REVIEW

Tension pneumothorax—time for a re-think?

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This review examines the present understanding of tension pneumothorax and produces recommendations for improving the diagnostic and treatment decision process.

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Tension pneumothorax (TPT) is an uncommon disease with a malignant course leading to death if untreated.[1 2] It is most commonly encountered in prehospital trauma care, emergency departments, and intensive care units (ICUs).[3] Resuscitation and trauma courses usually illustrate a patient in extremis and assume that the clinical diagnosis is straightforward and the response to needle chest decompression is rapid and reliable.[4 5] However, this might not be the case in real life. Texts differ when describing the diagnostic symptoms and signs[5–9] and there are several case reports of diagnostic difficulty or missed diagnosis because of an absence of ''classic'' signs.[1 10 11] Lack of chest signs along with poor correlation between the signs present and those picked up by experienced physicians have been specifically noted.[12–14] There have also been multiple reports of ineffective needle decompression with adequate treatment only being achieved with tube thoracostomy.[15–22]

This review examines the present understanding of tension pneumothorax and produces recommendations for improving the diagnostic and treatment decision process.

METHODOLOGY

An electronic search was performed without use of filters, on Medline (1966–2003), (once with Pubmed and once with Ovid interface). The terms ''pneumothora$/*'' (PNEUMOTHORAX) and ''tension$/*'' were searched separately, then combined to identify relevant papers, which once obtained were hand searched, with further references being obtained from these. Original papers were systematically reviewed for pathophysiology, predefined clinical features, and treatment options. Both authors performed this process independently, writing separate reviews, which were then collated to achieve a consensus article.

TENSION PNEUMOTHORAX—DEFINITION

TPT may be said to occur when a one way valve communicates with the pleural space,[5 23] but this in fact describes an injury with the potential to tension. We found a variety of definitions of tension pneumothorax (box 1).

Definitions using intrapleural pressure (IPP) are more accurate than clinical definitions and the definition of ''positive IPP throughout the respiratory cycle'' was used in early animal experiments. However, in the awake subject, the IPP must be less than atmospheric pressure during part of the respiratory cycle if the pneumothorax is to continue to develop, as this will only occur if air continues to enter the pleural cavity. This led to the definition of an ''expiratory tension pneumothorax.''[33 34] It is therefore important to consider that in any TPT (in an awake subject) there is a range from an IPP that is only just positive at end expiration to an IPP that is positive throughout the respiratory cycle.[35]

In some definitions supra-physiological IPP occurs before the defined TPT exists. This may have limited clinical relevance, however it makes the literature difficult to interpret as papers may be referring to different stages in development of the condition.

INCIDENCE

The incidence of TPT varies with the population studied and is not well established—often reflecting disease suspicion rather than true incidence. TPT was confirmed (hiss of air on decompression) in 5.4% of major trauma patients, (64% of whom were ventilated) treated by prehospital care doctors in London.[36] Other studies have reported needle decompression rates (as a surrogate for potential TPT) in the prehospital environment varying from 0.7% to 30%.[32 37 38]

Earlier postmortem studies on patients dying in ICUs show rates of undiagnosed TPT ranging from 1.1% to 3.8%.[25 26] Missed diagnosis was more probable if ventilation or cardiopulmonary resuscitation had occurred.

In ventilated patients TPT is more likely if simple pneumothorax diagnosis is delayed.[39 27] TPT would also seem to be more serious in ventilated patients reaching 91% mortality rates in one series.[29]

PATHOPHYSIOLOGY

In the awake patient IPP is sub-atmospheric (during inspiration and expiration) resulting from the elastic recoil forces of the lung and in a healthy awake subject it ranges from −5 to −8 cm H_20. A forced maximal inspiratory effort can achieve an IPP of −80 cm H_20 and this is important in the awake patient trying to overcome the effects of increasing IPP as a TPT develops.

Abbreviations: TPT, tension pneumothorax; ICU, intensive care unit; IPP, intrapleural pressure; CVP, central venous pressure

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Box 1 Different definitions of tension physiology in a patient with a pneumothorax

Presence of:

- severe clinical manifestations[24]
- hiss of air on thoracic needle decompression[24 25]
- mediastinal shift
  - on chest radiograph at postmortem examination[26]
  - +/− diaphragmatic depression on chest radiography[24]
  - diagnosed clinically or on chest radiography in a ventilated patient[27]
- haemodynamic compromise with
  - haemodynamic improvement and release of gas on tube thoracostomy[28]
  - opposite mediastinal shift on chest radiography[28]
  - expanding pneumothorax[30]
- ipsilateral intrapleural pressure
  - higher than atmospheric pressure[31 32]
  - positive throughout the respiratory cycle[30]
  - positive during expiration[33 34]

In contrast with this the paralysed, sedated, and ventilated patient has gas delivered under supra-physiological positive pressure. Inspiratory pressures are often in excess of +20 cm H_20 along with either physiological expiratory alveolar pressures or positive end expiratory pressure levels upwards of +5 cm H_20.

