**European Trauma Care Course**
**THORACIC TRAUMA**

**Epidemiology**
The rate of thoracic trauma in the United States is 12 per million population per day - and 20-25% of deaths due to trauma are attributed to thoracic injury. It is estimated that thoracic trauma is responsible of approximately 16,000 deaths per year in the United States. The incidence has increased rapidly in this century of high-speed vehicular travel. Immediate deaths are usually due to major disruption of the heart or of great vessels. Early deaths due to thoracic trauma occurring within 30 minutes to 3 hours after the injury are secondary to cardiac tamponade, airway obstruction and aspiration.

Two thirds of these patients reach the hospital alive. Only 10-15% of blunt trauma requires thoracic surgery, while 15-30% of the penetrating chest trauma requires open thoracotomy. 85% of patients with thoracic trauma can be managed by simple lifesaving manoeuvres that do not require surgical treatment.

Thoracic injury occurs in the chest wall, lungs and pleura, thoracic great vessels, diaphragm, heart, trachea, bronchus and oesophagus. The magnitude of these problems and the significance of the associated injuries serve to underscore the importance of thorough evaluation and timely intervention in the management of thoracic trauma.

Many patients can be successfully treated with tube thoracostomy, respiratory support and less commonly, emergency thoracotomy. Tracheal intubation supports and treats post-traumatic respiratory insufficiency. Although some of the complex and potentially fatal traumatic thoracic injuries require emergent surgical intervention, most of them can be treated nonoperatively by the proper application of certain fundamental principles of initial trauma management. These principles can substantially reduce both morbidity and mortality. Appropriate early management of the rapidly fatal and potentially fatal thoracic injuries can also significantly decrease the late complications.

Optimal treatment requires a through knowledge of the ethiology and pathophysiology of the thorax and expertise the therapeutic interventions. Improved prehospital care and rapid transportation have increased survival, but lethality remains high.

**Pathophysiology**
Thoracic trauma can induce two serious derangement:

1. Respiratory insufficiency due to:
   - pneumothorax
   - tension pneumothorax
   - open pneumothorax
   - flail chest
   - pulmonary contusion
   - aspiration
2. Hemorrhagic shock due to:
   - haemothorax
   - haemomediastinum

The thoracic cavity is made up of two structures: The rigid bony structures comprising the rib cage, clavicle, sternum, scapula and the dynamic respiratory muscles. Adequate ventilation and oxygenation depends on an intact chest wall. Significant injury with fracture and muscular disruption may allow direct injury to the underlying lungs, heart, great vessels and upper abdominal viscera. In addition, respiration may be seriously impaired by ineffective or paradoxical motion of a portion of the thoracic cage (as in flail chest) and the result is respiratory insufficiency.

Penetrating wounds of the chest (gunshot or stab wound) may cause comminuted fractures of a rib, with bone fragments driven into the lung substance. The most common manifestation of penetrating trauma to the visceral and parietal pleura is disruption of normal negative intrapleural pressure resulting in pneumothorax. Penetrating wounds cause both direct injury to structures encountered by the weapon and indirect injury. The extent of internal injuries cannot be judge by the appearance of a skin wound.

Blunt forces applied to the chest wall cause injury by three mechanisms: rapid deceleration, direct impact and compression. Rapid deceleration is the usual force involved in high speed motor vehicle accidents and falls from a height. The degree of external trauma may not fully predict the severity of internal injuries and clinical suspicion of cardiac and vascular trauma should be heightened.

Direct impact by a blunt object can cause localised fractures of the ribs, sternum or scapula with underlying lung parenchyma injury, cardiac contusion or pneumothorax.

Compression of the chest by a very heavy object, which prevents respiration and causes marked increases in blood pressure within veins of the upper thorax, may result in traumatic asphyxia. Anterior-posterior compression forces place indirect pressure on the ribs, causing lateral, mid-shaft fractures. Lateral compression forces applied to the shoulder are common causes of sternoclavicular joint dislocation and clavicle fractures. Massive blunt injury to the chest wall may comprise elements of deceleration, direct impact, and compression to yield multiple adjacent rib fractures. In this setting, a free-floating segment of the chest wall can move paradoxically with respiration causing ineffective ventilation.

