aortic disruption.
- Diaphragmatic injury.
- Oesophageal injury.

Tracheobronchial injury

Although uncommon, injuries to the trachea and main bronchi, caused by either blunt or penetrating trauma, may be fatal if not recognised and extensive free air in the neck, chest wall or mediastinum should always raise suspicion of damage to major airways.

Laryngeal fractures are rare and are indicated by hoarseness, subcutaneous emphysema and palpable fracture crepitus. If the airway is threatened or completely obstructed or if there is severe respiratory distress, intubation is warranted. This may be extremely difficult to achieve due to distortion of anatomy from the injury itself, subcutaneous emphysema, local haematoma or associated oropharyngeal injuries. Fibre-optic laryngoscopes or even immediate tracheotomy may be required to secure the airway, followed by surgical repair.
Penetrating injuries to trachea or bronchi are usually apparent and all require surgical repair. They may be associated with injury of adjacent structures, most commonly the oesophagus, carotid artery or jugular vein. With missile injuries, there can be extensive tissue damage to the surrounding area because of the cavitation caused by the velocity of the projectile. Transections of the trachea or bronchi proximal to the pleural reflection cause extensive deep cervical or mediastinal emphysema, which rapidly spreads to the subcutaneous tissues. Injuries distal to the pleural sheath result in pneumothoraces.

Blunt tracheal injuries may not be obvious, particularly if the patient has a depressed level of consciousness. Laboured breathing may be the only initial sign of airway obstruction. The diagnosis can be confirmed with bronchoscopy, preferably rigid, and this may be used to improve airway clearance of blood and debris. Early surgical repair is required.

The majority of blunt bronchial injuries occur within 2.5 cm of the carina. There are often severe associated injuries and most victims die at the site of the accident. For those who reach hospital, mortality is at least 30%. Signs of bronchial injury may include haemoptysis, subcutaneous emphysema, tension pneumothorax and pneumothorax with a large, persisting air leak. The diagnosis is confirmed by bronchoscopy although mucosal oedema and debris can obscure the extent of a bronchial injury, so the site should be carefully inspected. Distortion of airway anatomy by adjacent haematoma makes management more difficult, and surgery is occasionally indicated. Bronchial tears must be repaired urgently in the operating theatre via a thoracotomy.

Simple pneumothorax

A simple pneumothorax is a non-expanding collection of air in the pleural space. Classic signs of resonance to percussion and reduced breath sounds may be difficult to detect and diagnosis may only be made on chest X-ray evidence or even CT scan. Chest X-ray signs may be subtle, with increased radiolucency on one side the only evidence. In blunt trauma, the presence of rib fractures or haemothorax should raise a strong suspicion of associated pneumothorax. The diagnosis is especially important if the patient requires positive pressure ventilation or needs transfer to another hospital, as simple pneumothorax may rapidly tension.

In the trauma setting, all but trivial pneumothoraces detected on CT scan generally require intercostal drain insertion and even in the latter situation, the decision is usually influenced by the need for transfer, ventilation or general anaesthesia.

Haemothorax

Most small or moderate haemothoraces are not detectable by physical examination and will be identified on chest C-ray, CT scan or FAST. Small haemothoraces may be difficult to appreciate on chest X-ray in the supine patient and any increase in overall radio-opacity on one side of the chest, especially if associated with rib fractures or penetrating trauma, should raise the suspicion of a haemothorax. Treatment is intercostal drain insertion on the affected side.

Failure to drain haemothoraces is usually the result of failure to diagnose them. If missed, haemothoraces may become infected, leading to empyema, or may organise and fibrose, leading to permanent loss of lung volume which may impair pulmonary function.

Pulmonary contusion

Pulmonary contusion is an injury to lung parenchyma associated with blunt trauma which leads to blood and oedema accumulating in the alveoli. The resulting loss of normal architecture causes impairment of gas exchange, increased pulmonary vascular resistance and decreased lung compliance. There is associated atelectasis with shunting of blood and increased airway resistance all contributing to hypoxia. It develops insidiously over 24 h and is usually seen in the presence of signs of blunt chest wall trauma with bruising or underlying rib fractures, complicating the clinical picture of the associated injuries. In children, severe pulmonary contusion frequently occurs without rib fractures. Around 50% of patients with significant pulmonary contusions will go on to develop acute respiratory distress syndrome (ARDS).

Pulmonary contusion is seldom diagnosed on clinical examination, but the mechanism of injury and identification of injuries associated with contusion should raise the index of suspicion. Occasionally crackles may be heard on auscultation, but initial diagnosis is frequently on chest X-ray with the presence of nodular exudates in the lung fields. X-ray appearance typically lags behind the clinical picture which is of increasing respiratory distress and hypoxia. CT scan is a sensitive investigation which helps to distinguish contusion from atelectasis or other pathology.