Between the two extremes of awake and ventilated patients lie various levels of sedation and ventilatory support modes commonly used in ICUs. However, for simplicity we have assigned the evidence to two groups—awake (spontaneously ventilating) or ventilated.

Pneumothorax develops secondary to a breach in the visceral, parietal, or mediastinal pleura and is termed spontaneous (simple, idiopathic), traumatic, or iatrogenic depending on the precipitant. It is termed primary when in the absence of underlying clinically apparent respiratory disease and secondary if resultant from underlying pulmonary pathology.

If the pleural defect functions as a one way valve, air enters the pleural cavity on inspiration but is unable to exit on expiration, leading to increasing ipsilateral IPP. This will cause further lung collapse, chest wall expansion, diaphragmatic depression, and (dependent on mediastinal distensability) contralateral lung compression.

ANIMAL MODELS—AWAKE

Animal experiments use intrapleural catheters to induce expanding pneumothoraces and early experiments in awake dogs noted a significant fall in cardiac output,[40 41] which was most pronounced in the anaesthetised animals.[42] This led to the theory of thoracic vein compression, right heart compression, and great vessel kinking causing reduced cardiac filling.[40 41] It was however noted that—unlike humans—the dog's mediastinum was flimsy and unable to maintain a pressure gradient between the two pleural spaces,[42 43] along with being perforate, resulting in unilaterally introduced air spreading bilaterally.[40 44] These differences permit more direct transmission of IPP to central structures.[3] Despite this difference the theory of great vessel kinking as a cause of cardiovascular collapse persists in studies and textbooks.

Later studies used goats, swine, and sheep, which—like humans—have an intact and fixed mediastinum.[30 33 45 46] In one of these studies the animals were lightly anaesthetised (but spontaneously ventilating),[30] and in all others they were fully awake.[33 45 47] These animals were able to maintain negative contralateral IPP, even after significant positive ipsilateral IPP occurred in the end expiratory phase[33 47] and sometimes throughout the whole respiratory cycle.[30]

One study induced bilateral TPT in dogs[45] to overcome the dog model validity problems mentioned above, but this may negate any contralateral thoracic compensatory mechanisms, which occur with unilateral TPT.

These studies showed a variety of progressive compensatory mechanisms (box 2) before final collapse with some animals tolerating extreme degrees of ''tension'' very well.[30 45]

Minute volume and ventilation (to normocapnia) were preserved until a very late stage despite universal findings of progressive and severe hypoxia,[30 33 45 47] which suggests pulmonary shunting and parenchymal collapse as the main causes of hypoxia.[30 45 47]

Despite falling stroke volumes cardiac output was almost universally preserved throughout[30 33 45 47] with one study noting a 19% increase in BP.[47] In all these studies just two animals (both immature monkeys with mobile mediastinums) developed hypotension immediately before death from respiratory arrest, but cardiac output was still 70% normal at this stage.[30] Preservation of cardiac output is through a progressive tachycardia, incomplete transmission of positive IPP to the mediastinum, and increasingly negative contralateral intrathoracic pressure preserving the venous return.[30 33 47]

There was minimal evidence of right sided venous obstruction with only bilateral TPTs causing a linear rise in central venous pressure (CVP).[45] Only one of the three unilateral TPT studies found increased superior and inferior vena caval pressures and these occurred in the agonal stages of respiration.[30] Despite this there was no evidence of one specific point of obstruction or kinking in the circulation[30] and it was felt that unilateral tension does not mechanically compromise cardiac output.[30 33]

The dominant physiological feature during decompensation was progressive respiratory failure with death from respiratory not cardiovascular arrest.[30 33] This was postulated to be attributable to; myocardial hypoxia, central hypoxemia with hypoxic suppression of the respiratory centre, restrictive ventilatory defect, decreased efficiency of hypoxic intercostal muscles at the hyperinflated chest wall, and depression of the ipsilateral hemi-diaphragm from positive IPP.[30 33 47]

Box 2 Compensatory mechanisms during tension pneumothorax in awake animals[30 33 45 47]

Respiratory

- Increasing respiratory rate
- Increasing tidal volume
- Contralateral
  - increasingly negative IPP excursions
  - increasing chest wall expansion

Cardiac

- Tachycardia

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ANIMAL MODELS—VENTILATED

The effect of raised IPP is very different if the animal is ventilated and sedated, as the cardiorespiratory compensatory mechanisms (box 2) are impaired. Also, during ventilation increased alveolar pressure may obstruct cardiac output, and if alveolar pressure rises above pulmonary venous (or pulmonary arterial) pressure cardiac output will be reduced.[48]