As well as respiratory insufficiency thoracic trauma can cause haemorrhagic shock due to haemothorax and rarely to haemomediastinum. Haemothorax is common in both penetrating and non-penetrating injuries to the chest. If the haemorrhage is severe, it may not only cause hypovolemic shock but also dangerously reduces vital capacity by compressing the lung on the involved side. Persistent haemorrhage usually arises from an intercostal or internal thoracic (internal mammary) artery and less frequently from the major hilar vessels. Bleeding from the lung generally stops within a few minutes, although initially it may be profuse. In some cases haemothorax may come from a wound of the heart or from abdominal structures such as the liver or spleen if the diaphragm has been lacerated. Hypovolemic shock and haemomediastinum can derive from thoracic great vessel injury that may be result of penetrating or blunt trauma. The most common etiology is penetrating trauma; however, the descending thoracic aorta, the innominate artery, the
pulmonary veins, and the vena cavae are susceptible to rupture from blunt trauma.

**Aims of the Chapter**

At the end of this chapter the student should be able to identify and treat chest trauma and to understand and avoid the most common mistakes and complications in its management. The initial management of patients with thoracic trauma is frequently the responsibility of the emergency physician who is not a thoracic surgeon. It is therefore imperative that emergency physician is able to recognise the thoracic injuries that require urgent appropriate treatment. In unstable and critical circumstances, quick decisions and adequate manoeuvres based on recordings of vital signs and correct interpretation of clinical and diagnostic patterns are required. The purpose of this chapter is to establish a set of guidelines for the treatment of thoracic injuries.

**Priorities**

The evaluation of the patient's chest trauma is only a part of the total assessment; furthermore because thoracic injuries are severe and potentially lethal, diagnosis and therapy must go hand in hand.

In unstable and critical patients quick decisions based on the following vital signs are required.

**Airway patency**: in the initial survey is vital to maintain airway patency. The airway can be occluded by foreign bodies present in the month or by the tongue falling backwards, as occurs in unconscious patients. All airway manipulations must be performed with control of the cervical spine.

**Breathing**: in order to know if patient is breathing is necessary to check respiratory movements and excursion. Remember that cyanosis appears very late in hypoxia due to thoracic trauma because in shocked patients the skin blood flow is affected by blood redistribution throughout the body.

**Circulation**: the state of the circulation is evaluated by assessing the patient's pulses (radial, carotid or femoral). The blood pressure is evaluated along with pulse pressure. In hypovolemic shock the radial pulse is diminished and may become absent when the blood pressure is below 70 mm/Hg. In thoracic trauma it is important to assess the neck veins - flat in hypovolaemia and distended when there is cardiac tamponade. However if cardiac tamponade is associated with hypovolemic shock distension of the neck veins may be absent.

In thoracic trauma patients the following life-threatening injuries must be immediately identified and treated.

**Open pneumothorax**

Usually results from a penetrating wound of the chest that may create a communication between the pleural space and external environment. As the size of this defect approaches two thirds the size of the tracheal diameter, air passes preferentially through the lower resistance injury tract rather than through the normal airways. This severely compromises oxygenation and ventilation and is immediately life-threatening. In an open or "sucking" wound of the chest wall, the lung on the affected side is exposed to atmospheric
pressure, which results in the lung’s collapse and a shift of the mediastinum to the uninvolved side. The severe venoarterial shunting that occurs in both lungs results in profound ventilation-perfusion inequality and the patient becomes cyanotic and develops respiratory distress.

The open pneumothorax must be treated rapidly using one of two approaches. In the spontaneously ventilating patient, application of a sterile occlusive dressing with vaseline gauze large enough to cover the wound entirely taped securely on three sides is the treatment of choice. Tube thoracostomy should be performed at a different site. If the chest wall defect is relatively small, the pleura may soon seal and no further intervention is necessary. A second approach is to simply intubate the patient and initiate positive pressure ventilation. Often surgical repair is required.

**Tension pneumothorax**
Develops when air enters the pleural space but cannot exit. The consequence is progressively increasing intrathoracic pressure in the affected hemithorax resulting in impaired central venous return and mediastinal shift. Air enters pleural cavity through lung wound or ruptured bleb with a valvelike opening. The ipsilateral lung collapses and the mediastinum shifts to opposite side compressing the contralateral lung and impairing its ventilating capacity. Clinically, the patient experiences dyspnea, complains of chest pains, and becomes cyanotic because of shunting in the collapsed lung and has haemodynamic instability due to decreased venous return to the heart. The presence of hyperresonance and the absence of breath sounds, should be useful in confirming diagnosis.