Treatment is supportive while the contusion resolves and many patients can be managed without the need for intubation and ventilation. However, as the condition is progressive, at-risk patients should be closely monitored over the first 24–48 h. Fluid balance must be carefully monitored as both over- and underhydration contribute to secondary lung injury. Certain patients will, however, benefit from early intubation and ventilation (Box 13). The majority of patients, in the absence of complications, will resolve within 3–5 days. Complications include ARDS and pneumonia.

Box 13
Relative indications for early endotracheal intubation in pulmonary contusion

- Hypoxia or worsening respiratory status.
- Decreased level of consciousness.
- Pre-existing chronic pulmonary disease.
- General anaesthetic required for other injuries.
- Extremes of age.
- Organ failure in another organ system.
- Necessity for transfer.

Blunt myocardial injury

Blunt myocardial injury or myocardial contusion is the most commonly undiagnosed fatal thoracic injury. It occurs when there is direct compression of the heart due to blunt trauma or rapid deceleration. Blunt cardiac injury may also lead to chamber or valvular rupture. Blunt myocardial injury is often associated with sternal fractures and in this setting, the right ventricle is most frequently damaged. Patients with blunt myocardial injury are prone to all the complications associated with myocardial infarction and may complain of chest pain which is either overlooked or attributed to the overlying chest wall injuries. Around 20% of patients experience arrhythmias, which include sinus tachycardia, supraventricular tachycardia, atrial fibrillation, ventricular ectopics, ventricular tachycardia and ventricular fibrillation. Conduction defects, ranging from bundle branch block to complete heart block, may also develop.

The diagnosis is established from the mechanism of injury, serial cardiac enzyme measurements, ECG changes such as ST elevation, two-dimensional echocardiographic evidence of ventricular wall dysfunction and pericardial effusion. A significant rise in cardiac troponin following blunt chest trauma is highly suggestive of blunt myocardial injury, but does not predict the risk of complications. Elevation of jugular venous pressure following blunt trauma may indicate right ventricular dysfunction. The presence of a completely normal ECG makes the diagnosis of significant blunt myocardial injury unlikely.

Areas of contused myocardium behave in the same way as areas of infarction, and the patient should be treated accordingly. Conduction defects may rarely require pacemaker insertion. Cardiogenic shock is rare but some patients may need temporary intra-aortic counterpulsation balloon pumping to improve the perfusion of viable myocardium. Urgent surgical repair is sometimes necessary for cardiac rupture (particularly posterior), ventricular septal rupture or valve damage.

Aortic disruption

Tears of the aorta or pulmonary arteries are associated with blunt or deceleration injuries such as road traffic accidents and falls from a height. The aorta may be completely or partially transected or may have a spiral tear. Anatomically, the aorta is firmly fixed at three points: the aortic valve, the ligamentum arteriosum and the hiatus of the diaphragm. Sudden deceleration will allow the mobile parts of the aorta to move relative to the fixed parts, creating substantial shear forces. The most common site of rupture is at the attachment of the ligamentum arteriosum, where the aorta remains tethered to the main pulmonary artery.

Aortic rupture is immediately fatal in about 90% of cases and accounts for around 15% of immediate trauma deaths due to road traffic collisions. The immediate survival of the patient depends on the development of a contained haematoma, maintained by the intact adventitia. The survival of patients after reaching hospital depends on early diagnosis followed by urgent surgical repair.

Specific signs and symptoms of aortic injury are usually absent and an index of suspicion based on mechanism of injury should be maintained. Any suspicion of aortic injury raised by the plain chest radiograph (Box 14) must prompt further investigation. No single radiographic sign absolutely predicts aortic rupture, but a widened mediastinum >8 cm at the level of the aortic arch is the most consistent finding. In 1–2% of cases with aortic injury, the chest X-ray may be completely normal. Contrast-enhanced spiral CT scan is becoming an
investigation of choice for suspected aortic injuries, with sensitivity and specificity approaching 100%, similar to aortography. The difficulty for the clinician is that in order to obtain such investigations, patients require transfer away from the emergency department to the less safe environment of a radiology department. Instability of the patient due to other injuries or to the aortic injury itself may make this extremely difficult. Even if identified, aortic injuries that are not the cause of haemodynamic instability may be low on the list of management priorities in the multiply injured patient.

The treatment of traumatic aortic rupture is surgical repair, directly or by resection of the damaged segment and interposition of a vascular graft. All clinical situations are different, and priorities and management need to be tailored to the individual. Prior to surgery, a cautious approach to fluid resuscitation must be maintained and overvigorous volume replacement avoided at all costs (permissive hypotension). Elevations of systemic blood pressure may rupture the flimsy adventitial layer containing the rupture, causing fatal haemorrhage. Each individual situation needs to be considered on its merits and in the light of other injuries that require treatment.

Box 14

Radiographic features suggestive of aortic disruption

- Widened mediastinum >8 cm.
- Loss of the aortic ‘knuckle’.
- Tracheal deviation to the right.
- Oesophageal deviation to the right (NGT).
- Filling of the aortopulmonary window.
- Widened paratracheal stripe.
- Pleural cap.
- Elevation of right main bronchus.
- Depression of left main bronchus.

