Chest Trauma: Diagnosis and Management of serious injuries.

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Introduction

“Chest trauma will continue to be a significant cause of acute respiratory distress. It is a major source of morbidity and mortality in otherwise healthy young people. Airway control, oxygenation, drainage of pneumothoraces and hemothoraces and fluid resuscitation are the cornerstones of therapy. With these basic maneuvers, most chest trauma patients can be treated successfully.” Carrero, R. and M. Wayne (1989) [1].

These words, written over 20 years ago by Carrero and Wayne bring a grounding sense of calm when confronting the grievously injured chest trauma patient. The combination of clinical foreknowledge, ability to spot changing clinical signs, and even-tempered surgical courage to perform simple but lifesaving procedures can bring about a profound difference in outcome for the chest injured patient - even in resource limited settings.

In this chapter we shall review the basic physiology of breathing, the life-threatening pathophysiology of chest trauma, those conditions that require immediate management during the primary survey, and those conditions managed following secondary survey. Comment will also be made on chest-injury associated conditions the clinician ought to bear in mind.

Physiology of Breathing

At its simplest the respiratory system is composed of three main components:

- a membrane across which gas exchange takes place,
- a bellows mechanism to move gases to and from the membrane,
- and a control mechanism that drives the process while monitoring and making adjustments.

We will consider each component in turn. Trauma patients may suffer compromise at all three levels.
**The control mechanism**

Breathing is automatic and subconscious with a conscious over-ride control mechanism. The automatic control center (central pattern generator) is located in the medulla oblongata of the brain stem and is the primary source of automatic respiratory rhythm determining rate and depth of breathing. At rest the pacemaker cells discharge every 5 seconds giving a resting breathing rhythm of approximately 12 breaths per minute. The brain stem automatic control center has inspiratory and expiratory neurons and receives input from chemoreceptors (CO$_2$, O$_2$, pH etc), mechanoreceptors (in the ventilatory tissues), nociceptors, pathways that integrate breathing with other physiologic processes (such as swallowing), and from higher centers with behavioral and volitional activity (e.g. speech and breath-holding). Although the higher centers have neurons providing input to the automatic control center they are not dependent upon the automatic control center for their effect on breathing. Sensory receptor information from within the lungs is conveyed to the brain in the vagus nerve. The pacemaker cells have, amongst other receptors, opioid mu-receptors on their surface.

The effective neurologic output of the control mechanism is via the peripheral nerves to:

- the muscles of ventilation
  - diaphragm
  - intercostals
- accessory muscles of ventilation:
  - those affecting patency of the airways:
    - oro-facial
    - larynx
    - pharynx
  - and those attaching upon and affecting the chest wall:
    - sternocleidomastoid
    - pectorals
    - abdominal wall muscles, etc.

**The bellows mechanism**

The bellows mechanism facilitating gas flow is composed of:

- the neurological output from the central control mechanism (peripheral nerves),
- muscles of respiration,
- chest wall bones (e.g. rib cage, sternum, etc) and soft tissues (e.g pleura), and
- conducting airways.

The thoracic vertebrae, rib cage, costal cartilage, and sternum compose the bony thorax. When acted upon by the muscles of ventilation under the neurologic control of the central control mechanism, the ribs move up-and-out in a ‘bucket-handle’ manner increasing the intra-thoracic volume. With increased ventilatory requirement the intra-thoracic volume can be increased further by postural changes to straighten out the thoracic kyphosis in time with inspiration.

The co-ordinated movement of the bellows mechanism must move approximately 5-6L of gas (room air)/min for the average healthy adult at rest in an energy efficient way. At rest the diaphragm (innervated by the phrenic nerve with nerve roots C3,4,5) is the principal
muscle of respiration (accounting for ~75% of the change in intra-thoracic volume) with additional input from the external intercostal muscles. Chest wall movement produces negative intra-thoracic pressure relative to atmospheric pressure and facilitate gas entry to the lungs via the conducting airways. Expiration, at rest, is facilitated by the elastic recoil mechanism of the lung substance and is passive. For ventilation requirements of up to 30-40 litres/min increasing the tidal volume (deepening the breath, increasing the cyclical intra-thoracic volume) is usually sufficient in a healthy adult, however, higher ventilatory requirements necessitate increasing the rate of breathing and then recruitment of accessory muscles. At rest approximately 500ml of gas are moved per breath giving a pulmonary ventilation (or respiratory minute volume of ) 6 litre with 12 breaths per minute. The maximal voluntary ventilation is 125-170litre/min.