Studies of increasing IPP in ventilated sheep and swine support this suggestion, along with showing progressive hypoxia as the earliest sign, and an immediate rise in CVP.[46 49] Hypotension and tachycardia occurred after pneumothorax of 47% total lung capacity and were progressive, being well tolerated up until eventual and sudden cardiovascular collapse at pneumothorax of roughly 90% total lung capacity. Death occurred soon after at which point SpO_2 was <50%, and CVP equalled IPP suggesting complete occlusion of venous return at this pre-terminal stage.[49] Other findings were; V/Q mismatch, alveolar compression, and hypercapnia.[46 49] These results suggest that a more rapid deterioration and early reduction in cardiac output is expected in ventilated patients.[3 50]

TENSION PNEUMOTHORAX IN HUMANS

Awake patients

Box 3 summarises the symptoms and signs from the 18 case reports found in our literature search. The time lag from initial symptoms or thoracic insult to diagnosis ranged from a few minutes to over 16 hours. Awake patients (unlike ventilated patients) manifest the compensatory mechanisms,[3] which have been clearly shown in animal studies (box 2). This leads to the disease being progressive and primarily respiratory (at least in appearance to the clinician) during the compensatory phase,[30] with hypoxemia being predominant.[3] The evidence suggests that patients have progressive respiratory deterioration with final respiratory arrest, although rare cases of cardiac collapse are reported for which the mechanism remains unclear. Cyanosis and neurological sequelae are partially treatable with oxygen delivery to the patient and are hence variably present. The key to interpreting the early signs of hypoxia and respiratory distress is the degree of severity, but more importantly a pattern of relentless progression in a patient at risk of tension pneumothorax.[3 51]

Box 3 Symptoms and signs of tension pneumothorax from case reports in awake patients[3 11 12 20 51–58]

Universal findings

- Chest pain
- Respiratory distress

Common findings (50%–75% cases)

- Tachycardia
- Ipsilateral decreased air entry

Inconsistent findings (<25% of cases)

- Low SpO_2
- Tracheal deviation
- Hypotension

Rare findings (about 10% cases)

- Cyanosis
- Hyper-resonance
- Decreasing level of consciousness
- Ipsilateral chest
  - Hyper-expansion
  - Hypo-mobility
- Acute epigastric pain
- Cardiac apical displacement
- Sternal resonance

Ventilated patients

One case series of 71 patients with advanced TPT because of significant time delays before diagnosis (table 1) and 25 other case reports (box 4) were found in the literature search.

Diagnosis of TPT in ventilated patients rests with index of suspicion and recognition of supportive diagnostic features that the evidence shows to have more consistency than those found in awake patients. The development of tension is dependent on a pressure gradient between IPP and alveolar pressure. Ventilation will increase gas flow through the pleural defect, allowing more air to pass per unit time. This gives a more rapid IPP rise with earlier mechanical compressive effects and rapid progress to cardiorespiratory collapse, which will be further hastened by trauma and greater lung damage.[3 78 79] Lung disease (ARDS/pulmonary haemorrhage/contusion) may also hasten decompensation by reducing lung compliance hence limiting lung collapse and thereby resulting in a greater IPP increase for any given gas volume.[80]

A sudden fall in SpO_2 followed by hypotension (over a few minutes) was noted in Steier's large case series, is consistently found in the individual case reports and is also seen in reports from prehospital trauma care.[36 81] There was only one report of slow development of TPT in a ventilated patient—and this patient was partially self ventilating while unsedated.[75]

Because of this sudden presentation, TPT in ventilated patients is more likely if the original pneumothorax is missed.[39] In the prehospital environment this means a low threshold for performing tube or finger thoracostomy must be maintained in ventilated trauma patients.[36 81 82] In hospital, prevention depends on early diagnosis of pneumothorax. Although the chest radiological signs signs may be subtle (box 5) other alternatives for diagnosis exist such as: computed tomography,[83] or ultrasound[84] (with a negative predictive value as high as 100%).[85]

The risk factors for pneumothorax misdiagnosis in ICU include: ventilation, unusual radiological location of pneumothorax, changed patient mental status, and lack of senior medical cover at the time of original presentation.[87–89]

Table 1 Signs of advanced tension pneumothorax (TPT) in ventilated patients—case series of 71 patients.[27]

Signs of advanced TPT	Percentage
Subcutaneous emphysema	100
Tachycardia	95
Decreased breath sounds	87
Hyper-resonance	85
Systolic BP<90	81
Cyanosis	75
Low paO_2	70
Tracheal deviation	60

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Box 4 Signs of tension pneumothorax in ventilated patients—individual case reports[1 10 28 49 50 59–77]

Universal findings

- Rapid onset
- Immediate and progressive decrease in arterial and mixed venous SpO_2
- Immediate reduction in cardiac output +/− BP

Common findings (each in about 33% of cases)

- High ventilation pressures
- Ipsilateral chest:
  - Hyper-expansion
  - Hypomobility
  - Decreased air entry

Inconsistent findings (each in about 20% of cases)

- Surgical emphysema
- