

# Traumatic Diaphragmatic Injury

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## Abstract

**Traumatic diaphragmatic injury is a not uncommon accompaniment to blunt or penetrating trauma to the abdomen or thorax; it may present acutely with haemodynamic and respiratory compromise and be associated with significant injury to other organs or may not be diagnosed at the initial trauma at all and present later as a diaphragmatic hernia. This overview examines the incidence, pathophysiology and management of this condition.**

## Introduction

Diaphragmatic injury remains a diagnostic and therapeutic challenge to trauma physicians. Its presentation varies from gross haemodynamic instability and cardiac arrest [1] to gastrointestinal obstruction and respiratory insufficiency weeks or even many years later [2]. The involvement of two body cavities may cause problems in identifying the path of ballistic injury, deciding on the priority of intervention and limitation of contamination. Diagnosis is complicated by the lack of a single reliable radiological diagnostic modality and the frequent presence of concomitant multisystem injuries which direct attention away from the diaphragm.

Traumatic diaphragmatic injuries (TDI) have been recognised since the 16th Century, after death initially [3], with the first ante mortem report being described by Bowditch in 1853 [4]. The first successful repair of a penetrating diaphragmatic injury was credited to Riolfi in 1886 [5] and that of a blunt injury by Walker was reported in 1900 [6]. Thereafter several large case series have been published including 325 cases reported by Hedbloom [5] in 1925 and Hood's 428 cases in 1971 [7]. The mortality in Hood's series was 18% which he attributed to the diagnosis of TDI being frequently overlooked and the impact of the associated injuries.

TDI reflects transfer of energy across the diaphragm which may be a penetrating ballistic fragment, stab wound or a pressure gradient from compressive blunt trauma and by definition represents the possibility of visceral injury in either cavity; it is the likely associated visceral injury that will dictate the diagnostic and therapeutic approach to the casualty and contribute to the morbidity and mortality.

## Anatomy

The musculotendinous diaphragm separates the thoracic from the abdominal cavity. It arises embryologically from the pleuroperitoneal membranes and body wall, the dorsal mesentery of the oesophagus and the septum transversum. Defects in the development and fusion of these components give rise to the congenital hernias of Morgagni and Bochdalek which may predispose adults to traumatic diaphragmatic herniation. The muscle fibres arise radially from the margins of the thoracic aperture and converge into a large central tendon which is fused to the pericardium above. The posterior attachments are lower than those anteriorly and the right dome lies higher than the left. It is pierced at the crura by the passage of the inferior vena cava,

oesophagus and aorta from in front backwards. It is covered by parietal pleura and peritoneum except at the 'bare area' of the liver which lies immediately adjacent to the diaphragm itself. It receives motor and sensory supply from the phrenic nerves (C3-5 of the spinal cord). Aside from those structures that traverse it, the diaphragm's immediate anatomical relations are the pleural space, lungs, pericardium and heart above and the liver spleen, stomach and splenic flexure of colon inferiorly. An intact diaphragm is the most important muscle of respiration, performing up to 70% of the work of breathing at rest by creating a negative intrathoracic pressure during inspiration by contraction of the striated muscle which shortens and flattens the diaphragm, increasing intrathoracic volume and lowering intrathoracic pressure. The converse happens in expiration and this mechanism contributes to ensuring the venous return to the heart. Disruption of the mechanism coupled with compression of the IVC at the diaphragmatic hiatus by herniated intrathoracic abdominal viscera will significantly reduce cardiac venous return and may cause catastrophic cardiovascular collapse [8].

## Definitions

Previously a variety of terms such as diaphragmatic hernia, diaphragmatic injury or thoracoabdominal trauma have been used interchangeably, but it is now recognised that herniation of abdominal contents through a diaphragmatic injury occurs in less than 50% of injuries [9] and thus the two entities are not necessarily synonymous. In addition, diaphragmatic herniae may occur and be recognised at the time of diaphragmatic injury or be chronic, in which case the recognition of visceral herniation, but not necessarily its occurrence, is delayed.

## Epidemiology

It is difficult to accurately report the true overall incidence of diaphragmatic injury due to the high number of missed or delayed diagnoses and pre-hospital deaths [10]. A large US trauma database analysis reported 254 cases of TDI out of more than 20,000 data entries giving an approximate rate of just over 1.2% [11], and another trauma centre survey reports an incidence of 0.63% [12]. An 11 year Turkish review of over 12 000 trauma patients describes the incidences of TDI to be 0.4% of all trauma patients seen, 4.1% of all trauma admissions and 11% of trauma patients undergoing surgery [13]. An analysis focussing on thoracoabdominal trauma suggested rates of TDI of 2.1% for blunt trauma and 3.4% after penetrating trauma [10]; approximately 5% of all patients undergoing laparotomy or thoracotomy for trauma have a diaphragmatic injury [14,15]. Studies from heavily urbanised areas with a high incidence of violent crime predictably report a

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majority of penetrating TDI – over half the cases reported in one Nigerian study were from a penetrating mechanism [16], whereas in areas where violent crime rates are lower, such as the UK, TDI is attributable to motor vehicle collisions (MVC) in 80-90% of cases [17,18]. Three quarters of TDI are from blunt trauma [19] and three quarters occur on the left side, with 23% right sided injury and only 2% occurring bilaterally [12]. The incidence of TDI is increasing as the high energy MVCs increases and survival of other previously fatal injury complexes improves [20].

## Pathophysiology

Head-on impacts in a restrained conscious motor vehicle passenger will increase intra-abdominal pressure sufficient to generate a pressure gradient across the diaphragm greater than 1000cm of water, a tenfold increase in the pressure rise generated by coughing for example [21]. This pressure gradient is transmitted as kinetic energy through the domes of the diaphragm and solid organs typically creating long radial tears in the posterolateral aspect of the diaphragm – an embryological weakpoint surrounding the oesophageal opening; herniation of abdominal contents may follow. Lateral collisions, which are three times more common, cause ipsilateral tears secondary to thoracic distortion and shearing [21]. There is a significant association between thoracic aortic disruption and diaphragmatic injury in automotive deceleration trauma, with 10% having both [22]. Other commonly associated injuries in blunt TDI are head and long bone fractures as well as liver and splenic injury dependent on the side of trauma [23]. Comparing the collision details between those who suffered and did not suffer TDI after MVC shows that frontal or nearside compartment intrusion of more than 30cm or a change in velocity ( $\Delta V$ ) at the point of collision of more than 40 kilometres per hour are positively associated with TDI. Pelvic fractures and splenic injury were similarly associated but the sensitivity was low, whereas the specificity for thoracic aortic injury and TDI was 96.6%. Combining injury pattern and collision characteristics provided a better balance of sensitivity and specificity. Vehicle intrusion >30cm and splenic injury had a sensitivity and specificity for predicting TDI of 81% and 71% respectively, whereas  $\Delta V >40$ kph and splenic injury had values of 84% and 66% respectively [24].