There are approximately 23 branching points within the airways from the trachea down to the alveolar sacs via the terminal bronchioles. The first 16 or 17 divisions constitute the conducting zone and are the tubes through which gases flow but in which no gas exchange takes place. Their volume constitutes ‘dead space’ and is ~150ml. The first 6 branch points of the conducting airways account for the majority of the total resistance to airflow in the lung since velocity in peripheral airways is slow. The early conducting passages are lined by cartilage for support but the terminal bronchioles are <1mm in diameter and are supported by lung substance rather than cartilage in their walls. bronchioles are thus susceptible to collapse if pressure in the pleural space is greater than in the airway. The distal conducting passages contain proportionately more smooth muscle content and have autonomic innervation and receptors for circulating catecholamines. Activation of local acetyl choline receptors leads to bronchial smooth muscle constriction and stimulation of gland secretion. Activation of beta\textsubscript{2} receptors leads to smooth muscle relaxation and bronchial gland stimulation but bronchial gland stimulation is inhibited by alpha\textsubscript{1} receptor activation.

**The gas exchange membrane**

The gas exchange membrane is effectively the alveolar walls of the respiratory bronchioles, alveolar ducts and (predominantly) the terminal alveoli (respiratory zone of the lungs). There are approximately 300 million alveoli in the human lung. Each alveolar wall is composed of alveolar epithelium (type 1 pneumocytes) and pulmonary capillary endothelium with a diffusion distance for oxygen of <0.5 μm. The total surface area available for gas exchange in a healthy adult is 50-100m\textsuperscript{2} (70m\textsuperscript{2} for practical considerations). The alveolus size is relatively large compared to the 10 micrometer diameter size of the pulmonary capillaries; this allows over 1000 capillaries to come into contact with an alveolus. The healthy gas exchange membrane is thin at 0.5 micrometers minimizing the distance oxygen must diffuse from alveolar lumen to haemoglobin in the 7micrometer sized red blood cells squeezing through the pulmonary capillary bed. In a healthy individual the haemoglobin is fully saturated by the time it is ~25% along a pulmonary capillary.

There therefore is tremendous reserve capacity within the ventilatory system to meet increased metabolic demand for oxygen. The chest injured patient, however, not only has increased demands but has impaired ventilatory function exacerbating their condition. Relevant pathophysiology will be discussed with each particular type of injury.
Anatomic considerations

In addition to the functional relationships described above there are some key anatomic points to bear in mind when managing the chest injured patient.

Thoracic Inlet

The thoracic inlet is an oblique plane bounded by the first thoracic vertebra, the first ribs and the manubrium sterni. The oesophagus posteriorly and trachea lie in the midline between the body of T1 and the jugular notch of the manubrium. The dome of each lung rises to the inlet on either side of these structures and each is indented by the first rib. The anterior scalene muscles descend to attach to the scalene tubercule of the first rib with the subclavian arteries arching over the first ribs behind the anterior scalene muscles while the subclavian veins pass anterior to the anterior scalene muscles. The phrenic nerves descend in front of anterior scalene muscles and anterior to the dome of the pleura with the internal mammary arteries before accompanying the pericardiophrenic vessels into the superior then middle mediastinum. The vagus nerves descend next to the carotid arteries and behind the jugular veins giving off the right recurrent laryngeal nerve under the brachiocephalic trunk and eventually the left recurrent laryngeal nerve under the arch of the aorta below the thoracic inlet. The thoracic duct rises posteriorly on the left to arch over the left pleural dome and empty into the left subclavian vein as it unites with the left internal jugular vein to form the innominate vein.

The thoracic inlet is a relatively crowded and fixed space and major shifts of the mediastinum within the chest will lead to compression of the veins draining the head, neck, and upper limbs with resultant decreased venous return and preload.

The Phrenic Nerve

Within the chest cavity the phrenic nerves descend with the pericardiophrenic vessels on either side of the heart to penetrate the diaphragm and supply it from below. Since the diaphragm is the main muscle of respiration it is important to remember the course of the nerve when performing an emergency thoracotomy for penetrating injury to the heart. Dividing the phrenic nerve when incising the pericardium in the process will cause ipsilateral paralysis of the diaphragm, hypoventilation and atelectasis or segmental collapse, with the risk of secondary infection. There is sufficient laxity with the nerve that it may be swept posteriorly when working on the heart from a lateral approach.