Diaphragmatic injury

Penetrating injuries can cause small diaphragmatic perforations that are rarely of immediate significance, but may present months or years later. By contrast, blunt trauma produces large radial tears of the diaphragm and herniation of abdominal viscera into the chest. The right hemidiaphragm is relatively protected by the liver and left-sided ruptures are therefore more common and are more easily diagnosed because of the appearance of bowel or stomach in the chest. Bilateral rupture is rare.

The chest radiograph can be misinterpreted as showing a raised hemidiaphragm, acute gastric dilation or a loculated pneumothorax. Contrast radiography or locating an abnormal position of the stomach with plain radiography by passing and identifying the tip of a nasogastric tube confirms the diagnosis. Unless other injuries require immediate surgery, repair of the diaphragm should not be delayed. This is often performed through a laparotomy for associated abdominal injuries.

One of the main risks of diaphragmatic rupture in the acute trauma setting is the insertion of an intercostal drain into stomach or bowel. Clinical examination and correct technique should prevent this.

Oesophageal injury
Damage to the oesophagus is usually caused by penetrating trauma. The proximity of the oesophagus to major vessels and other mediastinal structures frequently means that the consequences of damage to these are a more immediate focus for the emergency physician than the oesophageal injury which may be overlooked. Blunt oesophageal injury is rare and typically associated with blunt trauma to the upper abdomen which forces gastric contents rapidly into the oesophagus, causing a linear tear with leakage of contents into the mediastinum or pleural space leading to mediastinitis or empyema. The resultant clinical picture is identical to postemetic oesophageal rupture (Boerhaave syndrome) and may have fatal consequences if not recognised (Box 15).

The diagnosis should be considered in any patient with severe blunt trauma to the abdomen who has a left pneumothorax or haemothorax in the absence of rib fractures. The presence of stomach contents in material draining from a correctly inserted intercostal tube is virtually diagnostic. The diagnosis is confirmed by cautious contrast study of the oesophagus or by endoscopy. Treatment is by formal surgical repair in the operating theatre, with drainage of the pleural space or mediastinum or both.

Box 15

Features suggestive of oesophageal rupture

- Blunt trauma to upper abdomen.
- Sharp, severe epigastric pain.
- Shock disproportionate to the apparent injury.
- Pneumomediastinum on chest X-ray.
- Left pneumothorax or haemothorax without rib fractures.
- Gastric contents in chest drain.

Diagnostic adjuncts to the secondary survey

Chest X-ray

Chest X-ray is typically performed as an adjunct to the primary survey, but is repeated after the insertion of endotracheal tubes, central lines and intercostal drains. It remains the default initial investigation in all forms of chest trauma.

Computed tomography scan

In the setting of thoracic trauma and indeed trauma in general, the multidetector spiral CT scan is being increasingly used as a screening adjunct to the secondary survey, providing vital information on injuries that might otherwise be overlooked and guiding management decisions and prioritisation. It has the advantage of being fast, relatively non-invasive (apart from the addition of contrast in certain settings) and yet having very high sensitivity and specificity for the majority of important intrathoracic injuries. It is increasingly replacing investigations such as aortography and may identify subtle injuries that would be overlooked on other imaging modalities. However, it must be stressed that CT scan is not a substitute for clinical assessment during the primary survey and key life-threatening injuries must be identified and treated in the emergency department. CT scanning requires the patient to be moved from the well-equipped environment of the emergency department and the potential benefits and timing of scanning must be weighed against the potential risks of transfer. It is telling that the phrase ‘ring of death’ has been used to describe the CT scanner. Patient safety is paramount.

Analgesia

Analgesia is an important part of the management of patients with thoracic trauma and is the mainstay of therapy for injuries such as rib fractures. Lack of adequate analgesia may even produce acid–base disturbances through rapid shallow breathing due to pain. In the acute setting of trauma, the analgesic of choice is usually administration of opiates via the intravenous route, maintaining caution regarding the potential for respiratory depression. Administration of small doses repeated as necessary is safer than large boluses. Oral and intramuscular analgesia are generally inappropriate in the acute setting of major trauma, particularly if surgery is anticipated.
Specific analgesic plans can be tailored to the individual situation. Patient-controlled opioid analgesia (PCA) may be very effective in the awake, co-operative patient recovering from trauma. For multiple rib fractures or flail chest not requiring ventilation, continuous epidural anaesthesia is extremely effective, providing complete pain relief and allowing normal inspiration, without the risks of respiratory depression associated with opiates. Intercostal nerve blocks with a long-acting local anaesthetic such as bupivacaine are not as effective as epidural anaesthesia, but may provide relief in isolated rib fractures. Local anaesthetic injected into intercostal drains in sufficient quantities to provide pain relief may lead to toxicity.

Each case should be considered on its individual merits, but the requirement for adequate analgesia in trauma management should not be overlooked.

Summary

Thoracic trauma is common in the patient with multiple injuries and continues to be associated with a high mortality. The application of evidence-based management principles with a structured approach to assessment and management has resulted in improved outcome for a large group of patients who reach the hospital with intact vital signs.