Penetrating wounds are more likely to result in diaphragmatic injury than blunt trauma [25], but cause smaller sub-centimetre holes compared to the 5-15cm linear tears from blunt injury, which may go unrecognised until years later [21]. If there is insufficient force to push the abdominal organs into the chest at initial injury, gradual enlargement of the defect occurs over years and the pressure gradient between abdomen and thorax aids herniation later.

Left sided rupture is three times more common than on the right [19,26]; diagnosis is easier on the left as the liver may well plug the diaphragmatic defect making plain radiography diagnosis less likely [27]. Cadaveric studies demonstrate the pressure required to rupture the left hemidiaphragm is consistently lower than that on

the right [28], due to the relative weakness on the left from the lumbocostal trigone and the point of embryological fusion. As the energy transfer required to rupture the right side is higher, associated visceral injury is both more likely and more severe potentially contributing to the higher rate of prehospital death from right sided injury that has been reported [10], hence any study that omits post mortem data will underestimate the true incidence of right sided TDI. It has been thought that right sided injury has a higher overall mortality rate compared to left sided injury [11, 29]] but the evidence is conflicting with other large studies [30] and a logistic regression analysis of the 105 cases of TDI admitted over a 13 year period in Montreal found no difference in mortality between blunt or penetrating mechanisms of injury nor between left or right sided injury in the perioperative period [9].

Bilateral rupture is rare but on the increase due to the increase in energy transfer in MVCs [31] and carries a much higher mortality than single sided injury [11] which reaches 100% in some series with small numbers of bilateral injuries [30] reflecting the huge amount of energy transfer required for such an injury and the associated visceral injury in both cavities compounded by the physiological insult of markedly impaired cardiovascular function.

Independent predictors of mortality in various studies include an Injury Severity Score (ISS) of >15 [9] or increasing ISS [11], haemodynamic status and increasing age[11,29] or raised revised trauma score, need for thoracotomy or transfusion of more than 10 units of packed red cells [32]. It is unclear whether it is the dual cavity nature of the injury, the almost invariably associated visceral injuries, both of which will contribute to the high ISS, or the diaphragmatic injury itself that contributes to the poor outcome [9]. It has been hypothesized that previously repaired diaphragmatic injuries or congenital hernias are more prone to rupture, which also raises the importance of considering the presence of a pre-existing hiatus hernia before diagnosing traumatic injury [33].

## Clinical Features

Traumatic Diaphragmatic Injury is known for its 'both ends of the spectrum' presentation, but most patients with TDI will display some symptoms [34]; these may vary from minor ones such as pleuritic discomfort or abdominal cramps in uncomplicated injury to obstructive or ischaemic symptoms in those with incarcerated or strangulated hernias [2]. The physiological penalties of TDI have been described and may include dyspnoea, chest or abdominal pain and haemodynamic shock [35].

Bowditch described five cardinal signs of TDI [4] which were considered irrefutable for a long time until Grimes described three distinct phases of presentation of TDI [36] and their presentation and these descriptions (Box 1) are now more widely accepted. Bernatz et al [34] identified a fourth group with almost immediate haemodynamic compromise, not only due to hypovolaemic shock but by the tamponading effect of an intrathoracic mass.

**Acute Phase:** This is from the initial insult to apparent recovery from injury

- Abdominal Pain
- Other injuries (Chest/abdominal wall, pelvis, head, extremities, haemopneumothorax, abdominal viscera)
- Haemodynamic instability or lability
- Respiratory Distress
- Decreased air entry on affected side
- Auscultation of bowel sounds in chest (pathognomic)
- Chest x-ray abnormality
- Defect identified at emergency surgery (traditionally laparotomy)

**Latent Phase:** This occurs as intra-abdominal content traverses the defect into the thorax and may occur from hours to weeks after injury. It decreases the functional capacity of the thorax

- Upper gastrointestinal complaints
- Dyspnoea/cyanosis
- Tachycardia
- Substernal pain/referred shoulder tip pain
- Restlessness
- Dyspnoea exacerbated by lying flat
- Dull percussion note / decreased breath sounds on affected side
- Auscultation of bowel sounds in chest
- Abnormal chest x-ray findings

**Obstructive phase:** It occurs months to years after injury as the herniated viscera obstruct or strangulate. 85% of strangulations occur within three years of initial injury [37]

- Nausea/vomiting
- Symptoms of intestinal obstruction/ischaemia/haemorrhage
- Chronic respiratory difficulty secondary to atelectasis and resultant pneumonitis
- Mediastinal shift
- Borchardts triad (Upper abdominal pain and distension, retching and inability to pass an NG Tube) [19]
- Auscultation of bowel sounds in chest

Box 1 The three phases of traumatic diaphragmatic injury [36]

## Immediate Management

Immediate management is often resuscitative, due to the associated injuries, and should follow a recognised trauma management system, even in the absence of a diagnosed TDI. TDI hardly ever occurs in isolation with chest, splenic and hepatic injuries being the commonest concurrent injuries [24]. Maintenance of adequate oxygenation, ventilation and perfusion is essential to allow the best chance of recovery. Approximately 15% of TDI diagnoses are made more than 48 hours after injury due to necessary resuscitation and stabilisation preventing further investigation [19], and up to 65% may still be diagnosed at surgery [13].

## Investigation

Many aids to diagnosis have been suggested over the last 50 years with the plain chest radiograph being the earliest, most familiar and accessible. ‘Pathognomic’ signs of TDI are still contentious (Table 1) and the diagnosis of uncomplicated TDI remains difficult [30].

Hard Signs	Soft Signs
NG Tube visible in thorax	Irregularity of diaphragmatic outline
Hepatic displacement	Atelectasis of lower lobes
“Collar sign” of herniated abdominal viscus	Mediastinal shift
Bowel loops in chest	

Table 1 Hard and Soft signs of TDI on chest x-ray

The diagnostic accuracy of the plain chest x-ray is almost four times greater (62% v 17%) for left sided injury than right [38], but 20-50% of initial chest x-rays of patients with later confirmed TDI are reported as normal [12]. Diagnostic capability is increased by studying serial chest x-rays, especially in those mechanically ventilated as visceral herniation may only occur after intrathoracic pressure equalises with that in the abdomen [39]. Plain radiographic diagnosis of TDI depends on the recognition of fluid/gas levels in the thorax, which in the past has led to the deliberate injection of air and water into the peritoneum followed by an erect chest X-Ray (pneumoperitoneumography) [2]; diagnostic accuracy is not reported. Further modifications included the deliberate insertion of radio-opaque nasogastric tubes, the use of oral barium and screening fluoroscopy of diaphragmatic movement. These were largely superseded by the advent of computed tomography (CT). Plain film radiography in the trauma situation remains the immediate and accessible investigation which may demonstrate TDI; it is insufficiently accurate however to be used to exclude diaphragmatic injury in the presence of a suggestive mechanism of injury or clinical presentation. Introduction of an intercostal thoracic drain in the presence of acute diaphragmatic herniation is potentially dangerous and runs the risk of iatrogenic visceral injury [1]; atypical chest radiographs following acute thoracoabdominal injury (particularly on the left side) should prompt further imaging –preferably by CT if the patient is stable enough – prior to chest drain placement [1].

In healthy study participants, ultrasound is a reproducible method of assessing diaphragmatic movement [40] comparable to fluoroscopy [41], but few studies have specifically examined the role of ultrasound in diagnosing diaphragmatic injury in trauma. Focussed Abdominal Sonography for Trauma (FAST) may be extended to demonstrate abnormal or absent diaphragmatic movement suggestive of TDI. Blaivas et al described three patients in whom emergency department (ED) ultrasound identified decreased diaphragmatic movement with m-mode Sonosite FAST and subsequently had surgical repair of a diaphragmatic tear [42]. Mihos et al [38] claimed a diagnostic accuracy of 26% although other studies report a zero detection rate of TDI by ED ultrasound

[43].

Historically CT scanning has been regarded as poor at visualising the diaphragm due to movement artefact [44], but the advent of fast multislice spiral CT scanning with image acquisition in a single breath hold coupled with the availability of multiplanar reformatting of images has markedly improved its diagnostic ability for TDI [45]. It also allows rapid diagnosis of visceral injury in the polytrauma patient that may mask or detract attention from the diaphragmatic injury; the usual caveats regarding the transfer of the unstable patient to the CT scanner apply to suspected TDI as well as other trauma. Spiral CT has increased the sensitivity of detection of TDI from as low as 17% to approximately 80%, with a specificity of 100% [46]. A variety of radiological CT signs have been described to aid the CT diagnosis of TDI (Table 2).

Magnetic Resonance Imaging provides excellent images of the soft tissues especially around the diaphragmatic hiatus and is accurate in diagnosing diaphragmatic injury [52] but the practicalities of MR scanning an unstable intubated patient prohibit its widespread use acutely. Its main applicability is in the assessment of chronic diaphragmatic herniation [47].

Sign	Explanation
Diaphragmatic discontinuity	An obvious break in the smooth contour of the hemidiaphragm
Collar sign	Waist like constriction of bowel and mesentery which has herniated through a diaphragmatic defect [47].
Hump sign & Band sign	Two variants of the collar sign were apparent on high-quality sagittal and coronal reformations. The first, termed the "hump sign", describes a rounded portion of liver herniating through the diaphragm forming a hump-shaped mass, and the second, termed the "band sign," is a linear lucency across the liver along the torn edges of the hemidiaphragm [48].
Dependent Viscera Sign	The dependent viscera sign is seen with diaphragmatic rupture. The absence of posterior support by the diaphragm allows viscera to “fall” against the posterior ribs to a dependent position [49].
Diaphragmatic thickening	Thickened leaves of the diaphragm in comparison to the other side because of either retraction of the leaves of the ruptured diaphragm or muscular haematoma [50].
Dangling Diaphragm	The torn edge of the diaphragm curls inwards away from its normal parallel course with the abdominal wall. It has a sensitivity of 54% and specificity of 98% [51].

Table 2 CT signs of TDI

## Surgical Management

The surgical management of TDI may be diagnostic or therapeutic, open or minimally invasive and be via the chest or abdomen and the choices made largely reflect the experience and facilities of the individual trauma units and surgeons.

Ref.	Duration of Study (years)	Mechanism & Side of Injury			Cavity Used for Repair			Mortality Rate (%)
		Blunt (Left/Right/Bilateral)	Penetrating (Left/Right/Bilateral)	Unspecified (Left/Right/Bilateral)	Abdomen <sup>s</sup>	Chest	Both	
[11]	13	99 (58 / 17 / 5)	155 (71/61/4)		161 (4)	8	21	22*
[13]	11	26	25	40 / 10 / 1	51			31 / 8 <sup>s</sup>
[29]	9	41 (24 / 15 / 2)			22	10	4	16
[30]	9	44 (30 / 12 / 2)			4	37	3	13
[35]	10	24	5	23 / 6 / 0	8	20	1	13.8
[53]	3		22 (20 / 1 / 1)		22			0
[54]	8		60 (18 / 42 / 0)		56 (1)	3		6.7
[55]	4	12 (9 / 2 / 1)			11		1	25
[56]	11	12 (11 / 1 / 0)	3 (1 / 2 / 0)		0	11	4	6.7
[57]	10	6	4	7 / 3 / 0	5	2	3	10
[58]	11	17	51	57 / 11 / 0	61	5	1	16.2

**Table 3 Injury characteristics, operative technique and outcome from a selection of recent case series of TDI.**

\*54/254 did not survive to undergo surgery; mortality figure is for those who survived to surgery; <sup>s</sup>All by laparoscopy except number of laparoscopic repairs in brackets; <sup>M</sup>Mortality rate 31% for blunt injury and 8% for penetrating TDI

### Which Cavity?

Review of recent series of thoracoabdominal trauma with diaphragmatic injury (Table 3) demonstrates the distribution of penetrating and blunt, right and left diaphragmatic injury. It demonstrates that the cavity of approach of choice is heavily influenced by the reporting surgeons speciality; units in whom the thoracoabdominal trauma is managed by the thoracic surgeons report much higher rates of thoracotomy [30, 35, 56] than series reported by general surgeons [1,11,57,58] although the use of VATS to assess the integrity of the diaphragm by Freeman et al [54] before converting to laparoscopy for repair is an interesting divergence from this.