Intercostal Neurovascular Bundle

The intercostal neurovascular bundles run deep to the intercostal muscles between internal intercostal and the often attenuated transversus muscles outside of parietal pleura. The bundle is oriented with vein uppermost, then artery then intercostal nerve lowermost and all intimate to the undersurface of the rib above. When entering the chest cavity to place a drain or for access to structures demanding surgical attention incise intercostal muscles along their attachment to the upper border of the rib below to minimize injury risk to the bundle and nerve in particular.
Extensions of the Pleura

As mentioned above, the dome of the pleural over the apices of the lungs rise into the thoracic inlet where fascial thickening of the suprapleural membrane (Sibson's fascia) anchors the pleura to the internal surface of the first rib and transverse process of C7. This prevents ballooning of the pleura up into the neck. Inferiorly the parietal pleura descends to the level of the neck of the 12th rib forming the posterior costophrenic recess. Though the lung does not usually descend down to this level it is important to be aware of this inferior reflection when dealing with a penetrating injury to the kidney. The antero-left pleural reflexion usually occurs before the left border of the sternum so that a needle may be passed through the 4th and 5th intercostal spaces immediately lateral to the sternum and into the pericardium without crossing the pleura.

Thoracic Aorta

The aorta arises in the middle mediastinum, arches through the superior mediastinum and descends as a relatively fixed structure in the posterior mediastinum. The aortic root and heart have a greater degree of mobility and during sudden deceleration shearing forces occur across the aortic wall at the point where it becomes fixed. The aortic wall just distal to the left subclavian origin is where these forces are usually maximal and this is typically the point of initiation of an aortic dissection or rupture.

Pathophysiology of Interference with Breathing

As can be appreciated already, the chest injured patient is in a particularly perilous situation for the patient faces not one but three evolving injuries. Firstly there is the injury to the tissues themselves. Secondly there is the effect of hampered ventilation so that oxygen supply to meet the increased metabolic demands after trauma cannot be met. Thirdly the patient can enter into a negative cycle where ventilatory effort becomes further uncoupled or ineffective due to hypoxia and acidosis, exacerbating all other injuries. Time is of the essence and knowledge of serious injuries associated with blunt and penetrating chest trauma, and how to manage them simply, will benefit patients.

Diagnosis and Management of Life Threatening Injuries:

Primary assessment

The purpose of the primary survey is rapid targeted assessment of the airway, breathing, and circulation to identify those injuries that MUST be corrected immediately to prevent rapid death.

The conditions to look for during rapid and systematic primary survey are:

- tension pneumothorax
- open pneumothorax
- flail chest
- massive haemothorax
- cardiac tamponade
The airway (with C-spine control), breathing and circulation are assessed with the patient usually in a supine position (unless airway management mandates an erect posture) and adequate exposure. Control of the examination environment is necessary to prevent undue cooling of the patient and to preserve dignity.

Airway assessment and management has been addressed in previous chapters. Breathing and chest examination follows a rapid and clinically fluid progression through inspection, palpation, percussion and auscultation.

**Secondary survey**

Conditions to think about during the secondary survey are:
- lung contusion
- cardiac contusion
- rib fractures and flail segment
- blunt aortic injury
- oesophageal injury
- diaphragmatic rupture

During a more methodical examination of the respiratory system consider what may be found and be recorded with:

**Inspection:**
- signs of cyanosis?
- depth and rate of breathing?
- use of accessory muscles?
- tracheal tugging?
- dilated neck veins?
- obvious wounds?
  - penetration points
  - open fractures
  - abrasions, bruising associated with deceleration injury / blunt trauma
- don’t forget the posterior chest

**Palpation:**
- tracheal position - is it deviated to one side?
- chest wall deformity?
- normal chest wall excursion?
- asymmetric chest wall movement?
- flail chest segment?
- crepitus from rib fractures?

**Percussion:**
- resonant - is it normal?
- hyper-resonant - is there a pneumothorax?
- dull to percussion - is there haemothorax? or collapse? is it too early for dullness from lung contusion or consolidation?
- do percussion notes change with altered posture from supine to erect?
Auscultation:
- Are breath sounds present and normal?
- Are breath sounds present throughout both lung fields?

Pulse-oximetry and chest x-ray (CXR) are adjuncts to your assessment; therefore do not wait for their availability before starting your assessment. Act to treat what you find that is of immediate threat to the patient.

**Immediately Life-threatening injuries:**

**Tension Pneumothorax**

Tension pneumothorax is a clinical diagnosis and requires immediate action, with ipsilateral needle decompression and then a chest drain [2]. Action, not X-Rays, is necessary for immediate survival.

**Definition:** Tension pneumothorax is a consequence of a flap-valve, one way mechanism in the pleural membrane where the pleural space is in communication with the outside atmosphere or a conducting airway. Air flows in one way only and creates positive pressure (tension) in the pleural space. It is rapidly life-threatening.

**Pathophysiology:** Inspiration generates negative intra-thoracic pressure. Each breath draws air into the pleural cavity, the air cannot escape. Initially the affected lung collapses, and with increased intrapleural volume, the mediastinum shifts away from the affected side. This compresses the superior and inferior vena cava. Venous return to the heart drops and cardiac arrest with pulseless electrical arrhythmia (PEA) rapidly occurs [3]. Increasing hypoxia leads to increasing air hunger and tachypnoea, which accelerates the pathological process, a negative vicious cycle.

An illustrative CXR of a right sided tension pneumothorax with mediastinal shift can be seen by following the link below:

(Alice please insert figure 1 about here – attribute to wikicommmons)

**Diagnosis:** The diagnosis of tension pneumothorax is CLINICAL. Chest X-ray is NOT required and can cause lethal delay.

A patient will present with one or more of:
- History of chest trauma (often penetrating trauma),
- Respiratory distress,
- Air hunger,
- Increased JVP or distended neck veins,
- Tracheal Deviation AWAY from the affected side,
- Hyper-resonance to percussion on the affected side,
- Diminished or absent breath sounds on the affected side,
- PEA arrest.
**Immediate Management = Needle decompression:** No further investigations are required. Immediate action is essential. Needle decompression by insertion of a 14 gauge, 5cm long needle in the second intercostal space in the mid-clavicular line should be performed.

Be sure to use a long enough needle. Cadaveric studies indicate that at this site, the pleural cavity can be deeper than perceived, and you are unlikely to cause significant harm through this procedure [4].

Once needle decompression has been performed, the pleural space is decompressed. This buys time for definitive management, which is insertion of a formal chest drain.

In Summary:
1. confirm the affected side clinically,
2. inform the patient,
3. antiseptic swab the skin at the 2nd intercostal space in the mid-clavicular line,
4. insert a 14 Gauge cannula (usually orange or brown capped) +/- syringe,
5. listen for 'hiss' (or 'bubbling' if the syringe barrel is filled with water and the plunger removed),
6. protect with gauze swab, tape, and *leave in situ*,
7. set up chest drain.

Illustrations can be found at http://handbook.muh.ie/trauma/Chest/TensionPneumothorax.html

**Open pneumothorax**

**Definition:** A life threatening injury where penetrating trauma opens the pleural space, causing a pneumothorax and a ‘sucking’ chest wound [5].

**Pathophysiology:** Penetrating trauma to the chest can open the pleural space. If the communication is greater than two thirds (2/3) of the diameter of the trachea, air will preferentially enter the exposed pleural space through the wound on inspiration, leading to the ipsilateral lung to collapse. Chest wall excursion during breathing still generates negative intrathoracic pressure but air moves to-and-fro through the chest wall injury, creating a sucking chest wound. The patient is now dependent upon the contralateral lung for oxygenation but the function of this lung is severely compromised. Minimal air entry occurs as preferential air flow is through the sucking chest wound and progressive mediastinal shift can occur towards the contralateral lung. Again this can cause compression of the inferior vena cava, reduced cardiac return and PEA arrest [6]. If the air is unable to escape from the pleural cavity but still able to enter on inspiration then a tension pneumothorax will develop and the lethal process is accelerated..

**Clinical Signs:**
- Respiratory Distress
- Tachypnoea and Dyspnoea
- Cyanosis
- Visible chest wound
- Asymmetrical chest expansion
- No tracheal deviation initially, but later can be away from wound
Hyper-resonant to percussion
Diminished or absent breath sounds on affected side
Air movement through the wound; noticed as “bubbling” of blood at the wound site
PEA arrest

Management: Immediate management is life saving and consists of:
- Supplemental (100%) oxygen
- Applying a flap-valve dressing
- Inserting a chest drain and applying a totally occlusive dressing to the open wound.

A flap-valve dressing is a temporizing step using an occlusive dressing applied over the wound but only taped down on three sides. With inspiration the dressing occludes the wound but on expiration allows air in the pleural space to escape. After application of the dressing the patient should, if needed, be rolled towards the injured side to relieve pressure on the fully functioning lung. This should be performed with care not to exacerbate an unstable spinal injury.

If there is any concern about a possible tension pneumothorax decompression is performed simultaneously with a large bore needle as previously described.

Once the patient is stabilized, definitive treatment is performed. Under general anaesthetic the wound is explored, debrided and closed. A chest drain is left in-situ.

More can be seen at:
http://www.trauma.org/archive/thoracic/CHESTopen.html

Massive Haemothorax

Definition: Accumulation of blood in the pleural cavity caused by bleeding from chest wall, lung parenchyma or major thoracic vessels.

Pathophysiology: The common causes of haemothorax are laceration of the lung, intercostal vessels with rib fractures or an internal mammary artery. This is usually self limiting. Laceration to larger vessels can cause major problems. Major lung vessels can be injured by penetrating objects, including rib fragments during high impact blunt injury. Each adult chest cavity can hold up to 3 litres of blood, i.e. the chest cavity can hold their entire circulating volume. Bleeding from injuries to the great vessels leads to haemomediastinum and will not enter the pleural space unless there is a concomitant breach of the pleural membrane or injury occurs at the lung hilum. Haemothorax from azygous vein disruption is rare [7].

Haemothorax is a double insult to the patient as there is progressive deterioration of effective breathing and circulation. As circulating volume is lost into the large but fixed volume of the chest cavity there is less volume for lung expansion. Consequently as the lung collapses hypoxia develops more rapidly as there is ineffective ventilation to oxygenate the remaining blood in circulation. Circulatory collapse leads to trauma cardiac arrest.

Clinical Signs: Massive haemothorax should be suspected clinically in a patient who has signs of respiratory distress and shock.
Signs of bleeding and haemodynamic instability (e.g. tachycardia, hypotension) normally present before symptoms of respiratory distress.

Chest findings during the primary survey include cyanosis, tachypnoea, tachycardia, tracheal deviation away from the affected side, decreased chest expansion, dullness to percussion, and reduced or absent air entry on the affected side.

Early CXR is a useful adjunct to making the diagnosis but should not delay management in the unstable patient with suspected massive haemothorax. At least 400ml blood has to be lost into the pleural space before blunting of the costo-phrenic angle is seen on an erect CXR.

With blunt trauma one should have a high index of suspicion for injuries that may mimic massive haemothorax, e.g. massive lung contusion, diaphragmatic rupture with intrathoracic abdominal content, and occult tension pneumothorax with small haemothorax.

**Management:**
Management of massive haemothorax includes:
- 100% oxygen
- insertion of intercostal chest drain
- maintenance of circulating volume

Although insertion of a chest drain is therapeutic for respiratory compromise caused by the massive haemothorax, it will not address the primary problem of ongoing bleeding. With intercostals vessel injury bleeding will usually stop spontaneously, or with low pressure vessels such as lesser pulmonary veins, expansion of the lung will tamponade the bleeding. With major vessel bleeding surgery will be required. Following insertion of a chest drain, emergency thoracotomy is indicated for blood loss of
  - >1500ml blood in chest drain at insertion [8],
  - >200ml/h for 4 consecutive hours [8], or
  - >100 ml/h for > 6 hours.

The patient with a large haemothorax is also likely to have other significant chest injuries such as multiple rib fractures, flail segment, and possibly a tension pneumothorax. Late complications from inadequately drained haemothorax include empyema formation if the clotted blood becomes infected. Prophylactic antibiotics may decrease the incidence of empyema and pneumonia [9].

Further information and images of haemothorax and its management can be seen at:

http://www.trauma.org/archive/thoracic/CHESThaemo.html

**Cardiac Tamponade**

**Definition:** A life threatening condition where accumulation of blood (or other fluid) in the pericardial space around the heart restricts cardiac output and rapidly leads to cardiac arrest.
**Pathophysiology:** Penetrating trauma to the pericardium and heart occurs. The small hole in the pericardium rapidly seals with clot, but bleeding from the heart continues and fills the pericardial space. The fibro-elastic pericardial sac cannot dilate and the cardiac chambers are compressed, especially the atria, which are prevented from filling, leading to obstructive shock. Cardiac output falls and the patient progresses to cardiac arrest without intervention [2]. As little as 100ml blood can cause tamponade in the adult patient [10].

**Diagnosis:** A patient with penetrating chest trauma may present with mild cardiovascular instability which quickly worsens, major cardiovascular instability or in cardiac arrest. Classical clinical signs are Beck’s triad of:
- distended neck veins (elevated venous pressure)
- hypotension
- muffled heart Sounds

Other signs are:
- Kussmaul’s Sign: Rise in JVP on inspiration.
- Pulsus Paradoxus. An exaggerated fall in blood pressure on inspiration (>10 mmHg in systolic pressure). This can be difficult to elicit and not a reliable sign [11].
- PEA arrest.

On CXR there might be a globular heart shape and ECG may show small complexes with tachycardia. Again these are unreliable signs.

**NOTE:** Evidence of penetrating trauma to the central chest with hypotension should always raise the suspicion of cardiac tamponade.

**Management:** Urgent intervention can be life-saving.
Resuscitation should be continued, with 100% oxygen and administration of intravenous fluid or blood products if available. This increases cardiac filling pressure and can temporarily improve the situation. The aim is to maintain cerebral perfusion but not to chase a normal systolic pressure as this will increase the rate and volume of bleeding into the pericardial sac.

Needle pericardiocentesis can be performed by inserting a large bore needle between the xiphisternum and left subcostal margin, aiming at the left shoulder. Withdrawing 50ml of blood can improve the situation. Blood drawn from the pericardium usually does not clot whereas blood drawn from the heart does [12]. Often pericardiocentesis fails, however, and urgent surgery is necessary. Pericardiocentesis also carries the serious risk of damage to coronary vessels.

Definitive treatment is via thoracotomy which should be done in preference to pericardiocentesis or as soon as possible thereafter. Exposure can be via median sternotomy, a left anterior thoracotomy or ‘clam-shell’ thoracotomy. The ‘bulging’ pericardium is identified and incised, avoiding the phrenic nerve. Once this occurs the tamponade is released. Often only a small amount of bleeding from the heart is seen which can be repaired with silk sutures. If the surgeon is inexperienced in suturing the beating heart the cardiac defect can be closed temporarily with skin staples before referral to a cardiac surgery unit. Care should be taken to identify the coronary vessels and to check for posterior cardiac wounds.
Complications of management include internal mammary and coronary artery injury, ventricular puncture and aspiration, introduction of infection and precipitation of percarditis, and phrenic nerve injury during surgical approach through the pericardial sac.

Injuries associated with cardiac tamponade include cardiac contusion [13] and coronary artery injury which may have a delayed presentation [14].

Further information and images of cardiac tamponade (including how to perform pericardiocentesis) may be seen at:  

**Other Potentially Life Threatening Injuries**

**Flail chest**

**Definition:** Flail chest injury occurs when two or more contiguous fractures are present in two or more neighbouring ribs with paradoxical movement of the chest wall segment relative to the breathing cycle. This can also occur due to disruption at the costochondral junctions, which makes the whole sternum a flail segment. Costochondral injury and flail sternum is more frequent among children.

**Pathophysiology:** During inspiration the chest wall expands but the flail segment moves inwards due to the sucking effect of negative intrathoracic pressure on the flail segment. This limits lung expansion, with ineffective ventilation and hypoxia. Significant force is necessary to fracture ribs at multiple sites; therefore this injury is often associated with extensive lung contusion, haemothorax and pneumothorax due to the rib fractures. Underlying injuries are more likely to cause respiratory dysfunction than the flail segment itself. Severe pain due to multiple fractures leads to shallow breathing, worsening ventilation even further; combined with contusion this often leads to retention of secretions, airway collapse and pneumonia.

**Diagnosis:**  
Flail chest is a clinical anatomical diagnosis. It is important to look past the flail segment for underlying pathology [15].

Clinical examination will reveal a patient with tachypnoea, and signs of blunt trauma to the chest wall. The flail segment is identified by its paradoxical movement on spontaneous breathing and is often more obvious to feel than to see (If the patient is intubated this sign disappears with positive pressure ventilation). Palpation may identify crepitus from the broken rib ends and percussion exacerbates pain.

Moderate to severe respiratory distress occur proportional to the severity and extent of underlying injury.

**Management** is in the form of rib fracture treatment and management of underlying pulmonary contusion [16]. This includes:  
- 100% oxygen
- regular analgesia - consider using rib blocks with local anaesthetic
- chest drain(s) for associated pneumothorax or haemothorax
- consider assisted ventilation if there is inadequate ventilation or the patient is tiring. Ventilatory support is more likely with:
  - large flail segment or one involving the sternum,
  - extensive lung contusion.

Further information and images of flail chest injury may be seen at:

http://www.trauma.org/archive/thoracic/CHESTflail.html

**Pulmonary Contusion**

**Definition:** An injury to lung parenchyma secondary to blunt trauma. Young children have pliable chest walls and can have severe lung contusion without rib fractures.

**Pathophysiology:** Following blunt trauma, oedema and blood collect in the alveolar space. This causes ventilation/perfusion mismatch which evolves over a period of 24 hours. As the injury evolves, the patient suffers from impaired gas exchange, increased pulmonary vascular resistance and decreased lung compliance [17]. Adult Respiratory Distress Syndrome can occur in conjunction with this injury.

**Clinical Features:** Pulmonary contusion is difficult to diagnose clinically. The presence of rib fractures or flail chest and blunt force trauma should arouse suspicion. Have a high index of suspicion in all children who were unrestrained during an RTA or who have fallen from a height. The clinical picture is one of a patient with escalating oxygen requirements and respiratory difficulties as the underlying pathology evolves [18].

**Diagnosis:** Chest X-ray is useful, though radiographic changes can lag clinical signs. CT gives accurate diagnosis of pulmonary contusion and differentiation from other clinical entities such as atelectasis.

**Management:**
Supportive management of the patient is required for a period of 3-5 days to allow the contusion to resolve. In general this involves supplemental oxygen if necessary and adequate analgesia and physiotherapy to avoid complications such as pneumonia.

If contusion is severe and ARDS occurs with respiratory failure, further respiratory support will be required, usually with intubation and ventilation.

**Aortic injury**

**Definition:** Patients who sustain an aortic transection injury almost always die at the scene of the accident and account for around 15% of trauma related deaths. Only 15% of those who sustain a blunt aortic injury make it to the hospital alive [19] and these patients are likely to have a tear with dissection or pseudoaneurysm formation.

**Pathogenesis:** During a sudden deceleration injury, such as in a motor vehicle crash or fall from height, the ascending aorta and aortic arch move within the chest cavity, generating maximal shearing forces between the relatively fixed proximal and the descending thoracic
aorta; the majority of tears or transections therefore occur just distal to the left subclavian artery origin [20].

**Diagnosis:**
Clinical assessment may reveal an interscapular flow murmur in a patient with upper thoracic back pain. CXR findings suggestive of aortic injury include wide mediastinum (>8cm), indistinct aortic knuckle, and depressed left main bronchus. A left sided haemothorax that returns arterial blood on chest drain insertion should raise the index of suspicion. Other diagnostic investigations in the more stable patient include trans-oesophageal Doppler, CT scanning and angiography.

**Management** involves judicious resuscitation with blood pressure control. Overzealous fluid resuscitation may lead to re-bleeding from the site of aortic injury in the haemodiluted patient. Prompt surgical repair through either endovascular [21] or open approach is necessary; by-pass lowers the risk of post-procedure paraplegia [19].

Further information and images of thoracic aorta injuries may be seen at:

http://www.trauma.org/archive/thoracic/CHESTaorta.html


**Other chest injuries that can lead to severe complications if not treated optimally:**

**Simple pneumothorax**
Simple pneumothorax develops following transitory escape of air into the pleural space with partial collapse of the lung. It may occur following either penetrating or blunt trauma with rib fractures and may be diagnosed clinically (if large enough) or detected incidentally on Chest X-ray (this is one reason why compulsory CXR should be included in the “trauma series” after any serious injury). The patient may be tachypneic, have decreased chest expansion on the afflicted side, be hyper-resonant on percussion, and have decreased air entry on auscultation. The patient does not exhibit signs of shock or rapid deterioration as seen in tension pneumothorax or open pneumothorax. A trauma patient with a simple pneumothorax will still likely require a chest drain but this usually may wait until after the secondary survey. If the patient is transferred or is to have a general anaesthetic a chest drain is essential; a small pneumothorax will rapidly expand and become life threatening with positive pressure ventilation or at lower atmospheric pressure (e.g. in an aeroplane).

**Rib Fractures**
Rib fractures are commonly encountered in thoracic trauma.
**Pathophysiology:** Rib fractures per se are not problematic but associated pain limits both inspiration and expiration, and prevents effective coughing. The patient is at risk of hypoventilation, retention of secretions, secondary infection and pneumonia, which can have serious consequences.

**Clinical Features:** The patient will present with pain and or dyspnoea. Always consider a significant underlying injury if there is associated respiratory failure or haemodynamic instability.

**Management** of simple rib fractures
- Analgesia and targeted physiotherapy to prevent complications.
- Attention to underlying pathology.
- Rib fractures themselves will heal without specific intervention.

Management of pain from rib fractures is essential to prevent hypoventilation and pneumonia. Non-steroidal anti-inflammatory drugs provide excellent analgesia if there are no contra-indications. Paracetamol and opiate drugs could also be utilized if necessary. Occasionally pain control is problematic. Patient Controlled Analgesia (PCA) can be used as an adjunct to therapy, and intercostal/regional anaesthesia can be effective if a sufficiently experienced anaesthetist is present [22]. This is not without associated complications however.

Once analgesia is satisfactory, targeted physiotherapy is required to ensure hypoventilation does not occur.

**Injuries associated with specific rib and bony fractures of the chest wall:**

Chest wall bony fractures detected on CXR should raise suspicion for associated injuries to neighbouring organs:

1st rib: lung apices, subclavian vessels
2nd rib: ascending aorta, superior vena cava
Clavicle: lung apices, subclavian vessels
Sternum: myocardial contusion, internal thoracic vessels
10th rib: diaphragmatic, liver, splenic injury
11th rib: diaphragmatic, liver, splenic injury
12th rib: renal injury.

**Myocardial contusion**

Cardiac contusion usually occurs due to severe direct blunt trauma to the anterior chest. It is caused by rapid deceleration injury, e.g. against a steering wheel during a car crash. Shearing forces cause bleeding and bruising within the myocardium. This will usually not present with clinical features but with a range of ECG abnormalities once the patient has been stabilised and is monitored in the HDU or ICU. The ECG will almost always return to normal as the bruising settles. No specific treatment is necessary but life threatening ventricular arrhythmias need to be managed as with any other cause.
**Thoracic vertebral fractures**

It is important in the patient who has sustained blunt chest trauma that the thoracic vertebrae are not forgotten. A lateral thoracic spine X-ray is helpful in assessing the thoracic spine even when no ‘step’ deformity or spinous process injury is detected on palpation in the secondary survey. The integrity of each vertebral body should be inspected on the X-ray as well as integrity of the three conceptual columns within the spinal column.

**Oesophageal injury**

**Definition:** Oesophageal injury during trauma is rare but under-diagnosed; occult injuries are easily missed during initial assessment.

**Pathophysiology:** There are two possible mechanisms of oesophageal injury:
- raised luminal pressure against a closed glottis leading to a ‘blow out’ injury;
- crush injury between the sternum and the thoracic vertebrae with anterior compression injury.

**Diagnosis:** The patient complains of pain on swallowing. Crepitus or surgical emphysema may be felt in the neck and pneumomediastinum seen on CXR.

**Management** involves[23]:
- drainage of the chest cavity at the site of the perforation or tear
- delineation of the extent of the injury
- debridement of necrotic tissue
- decortication of soiled pleural space
- defect closure with flap or pedicle buttressing
- diversion? - avoid if at all possible.

Life threatening mediastinitis may develop following oesophageal injury and particularly with late diagnosis. Wound toilet and antibiotic prophylaxis (with anaerobic cover) is necessary.

**Chest Drain / Thoracostomy Tube:**

A chest drain is indicated in the management of a tension pneumothorax and should also be considered to manage open pneumothorax, simple pneumothorax, haemothorax, and the trauma patient who arrests. A chest drain may be placed prophylactically in trauma patients prior to transfer to another institution e.g tertiary care centre, and in patients with rib fractures who require ventilation.

To place a chest drain:
1. inform the patient,
2. check all equipment required,
3. confirm the side requiring the drain,
4. prepare the chest wall skin with antiseptic and a sterile field,
5. identify the optimum site for access in the ‘triangle of safety’ - 4th or 5th intercostal space in the anterior- or mid-axillary line,
6. infiltrate local anaesthetic to skin, subcutaneous tissues, periosteum of upper edge of the rib below, intercostal spaces (avoiding intercostal vessels), and to parietal pleura,
7. aspiration with the infiltration needle at each advancing step, prior to instillation of local anaesthetic, will avoid intravascular injection and confirm entry into the pleural cavity, when air is returned, as well as gauge the depth of the chest wall,
8. incise the skin along the upper border of the rib below,
9. use a curved haemostat / curved forcep to bluntly dissect down to pleura,
10. explore the track with a sterile gloved finger to breach pleura and confirm pleural cavity entry, (think about what lung, diaphragm, liver, intestinal tissues would feel like),
11. occlude the track with a finger during inspiration,
12. remove the metal trocar from a large bore (32-36F) chest drain and either pass it gripped in the curved forcep or by hand into the pleural cavity angling the direction towards the apex to manage a pneumothorax or postero-inferiorly to manage a haemothorax,
13. do not force the drain in but re-explore the track if there is significant resistance,
14. ensure all the fenestrations / holes in the tube are inside the patient,
15. secure the drain and approximate skin around the tube with a heavy suture and apply an occlusive dressing,
16. connect the drain to an underwater seal placed below the level of the patient,
17. re-examine the patient for clinical change,
18. examine what is drained: arterial or venous blood / lymph / intestinal content?,
19. confirm drain orientation, position, and effect with CXR.

Illustrations and guidance for chest drain insertion may be seen in the following link:


Summary

Chest trauma can cause immediately life threatening pathology that must be recognised and managed during primary survey and resuscitation. Often the patient can be kept alive through simple emergency room procedures until help arrives and/or definitive treatment for specific injuries can be instituted. Other serious injuries may not need life-saving treatment within minutes, but still need recognition and a treatment plan while the patient is in the emergency room to prevent mortality. Lastly there are injuries that can seem innocuous or are not easily diagnosed, but will have significant morbidity and mortality if missed or treated suboptimally.

All these injuries should be managed appropriately through proper knowledge, a system of rapid primary and secondary assessment, supported by effective resuscitation, rapid emergency procedures and definitive management, and ultimately by repeated practice in simulated and real trauma environments.
References