A chest X-ray film indicates that the trachea and mediastinum are deviated to the side opposite the tension pneumothorax, while on the ipsilateral side intercostal spaces are widened and the diaphragm is pushed downward. The emergency require immediate thoracostomy with underwater-seal drainage. When a tension pneumothorax is suspected, the lung must be decompressed before waiting for a chest X-Ray. If the lung does not fully re-expand after tube thoracostomy and there is a large ongoing air leak the airways should be evaluated bronchoscopically to exclude a major injury. However, in most cases, no further treatment for tension pneumothorax will be required after chest tube insertion.

**Massive haemothorax**
Common in both penetrating and blunt chest injuries. Patients who sustain an acute haemothorax are at risk of haemodynamic instability due to loss of intravascular volume and compromised central venous return due to increased intrathoracic pressure. Lung compression due to massive blood accumulation may also cause respiratory compromise. Sources of haemothorax are lung, intercostal vessels, internal mammary artery, thoracicoaomrial artery, lateral thoracic artery, mediastinal great vessels, heart, and abdominal structures (liver, spleen) when there is diaphragmatic rupture.

The diagnosis is readily made from the clinical picture and X-ray evidence of fluid in the pleural space. Therapy consists of the placement of a large (32-36 French) chest tube. A moderate size haemothorax (500-1500 ml) that stops bleeding after thoracostomy can generally be treated by closed drainage alone. However, a haemothorax of greater than 1500 to 2000 ml or with continued bleeding of more than 200 ml per hour is an indication for emergency thoracotomy or thoracoscopy.

A small percentage of haemothoraces proceed to clot and cannot be
evacuated by thoracentesis. Massive clots may lead to respiratory difficulty and infection, and should be evacuated surgically. Small clots will probably be resorbed and do not require operative removal.

**Cardiac tamponade**

Penetrating cardiac injuries are a leading cause of traumatic death in urban areas and are generally more severe than blunt injuries. Patients with penetrating wounds of the heart can be classified into 3 general groups:

1. Patients who have received extensive lacerations or large-calibre gunshot wounds, that die almost immediately, as a result of rapid blood loss

2. Patients with small wounds of the heart, caused by ice picks, knives or other small agents who because of the development of cardiac tamponade, reach the hospital alive. Cardiac tamponade, by bringing pressure to bear on the bleeding heart wall, also plays an important role in controlling the haemorrhage.

3. Patients with associated serious injuries in the chest and/or elsewhere in the body which, in themselves, may contribute to death.

The condition of the patient, when he is admitted to the hospital, must not be used as an index of the severity of the injury. There are moribund patients with no blood pressure and impalpable pulses, who survive operation and recover while there are patients in fair condition, with a systolic blood pressure ranging from 70 mmHg to normal and good pulses, who die before surgery. The immediate cause of death is either exanguination, cardiac tamponade or interference with the conducting mechanism of the heart.

Diagnosis generally is easy if the physician maintains a high degree of suspicion of heart injury in every chest wound he encounters. The safest approach is to remove the patient's clothing and survey the entire body surface quickly for evidence of multiple injuries. Auscultation of the thorax is performed specifically to evaluate the clarity of heart tones and breath sounds. Muffled heart tones are an indication of blood in the pericardium. A systolic - to diastolic gradient of less then 30 mmHg, associated with hypotension is consistent with cardiac tamponade. Neck veins are distended. Central venous pressure is elevated. The X-ray film may demonstrate a widening of the cardiac silhouette. An ultrasound scan shows presence of blood in pericardial space. Electrocardiograph may not be particularly helpful. Prompt definitive therapy is imperative. This includes antishock therapy, emergency thoracotomy and repair of the wound.

The following lesions are life-threatening but give to the physician more time to diagnose and treat them.

**Thoracic cage injuries**

With severe trauma, fractures of any rib or combination of ribs and fractures in this area are likely. However, the fracture usually occurs at the point of impact, often laterally and such fractures are hard to see on X-ray films. Crushing injuries may produce multiple eggshell fractures, the sites being dependent on the direction of the compressing forces. For example, impaling the anterior chest on a steering wheel as in an automobile accident, often fractures the sternum and several ribs anteriorly on both sides. Besides rib fractures, costovertebral dislocation may occur at any level, as may occur costochondral and chondrosternal separations.

Fractures may be transverse or oblique and the fragments may override or a
pointed fragment may be pushed inward, tearing the pleura and underlying lung.

In the elderly patient with atrophic, decalcified ribs, fractures may result from simple trauma, coughing or any severe muscle pull. Fractures of the rib or sternum or costovertebral separations are diagnosed clinically from movement of fragments, ecchymosis and crepitus, as well as by X-ray examination. Since pain characteristically occurs with inspiration, the patient tends to splint the chest wall and therefore, hypoventilates.

A chest X-ray film must be performed, not only to identify the number and the extent of rib fractures, but also to determine whether there is an associated pneumothorax, haemothorax or pleural effusion. Rib fractures usually heal readily if complications are handled properly. However, the pain associated with the fracture can prevent proper ventilation and coughing, leading to atelectasis, retained secretions and pneumonia, especially in the elderly. Damage to the underlying lung may cause haemothorax, pneumothorax or pulmonary contusion. Multiple rib fractures may produce paradoxical movement of the chest wall, with a flail segment. Pain from a rib fracture can be treated by intercostal or paravertebral block; this promptly relieves the pain and quiets the laboured respiration which may be accentuating paradoxical motion of the chest. The major problem with a block are increased reflex bronchial secretions; these must be removed if patients are to avoid an obstructive type of pneumonia which is particularly dangerous in the elderly.

If coughing is inadequate, tracheal aspiration by catheter or by broncoscopy and occasionally via tracheal intubation may be necessary. The ribs usually become fairly stable within 10 days to two weeks. Firm healing with callus formation is seen after about six weeks.

Flail chest
A segment of chest wall does not have continuity with the rest of thoracic cage due to multiple rib fractures, leading to serious respiratory distress.

In flail chest injury, the unstable segment wall moves separately and in an opposite direction from the rest of the thoracic cage during the respiratory cycle. In polytrauma patients flail chest injury is quite common. 31% percent of 50.000 trauma patients included in the Major Trauma Outcome Study had chest injuries. 5% of these patients had flail chest injury.

Flail chest injury usually results from direct impact. If the crushing force is from the lateral direction, the injury usually consists of fractures in at least two sites in multiple adjacent ribs on the involved side, resulting in a "floating" central portion of the chest wall, which ventilates in a reverse or paradoxical manner to the rest of the chest wall. This type of respiration markedly decreases the efficiency of ventilation, and is usually accompanied by severe pain that makes the coughing mechanism ineffectual. If the crushing blow is directly over the sternum, as often happens in steering wheel injuries, fractures of the sternum may occur. Such an injury is most frequently associated with bilateral costochondral fractures usually from the first rib down resulting in extreme flailing of the anterior portion of the chest wall.

The physiopathologic effects of flail chest are a significant decrease in vital capacity and functional residual capacity. Often there is ventilation-perfusion imbalance, hypoxemia, decreased compliance, increased airway resistance and increased breathing effort. The diagnosis of flail chest injury is made by physical examination. Examination after blunt chest trauma must include
inspection of an unclothed patient from anterior, posterior and both lateral angles. Clinically, the unsupported portion of the chest wall is seen to move paradoxically with respiration. On inspiration, the flail area moves in; on expiration or coughing it moves outward. If the flail segment is large, the important effect are those of inadequate ventilation, inadequate perfusion resulting in progressive hypoxia and hypercapnia and inefficient coughing with retained secretions. Radiographs can document multiple rib fractures. The treatment of flail chest involves selective use of tracheal intubation and mechanical ventilation. Not all patients require intubation. Flail chest patients without respiratory impairment generally do well without ventilatory assistance. The primary indication for tracheal intubation and mechanical ventilation is respiratory decompensation. Aggressive pulmonary physiotherapy with suctioning, incentive spirometry, early mobilisation, and humidification of air is appropriate for all patients. Intermittent positive pressure breathing, postural drainage, cupping or clapping and therapeutic fiberoptic bronchoscopy to suction retained secretions and treat atelectasis are often necessary.

Further than internal fixation effected by tracheal tube or tracheostomy, rarely stabilisation of the chest wall may be performed by external surgical fixation.

**Pneumothorax**
Lung lacerations or bronchial lesions permit air entry into the pleural space. This results in collapse of the lung, increase in intrapleural pressure and compression of the contralateral lung by mediastinal shift. In this case a severe alteration of ventilation perfusion ratio may occur because of blood circulation in unventilated pulmonary tissue. Chest percussion in patients with pneumothorax shows hyperresonance and breath-sounds are decreased or absent. Confirmation of the diagnosis is obtained by chest X-ray.

In the absence of haemodynamic compromise, even large pneumothoraces rarely require emergent management. However, any post-traumatic pneumothorax should be treated as expeditiously as possible. A chest tube is inserted after cardiologic evaluation and lung reexpansion carefully and repeatedly assessed with follow-up chest X-rays. Placement of an intercostal tube or catheter can be readily accomplished under local or intercostal nerve block anaesthesia, or both.

It may be done at the bedside, but strict aseptic precautions need to be observed. The site for tube insertion should be one that is away from adherent lung. Generally preferred is the fourth or fifth intercostal space in the midaxillary line. To help select the optimal point of entry, chest X-ray films should be reviewed unless the clinical situation is an of extreme urgency.

**Pulmonary contusion**
Pulmonary contusion is a potential life-threatening condition mainly because the onset of symptoms is insidious. Also, since the force required to produce a lung contusion must be great, the lesion is likely to occurs principally in cases of high speed accidents, falls from great heights and injuries by high-velocity bullets. Patients suffering such accidents often have so many other obvious injuries that a chest injury may escape detection. After seemingly negligible initial signs and symptoms, the outcome may be fatal. Symptoms and signs of pulmonary contusion are: dyspnea, hypoxemia, cyanosis, tachycardia, decreased or absent breath sounds and rib fractures.

Haemorrhage, oedema and microatelectasis are the morphologic consequences of pulmonary contusion. A prompt diagnosis is the main factor in initiating management and determining whether treatment will be effective.
The diagnosis is first suspected from history of major trauma. The chest X-ray film is very important, and may show patchy, undefined densities or homogenous consolidation. Patients who sustain pulmonary contusion with respiratory compromise have to be treated with intubation, mechanical ventilatory support and antibiotic therapy. Chest CT scan permits a more complete evaluation of the intrapulmonary lesion.

**Rupture of trachea or major bronchi**

Intrathoracic rupture of the trachea or major bronchi is usually secondary to an injury to the chest as a result of an automobile accident. It is a serious injury with an estimated mortality of 30%. More than 80% of ruptures of bronchi are within 2.5 cm of the carina. Injuries to the main bronchi and intrathoracic trachea are more prevalent than those to the cervical trachea because the latter is protected by the mandible and sternum anteriorly and by vertebrae posteriorly. The intimate anatomical relationship of the trachea to the great vessels, lungs and heart explain the high incidence of serious associated injuries in both blunt and penetrating trauma. The clinical picture appears in two forms, depending on whether or not there is free communication between the rupture of the trachea-bronchial tree and the pleural cavity. If there is free communication, a large pneumothorax results. The usual signs of tracheobronchial disruption are:

- haemoptysis
- dyspnea
- subcutaneous and mediastinal emphysema
- occasionally cyanosis.

Tube thoracostomy shows continuous bubbling of air in the water seal, and suction fails to reexpand the lung. The chest X-ray demonstrates pneumothorax, pleural effusion, pneumomediastinum or subcutaneous air. Overall 90% of these patients will have an abnormal chest X-ray on admission.

Bronchoscopy should be carried out promptly when tracheobronchial rupture is suspected, since it is the most reliable means of establishing the diagnosis. Bronchoscopy has a role not only in diagnosis of tracheobronchial disruption, but also may be a valuable tool in resuscitation.

Tracheal and selective bronchial intubation allows ventilation of the intact lung while bronchoscopy is performed on the contralateral bronchial rupture. Bronchoscopy should be carried out promptly; if it indicates that the bronchial tear involves less than one-third of the lumen, the patient is stable and if the thoracostomy tube and under-water-seal drainage results in complete expansion of the lung, treatment may be conservative. However in all other types of tracheobronchial injury, thoracotomy should be performed as soon as possible.

In this situation, ventilatory support with high frequency ventilation has not been shown to be useful, whereas success has followed the use of double lumen tubes and selective bronchial intubation followed by early surgical repair, by mucosal - to - mucosal closure with interrupted sutures.

**Heart injuries**

Myocardial contusion is associated, in blunt chest trauma, with fractures of the sternum or ribs. The diagnosis is based on electrocardiogram abnormalities and elevation of serial cardiac enzymes.
Cardiac contusion can simulate a frank myocardial infarction. Electrocardiographic findings are multiple, premature ventricular contractions, atrial fibrillation, right branch block and changes in ST segments. Patient must be admitted for observation with cardiac monitoring.

**Thoracic great vessels injuries**
These are principally civilian phenomena and currently account for 8-9% of vascular injuries seen in trauma centers. They may be result of penetrating or blunt trauma.

The patient with chest trauma may present with respiratory distress due to haemothorax. An initial "rush" of a large volume of blood after tube thoracostomy may indicate great vessels injury. The classic signs of pericardial tamponade may be present. Suggestive radiological signs include presence of haemothorax, pneumothorax or the foreign bodies, and abnormalities of the superior mediastinum on chest X-Ray.

Aortography should be performed in the patient with moderate to severe injuries who is stable haemodynamically. CT and MRI have not been shown to be as accurate as aortography.

The patient with signs of respiratory distress and suspected pneumothorax should undergo immediate tube thoracostomy. As mentioned above, if blood loss is greater than 1 litre with continued bleeding a thoracotomy is indicated.

**Trauma to oesophagus**
In trauma patients rupture of oesophagus is very rare. More frequent is the perforation of the oesophagus for penetrating trauma. Oesophagus trauma is lethal if unrecognised because mediastinitis due to contamination mediastinal space by oesophageal content and subsequent bacterial necrosis. The patients complain of excruciating pain in the epigastrium, which lasts and radiates to the chest, to the back or both. Dyspnea, cyanosis and shock soon set in and dominate the clinical picture. Emphysema and pneumo- or hydropneumothorax develop, especially in the left chest and become visible radiologically.

Oesophagogram may be performed when the patient is stable. Oesophagoscopic visualisation of localised blood in the oesophagus or an actual laceration is diagnostic. Operation is advocated when the patient is unstable and when multiple major injuries are present.

The principles in the management of major oesophageal injuries are those of early operation, two layer surgical closure when possible and wide mediastinal drainage. Extensive tissue destruction or associated major mediastinal contamination, such as occurs when repair is delayed by more than 12-16 hours, are indications to consider a closure with a T-tube or an exclusion technique.

**Diaphragmatic injuries**
Once relatively uncommon, injuries of the diaphragm are occurring more frequently, paralleling the rise in automobile accidents, and improvements in diagnostic modalities. The diagnosis is often missed because of associated intraabdominal injuries. Diaphragmatic injuries may be caused by penetrating or blunt trauma. Diaphragmatic injury is suspected in any penetrating thoracic wound (gunshot, stab or accidental perforation) at or below 4th intercostal space anteriorly, 6th interspace laterally, or 8th interspace posteriorly, although sharply oblique wounds or missiles deflected by ribs may also
penetrate the diaphragm. The stomach and other abdominal viscera may herniate into left thorax; left lung may collapse, right lung may be compressed, mediastinum my be shifted and trachea deviated to the right.

Symptoms are related to the quantity of herniated viscera in the thorax. Clinical manifestations may include dyspnea, chest or shoulder pain and cyanosis, but may be subtle left upper quadrant tenderness. If the herniated organ is stomach, the dyspnea may be relieved dramatically by introduction of a nasogastric tube. The diagnosis is performed or suspected on chest X-ray that may demonstrate atelectasis with silhouetting of the ipsilateral diaphragm, evidence of a viscus in the thorax or an abnormal curvilinear shadow above the diaphragm. A contrast stomach X-ray may visualise the stomach herniated into thorax. Ultrasound, CT scan or laparoscopy will confirm the diagnosis.

The most common errors in diaphragmatic trauma are failure to inspect the leaflets adequately during operative exploration or penetrating injury and failure to suspect the possibility of diaphragmatic injury.

The treatment is always surgery by an abdominal approach. The decision to repair an isolated diaphragmatic rupture in an acutely injured patient should depend on how the patient tolerates the loss of normal, negative intrathoracic pressure. Gross signs of cardiorespiratory distress or shock are indication for immediate repair.

**Thoracic aorta rupture**

Rupture of the thoracic aorta is a common cause of sudden death in cases of unrestrained frontal or lateral collision and in falls from a great height. Blunt laceration of the thoracic aorta is most commonly near the attachment of the ductus arteriosus.

If the aorta is not completely interrupted and there is an intact adventitial layer it is possible to operate on the patient and to repair the lesion. If the patient is not diagnosed and not adequately treated most will die in a few hours or days.

The chest X-ray can show a widening of the mediastinum, right tracheal shift, elevation and rightward shift of the right bronchus, depression of the left bronchus, blurring of the aortic knob outline and deviation of the oesophagus to the right,. Transoesophageal ultrasound scan is a useful diagnostic tool if available Aortography is the gold standard investigation at present. The treatment is immediate surgery.