The operative technique is straightforward. Any hernia is reduced and the injured diaphragm debrided back to healthy muscle, concomitant visceral injury attended to and the diaphragmatic defect repaired. Direct suture with interrupted or running sutures using absorbable or non-absorbable sutures will normally suffice [57]. One study with small numbers of completed follow up has suggested that the recurrence rate may be slightly higher with absorbable sutures reporting 1/13 recurrences after a mean follow up of 4 years – a further case recurred on the first postoperative day after repair with absorbable sutures, but this is likely to represent a technical rather than suture failure so early after placement [9]. Large defects can be spanned [59] or reinforced [60] by prosthetic mesh and a single report has detailed the use of gelatine matrix to rapidly seal an iatrogenic diaphragmatic defect with omentum [61].

There appears to be no evidence of superiority of one cavity approach over the other in haemodynamically stable patients. The need for laparoscopy is generally directed by the presence of intra-abdominal visceral pathology which is difficult to attend to through the chest. It has been suggested that acute right sided injuries and chronic hernias be approached through the right chest and left sided injuries should be approached through the abdomen [62]. Peer et al [35] from India recommend similar guidelines – thoracotomy for delayed presentations or acute presentations in whom concomitant abdominal injury has been excluded. Waldschmidt and Laws [63] reported 86 patients – the majority were treated by laparoscopy and only 1/65 need an additional thoracotomy compared to seven out of 15 initial thoracotomies needing an additional laparoscopy. Surgery for chronic

diaphragmatic hernia with abdominal viscera in the chest has traditionally been approached via the chest as the intrathoracic adhesions are difficult to deal with from the abdomen [35, 55,64] but Clarke et al [1] report six chronic diaphragmatic hernias repaired abdominally without recourse to thoracotomy.

The issue of which cavity to open first becomes more critical in the haemodynamically unstable patient and has been examined for penetrating thoracoabdominal injury by Asensio and colleagues who specifically discuss the sequencing of thoracolaparotomy rather than the management of TDI per se [65]. In a 4 year period they examined the charts of 254 patients admitted with penetrating thoracoabdominal injury - 73 required thoracolaparotomy. If the initial cavity procedure had to be interrupted to access the other cavity because of continued haemodynamic deterioration this was classed as incorrect sequencing. When laparoscopy was performed first 18/34 (53%) were interrupted compared to 14/39 (36%) when the chest was opened first. The commonest reason for opening another cavity was persistent hypotension unexplained by the findings in the first cavity (18%) and erroneously high chest drain outputs thought to be from significant intrathoracic haemorrhage but actually originating from abdominal visceral injury and traversing an injured diaphragm (10%). Overall, nearly half of the thoracoabdominal procedures were incorrectly sequenced and the mortality for these two cavity procedures was almost double that of patients in whom only one cavity was opened (31% v 59%).

### Minimally Invasive Surgery

#### For Diagnosis

Minimally invasive surgical techniques may be used as a diagnostic or therapeutic tool and as in open surgery may be applied via the chest or abdomen. Diagnostic thoracoscopy for TDI was first described in 1976 [66] in 11 patients with penetrating wounds of the left chest; in six the diaphragm was visualised clearly, in two of which a clinically unsuspected TDI was revealed. Four other patients with diaphragmatic integrity confirmed were able to be managed conservatively. The corresponding first report of diagnostic laparoscopy came from Adamthwaite in 1984 [67] who laparoscoped 10 patients (8 acute and 2 chronic cases); he confirmed diaphragmatic injury in eight cases which seem to have

then undergo repair at laparotomy, two patients avoided unnecessary laparotomy after negative laparoscopy.

Murray et al laparoscoped 110 patients with penetrating injury to the left chest (94 stabs, 16 GSWs) who were haemodynamically stable and without other indication for laparotomy [68] within 6 hours of injury. Twenty six (24%) had occult diaphragmatic injuries, 22 of whom went on to have open repair to ensure there were no missed enteric injuries (which there weren't). Friese et al [69] confirmed laparoscopy to be both specific and sensitive in detection of TDI (100% and 87.5% respectively) in a series of 34 asymptomatic haemodynamically stable penetrating thoracoabdominal injury patients by performing mandatory laparotomy (or thoracoscopy in four cases) following diagnostic laparoscopy. Powell et al reported a rate of diaphragmatic injury of 20% at diagnostic laparoscopy following penetrating thoracoabdominal injury, two thirds of whom had a normal chest radiograph; all were repaired at laparotomy [53].

In the largest series of diagnostic thoracoscopy after unilateral penetrating chest injury [54] – in patients who were haemodynamically stable and without other indication for laparotomy or thoracotomy - 60/171 (35%) had diaphragmatic injuries. Comparison between those with and without TDI identified five factors to be predictive of TDI after penetrating chest injury: haemo- or pneumothorax on admission chest x-ray, associated intra-abdominal injuries on imaging, a chest entrance wound inferior to nipples or scapula, a high velocity mechanism of injury and a right sided chest injury. Nearly all (93%) were repaired at laparotomy.

### Operative Technique

For laparoscopy, the patient is positioned as for a trauma laparotomy in the supine crucifix position to allow access to the chest. After open umbilical intubation, pneumoperitoneum is established slowly to avoid a catastrophic decrease in venous return or significant increase in ventilation pressure because of free communication between chest and abdomen. Two working ports are used at the level of the umbilicus lateral to the midclavicular lines. The anterior diaphragm is easily visualised but retraction of the stomach, liver and spleen and lateral tilting of the table may be necessary to visualise the posterior portions. For thoracoscopy, the patient is positioned full or partial lateral with thoracic support and the ipsilateral arm flexed 90° at the shoulder and supported in a stand. Double lumen endotracheal intubation is recommended to allow lung deflation; the table is broken in a 30° jackknife to widen the intercostal spaces and a 2cm port incision placed in the 4th or 5th intercostal space for insertion of the camera. Subsequent working ports are placed in the same intercostal space as necessary to allow easy conversion to thoracotomy if necessary.

### For Therapeutic Intervention

Over a four year period Matthews et al [59] attempted laparoscopic repair of 17 TDIs (eight acute and nine chronic cases). The acute cases were selected for attempted laparoscopic repair on the grounds of haemodynamic stability, negative radiological workup and a laparoscopy negative for other injuries prior to attempting repair. Two acute and two chronic TDIs were converted due to proximity to the oesophageal hiatus or pericardium. Three large chronic defects (greater than 3x6cm) were closed using prosthetic patches, the remainder were closed directly. The mean operative time was 137 minutes and there were no recurrences at a mean of 8 months. Experimental data confirms that for small defects (2cm) laparoscopic direct closure by suture or staple or patch repair are all equally efficacious in terms of later burst pressure and equivalent to open direct suture repair [70].

Two thirds of the 52 haemodynamically stable penetrating

thoracoabdominal injuries admitted in Guatemala city in one year had TDI on thoracoscopy – all were closed by direct VATS suture with a zero mortality and recurrence rate at 12-24 month follow up [71]. The authors recommend it as the diagnostic procedure of choice in haemodynamically stable penetrating chest trauma victims as it allows the evacuation of the retained haemothorax that occurs in up to two thirds and which may not be visible on plain radiography. It is of note that all VATS procedures were performed at least 24 hours after admission by which time significant intra-abdominal injury would have disclosed itself.

Shaw et al [72] reported a hybrid laparoscopic-assisted technique utilised in 25 cases of TDI in whom there was no indication for laparotomy after a 24 hour period of serial observation. After diagnostic laparoscopy and identification of an isolated diaphragmatic injury a 4cm subcostal incision and body wall hooks were used to allow open suture repair of the defect. This technique was successful in 24/25 cases – the failure being due to poor diaphragmatic visualisation due to dilated bowel.

The concern that an acute VATS repair of TDI risks missing an abdominal injury has been partially addressed by one report of an acute VATS suture repair of a 10cm diaphragmatic tear followed by diagnostic laparoscopy confirming no abdominal visceral injury [73]. Alternatively on the left side, low grade splenic injuries may be amenable to VATS treatment [74].

## Diaphragmatic Injuries in Children

The data are limited but it appears that the salient features of TDI in adults applies to children as well, although blunt trauma predominates in children [75,76] and the major determinant of mortality is the concomitant injuries rather than the TDI itself. Soundappen et al [75] reported 8 cases (all blunt) over a 14 year period with a 25% mortality both of whom died from associated major vessel injury and Ramos et al 15 cases in an 11 year period. Most (13/15) were blunt injuries, five (33%) died of their injuries [76].

## Conclusions

Traumatic diaphragmatic injury requires a high index of suspicion for timely diagnosis and missed diagnosis may present years later with life threatening consequences. Missed injuries are commoner on the right side. No imaging modality is ideal – the initial chest radiograph may be normal, or be misinterpreted as normal and CT scanning requires high quality reconstructions to improve diagnostic accuracy. Surgery remains a common method of diagnosis unsuspected TDI and both diaphragmatic domes must be scrupulously inspected at laparotomy. In the stable patient without other indication for intervention, diagnostic laparoscopy or thoracoscopy should be considered after thoracoabdominal trauma to inspect the diaphragms. Repair may be performed by a minimally invasive technique via the chest or abdomen dependent on an individual surgeon's preference as long as concomitant visceral injury in the other cavity has been excluded. In most case series there is a high mortality from TDI, which is almost always due to the associated injuries acquired in a high energy transfer injury mechanism.

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# Anaesthetic and Critical Care Management of Thoracic Injuries

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## Abstract

Thoracic wounding has been a relatively common presentation of military wounds throughout modern conflict. When civilian casualties are included the incidence has remained constant at around 10%, although the frequency and severity of wounds to combatants has been altered by modern body armour. Whilst thoracic injury has a high initial mortality on the battlefield, those surviving to reach hospital frequently have injuries that only require simple management. In addition to penetrating ballistic injury, blunt chest trauma frequently occurs on operations as a result of road traffic collisions or tertiary blast injury. The physiological impact of thoracic wounds, however, is often great and survivors often require intensive care management and, where available, complex strategies to ensure oxygenation and carbon dioxide removal. This review examines the incidence and patterns of thoracic trauma and looks at therapeutic options for managing these complex cases.

## Introduction

Thoracic wounding remains common on current military operations. This is predominantly ballistic penetrating trauma but there is also a background incidence of blunt injury from tertiary blast injury and road traffic collisions.

Previous editions have discussed penetrating cardiac injury [1], non-cardiac thoracic trauma [2] and emergency department thoracotomy [3]. This review discusses the incidence and pathophysiology of chest injury with particular emphasis on the anaesthetic and critical care considerations from point of wounding through the resuscitation room, operating theatre and later phases of care in the intensive care unit.

## Background

Penetrating wounds to the thorax occur frequently during war fighting operations and these injuries are often fatal before the casualty reaches medical care. However, those casualties that do enter the medical chain usually only require conservative management and have good outcomes. Throughout the history of conflict, chest trauma has carried a high probability of death. This changed little from World War I where the mortality from all penetrating thoracic injuries was 74% through to Vietnam where a single assault rifle gunshot wound to the chest had an 80% mortality [4]. Throughout that period thoracic wounding rates remained relatively static at around 10% of casualties. Data from recent conflicts shows that despite improvements in protection for troops such as Combat Body Armour (CBA), chest injuries still occur. Wounding rates for US forces during Op Iraqi Freedom in 2003 were 5% [5], but the rate of thoracic injury amongst all presentations during the conflict, when civilians were included, was higher at 12.7% [6]. Thoracic injury remains common during the conflict in Afghanistan with approximately 13% of ballistic injuries requiring a thoracic intervention (chest drain, soft tissue

debridement or thoracotomy) [7] and contributing to 30% of combat deaths [8].

## Pathophysiology and Specific Injuries

The thorax is a semi-rigid structure which affords protection to the lungs, heart and great vessels. Injury to these structures therefore typically requires a significant magnitude of force. The injury may be either blunt or penetrating. Blunt injuries tend to be managed conservatively or with the placement of an intercostal drain, whereas penetrating injuries frequently necessitate thoracotomy during initial resuscitation and often require operative intervention [9].

Chest injury leads to hypovolaemia secondary to major organ or vessel injury which is usually rapidly fatal, or hypoxia as a result of disruption of the mechanics of ventilation (Figure 1). A combination of hypoxia and reduced cardiac output can occur as a result of cardiac tamponade or tension pneumothorax. This is a far more common problem following penetrating trauma. Both of these conditions are potentially reversible but rapidly fatal if unrecognised.

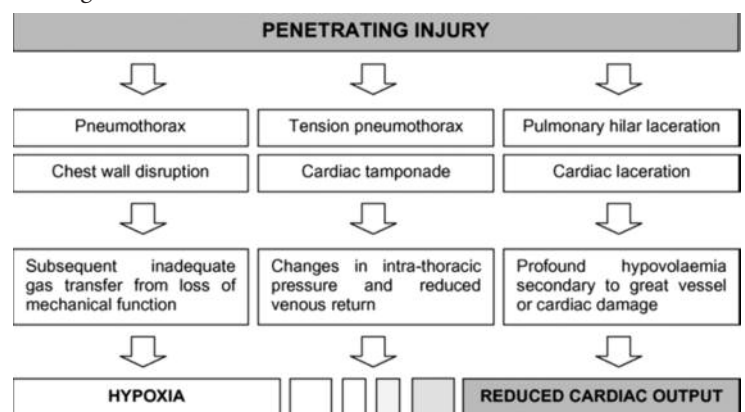


Figure 1. Pathophysiology of penetrating thoracic injury. Reprinted from *Injury Vol 37 (1)*. Hunt P, Owens A, Greaves I. Emergency thoracotomy in thoracic trauma – a review. Pages 1-19. Copyright (2006) with permission from Elsevier

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### Intrathoracic Airway Injuries

Tracheobronchial injuries are found in 0.8% of blunt thoracic trauma victims presenting for emergency surgery [10]. The vast majority of these injuries are found within 2.5 cm of the carina [11] and are associated with a high mortality due to difficulty in ventilation and maintenance of adequate oxygenation and delay in diagnosis [11,12].

The management of intrathoracic airway injuries should ideally involve a thoracic surgeon at an early stage as operative repair will usually be required [12]. This is often not the case on deployed operations; however there is evidence to suggest that conservative management can obtain good results in carefully selected stable patients. In a study of 20 patients with tracheobronchial injuries that were managed conservatively, four died. The authors concluded that surgery should be performed in cases of associated oesophageal injuries, progressive subcutaneous or mediastinal emphysema, severe dyspnoea requiring intubation, difficulty with mechanical ventilation, pneumothorax with air leak through chest drains, or mediastinitis. The remaining cases are likely to do well with conservative medical management [13].

The aim of initial management should be to improve ventilation and reduce air leak. This can be achieved by placing a cuffed airway device distal to the site of injury typically with the use of a fibre-optic bronchoscope. Alternatively a rigid bronchoscope may be used to facilitate this.

### Cardiac Injury

Cardiac injury can occur secondary to blunt or penetrating trauma. Blunt cardiac injuries can present across a spectrum ranging from isolated ECG abnormalities to myocardial rupture, with the right ventricle and interventricular septum the most frequently involved [14]. Cardiogenic shock may ensue as a result of arrhythmias, structural damage or impaired ventricular contractility.

ECG abnormalities that may indicate cardiac injury include ST segment changes and arrhythmias. These patients should have continuous ECG monitoring established. If haemodynamic instability is manifest, a trans-thoracic or trans-oesophageal echocardiogram (TOE) should be performed. Other imaging may include coronary angiography and nuclear medicine scans [15]. Measurement of troponin levels is probably of little benefit in management of blunt cardiac injury [16]. In the advent of cardiogenic shock, consideration should be given to the placement of an Intra-Aortic Balloon Pump (IABP) in order to off-load the left ventricle [17].

Behind armour blunt trauma (BABT) is a non-penetrating injury resulting from the deformation of body armour after sustaining a ballistic impact. Gryth et al have shown in animal models that apnoea occurring after BABT is a vagally-mediated reflex that results in severe hypoxia. They recommend that supportive ventilation should begin immediately in BABT casualties who are unconscious and apnoeic [18].

Penetrating cardiac injuries are typically fatal. Only 6% of patients with penetrating anterior chest wounds causing cardiac injury survive to reach hospital [19]. Presentation may frequently be the signs of cardiac tamponade which are classically Beck's triad of; hypotension muffled heart sounds and distended neck veins. Typically a globular heart is seen on chest x-ray although in practice this is a subtle sign (Figure 2). Cardiac tamponade has been reported with low energy ballistic wounds by Cooper et al [20]. Current practise of performing a rapid Focussed Abdominal Scan for Trauma (FAST) should include examination of the pericardium via the substernal window. Management is by needle pericardiocentesis or thoracotomy, with the latter being preferable and necessary for definitive treatment. Patients in extremis are candidates for emergency resuscitative thoracotomy. This has been defined in two reviews on the topic as those with vital signs absent for less than 10 to 15 minutes [3, 21]

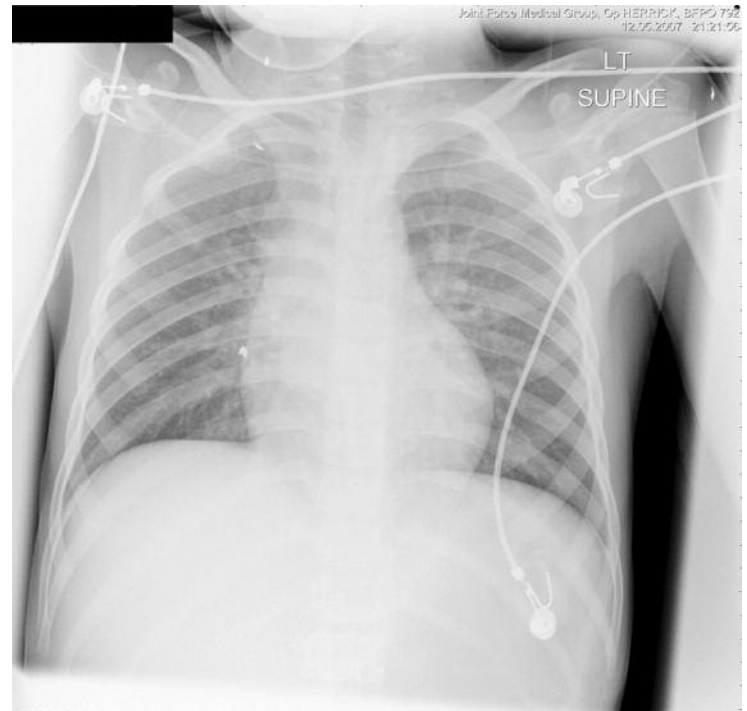


Figure 2. Chest x-ray showing fragments of land mine at right sternal edge and the globular heart of cardiac tamponade

### Aortic Injuries

Blunt aortic dissection is the second most common cause of death after head injury in blunt trauma [22]. Diagnosis can be difficult and a high index of suspicion should be maintained. Computerised Tomography (CT) and TOE are invaluable in establishing the diagnosis. Increasingly Endovascular Stent Grafting (EVSG) is being used as opposed to the conventional open aortic repair [21]. Neither of these options are likely to be available in forward surgical locations. Intra-operatively it is essential to maintain good blood pressure control. Glyceryl Trinitrate and Beta Blockers should be used to keep the systolic pressure less than 140 mmHg in order to minimise further aortic dissection.

Penetrating aortic injuries are often rapidly fatal. Early decision making and prompt thoracotomy with proximal control of the aorta is essential. Resuscitation with permissive hypotension should be instituted until control is established, with correction of coagulopathy, acidosis and hypothermia.

### Lung Injury

The pathophysiology of lung injury whether blunt or penetrating, can be seen as a two-fold process with direct injury to the lung parenchyma and a systemic inflammatory response causing alveolar-capillary changes. This leads to Acute Lung Injury (ALI) or Acute Respiratory Distress Syndrome (ARDS). ALI is defined by following features: an acute onset, bilateral infiltrates on chest radiograph, pulmonary artery occlusion pressure <18 mmHg, PaO<sub>2</sub>:FiO<sub>2</sub> <40 kPa. ARDS is defined by the same criteria except PaO<sub>2</sub>:FiO<sub>2</sub> is <27 kPa. These conditions represent a spectrum of increasing severity of lung dysfunction. Excessive bronchial secretions predispose to lobar collapse and decreased lung compliance with pneumonia ensuing in approximately 50% of cases. The end result is a significant ventilation/perfusion mismatch with the knock-on effect of decreased oxygen delivery to vital organs [23].

Blast lung results in an extreme form of pulmonary contusion. Shock waves from the blast cause both intra-alveolar and intra-bronchial haemorrhages with a sudden increase in lung weight [24, 25]. The haemorrhage decreases lung compliance and leads to severely impaired alveolar gas exchange and a rapidly worsening ventilation/perfusion mismatch.

Management of traumatic lung injury is supportive. It should aim to minimise the Systemic Inflammatory Response Syndrome (SIRS) and its progression to ALI/ARDS. This is achieved by utilising haemodynamic monitoring to avoid excessive fluid overload or profound hypovolaemia. The treatments indicated may include the use mechanical ventilation, crystalloids and colloids, diuretics and inotropes [26].

## Principles of Anaesthesia

The patient should be initially assessed and managed in accordance with Advanced Trauma Life Support or Battlefield Advanced Trauma Life Support in the deployed UK military setting.

Broadly speaking, the principles of thoracic trauma anaesthesia involve the restoration of circulating volume, maintenance of adequate oxygenation and correction of hypothermia and coagulopathy.

It is highly likely that a definitive airway will need to be secured at an early stage. Usual practice to achieve a definitive airway is a rapid sequence intubation (RSI) of the trachea with cricoid pressure applied and the cervical spine controlled with manual in-line stabilisation. This can increase the likelihood of difficult intubation occurring. Difficult intubation should be dealt with in accordance with the Difficult Airway Society (DAS) algorithms [27]. Any unexplained hypotension and difficulty in ventilation should arouse suspicion of a tension pneumothorax. A retrospective analysis of 978 penetrating chest trauma casualties during the Vietnam war found radiographic evidence of tension pneumothorax in 198 of these. They concluded that tension pneumothorax was the cause of death in 3 - 4% of fatally wounded combat casualties [28].

Emergency thoracotomy may be indicated and anaesthesia for this may include one-lung ventilation (OLV). OLV is most frequently achieved by means of a Double Lumen Endobronchial Tube (DLEBT). Alternatives to the DLEBT in providing OLV include bronchial blockers and the Univent tube [29]. It is recognised that DLEBT can be difficult to place by those not regularly undertaking routine thoracic anaesthesia and the tubes are commonly associated with malposition and a high complication rate. This can be compounded in the RSI situation [30]. In the authors' experience, most emergency thoracotomies carried out as part of damage control resuscitation can be managed on a single lumen tube and two lung ventilation. One lung ventilation (at least for surgery on the left lung) can be easily established by advancing a single lumen tube beyond the carina and into the right main bronchus.

In the shocked patient Ketamine is the induction agent of choice due to its preservation of blood pressure and cardiac output [31]. Hypotension immediately post-induction should be anticipated and treated with further intravenous fluid resuscitation (usually blood products) or sympathomimetic drugs in a euvoalaemic patient. Anaesthesia is usually maintained with a low dose volatile agent and non-depolarising muscle relaxation. The use of nitrous oxide should generally be avoided as it has a propensity to increase the size of gas filled cavities including air emboli and pneumothoraces. Monitoring should include invasive arterial blood pressure measurement as well as placement of a central venous catheter.

## Principles of Analgesia

The ongoing management of these casualties hinges on providing excellent analgesia; this will allow weaning from mechanical ventilation and hence restoration of normal respiratory mechanics. Local anaesthetic techniques have the benefit of avoiding the respiratory depressant side effects of opiates. However, regional techniques to provide adequate analgesia for significant thoracic injuries are relatively complex. Thoracic paravertebral blocks are attractive in that they have no unwanted effects on the uninjured side, are safe to perform, relatively easy to learn and provide analgesia comparable to that of epidural analgesia [32].

Paravertebral blocks can also be placed under direct vision, by the surgeon at thoracotomy, as described by Sabanathan [33]. Thoracic epidurals however are likely to be the technique of choice for most anaesthetists. These blocks provide bilateral analgesia but also muscle weakness, however the improvements in respiratory mechanics resulting from excellent analgesia more than offset this [34].

## Critical Care Management

A significant number of these patients will need critical care, including mechanical ventilation, after the initial resuscitative phase and/or post surgical intervention. Sputum retention is a major issue leading to pulmonary collapse with the increased risk of infection. Early pneumonia, within the first few days, may well be a result of aspiration. Chest injury, shock, emergency intubation and blood transfusion are all risk factors for development of later ventilator associated pneumonia (VAP) and sepsis [35]. Good intensive care nursing and the application of ventilator care bundles - specifically adopting a 30° head up posture for the patient and daily sedation breaks - can reduce the incidence of VAP [36, 37]. Sepsis remains the major cause of late deaths in the victims of trauma.

## Ventilation Strategies

The concept of ventilator - associated lung injury has changed the way that mechanical ventilation is delivered. The Acute Respiratory Distress Syndrome Network study used tidal volumes of 6 and 12 ml/kg to ventilate patients with ARDS. A significant absolute reduction in mortality was achieved using the lower tidal volumes and maintaining inspiratory plateau pressures less than 30cm H<sub>2</sub>O. However these protective ventilation strategies frequently result in hypercapnia and respiratory acidosis [38]. When conventional methods of mechanical ventilation fail in those with an asymmetric lung injury, it is likely that the intensive care doctor will resort to methods such as independent lung ventilation to maintain oxygenation.

One lung independent ventilation (OL-ILV) is a technique which allows ventilation of one lung while the other main bronchus is artificially blocked in order to isolate fluid such as blood or secretions, avoiding contamination of normal lung alveoli and improving gas exchange. OL-ILV can be facilitated by deliberate left or right main bronchus intubation with a normal endotracheal tube, the use of a DLEBT or placement of a bronchial blocker.

Two lung independent lung ventilation (TL-ILV) allows different ventilatory parameters and/or ventilatory modes to be applied to each lung. Separate ventilator circuits are used for each lung. TL-ILV can be applied synchronously or asynchronously. Synchronous TL-ILV maintains the same respiratory rate for both lungs but the flow rates, tidal volumes and positive end expiratory pressure (PEEP) are set separately. Asynchronous TL-ILV must use two separate ventilators to deliver different modes as well as different ventilator settings.

## Other Strategies

There are occasions when clinical deterioration continues despite standard intensive care therapies. There are numerous other techniques with a limited evidence base that may be employed.

### Nitric Oxide

Nitric oxide induces pulmonary vasodilatation in un-injured areas of the lung, thus favourably altering the ventilation/perfusion ratio. Johannigman et al demonstrated the greatest improvement in pulmonary function when nitric oxide was delivered to either normal lung or to both lungs (injured plus un-injured) [39]. The use of inhaled nitric oxide may also be useful in patients with bilateral lung injury consisting of multiple patchy contusions and acute respiratory failure.

### Prone Positioning

Prone positioning has been extensively used in ALI/ARDS patients. The postulated mechanism is one of change in regional distribution of ventilation and perfusion in the lung [40]. Despite the improvement in oxygenation seen in the majority of patients, there is no proven improvement in mortality.

### High Frequency Oscillatory Ventilation

High Frequency Oscillatory Ventilation (HFOV) was initially used to treat neonates suffering from respiratory failure. It has been used in adults to treat refractory hypoxaemia. It delivers low-amplitude proximal airway vibrations that result in sub-dead space tidal exchanges. The airway pressure, inspired oxygen fraction and oscillatory frequency are titrated to achieve adequate oxygenation and ventilation.

### Extracorporeal Membrane Oxygenation (ECMO)

ECMO may be used as a temporary replacement for the lungs in those situations where the lungs catastrophically fail to provide ventilation and oxygenation, after all other treatment strategies have failed. The aim of ECMO is to give the lung increased time to recover. It employs veno-venous or veno-arterial large bore cannulation (usually only veno-venous for oxygenation purposes) and a pump mechanism with a membrane oxygenator. The CESAR trial compared ECMO with conventional ventilatory strategies in patients with ARDS. They found a significant improvement in survival in the ECMO group but a longer hospital and intensive care stay [41]. There were, however, several problems with this study – all the patients in the ECMO group went to one centre whereas the standard group stayed in a specialist ICU. There was no set ventilatory strategy used in the non-ECMO group and a number of patients did not receive optimal ventilatory management, whereas most of those in the ECMO group did. ECMO remains a highly specialised technique that is only available in a few centres; there is just one adult ECMO centre in the United Kingdom.

### Extracorporeal Carbon Dioxide Removal

Lung protective ventilation strategies frequently lead to hypercarbia and acidosis. This often limits the ability to apply strict low volume, low pressure strategies. One technique that has shown promise in military casualties is extracorporeal carbon dioxide removal [42]. These devices use heparin bonded circuits, negating the need for anticoagulation, and arterio-venous cannulation, rather than needing a pump. The use of this device has also been reported in the retrieval of patients with ARDS [43].

### Partial Liquid Ventilation

The use of a perfluorocarbon solution to ventilate and oxygenate the lung has been postulated. There is limited evidence to support its efficacy. One small scale study comparing ECMO plus partial liquid ventilation to ECMO alone showed a greater improvement in lung compliance and reduction of physiological shunt in the liquid ventilation group [44]. The theoretical benefits of partial liquid ventilation stem from the perfluorocarbon allowing free diffusion of O<sub>2</sub> and CO<sub>2</sub> as well as acting like surfactant and increasing alveolar surface tension [23]. The benefits of perfluorocarbon liquid ventilation remain theoretical. Trials in respiratory failure patients have not demonstrated a significant improvement.

### Recombinant Factor VIIa (rFVIIa)

The use of recombinant activated factor seven in overcoming complex trauma-related coagulopathy and as an adjunct to surgical haemostasis has been described both anecdotally and in several studies [45, 46]. Theoretically, the administration of rFVIIa may rapidly control the pulmonary haemorrhage associated with blast lung. Prompt control of this haemorrhage may improve the

ALI/ARDS picture and avoid the need for mechanical ventilation. The Israeli military has reported full recovery in soldiers with life-threatening blast lung treated with rFVIIa [47]. This indication for rFVIIa remains controversial and off-licence, more studies are required to prove its efficacy in this situation.

## Conclusion

Thoracic injuries and especially those sustained in the military setting, present a major challenge to the surgeon, anaesthetist and intensivist. To compound this challenge, the initial management of these wounds in the deployed military setting will usually be carried out by non-thoracic surgeons and anaesthetists in an austere environment.

### The essentials for dealing with these injuries are:

1. Recognise the life threatening problems and intervene accordingly
2. Understand the prevailing pathophysiology
3. Adopt a multidisciplinary approach to provide care from point of wounding through resuscitation and surgery to intensive care.

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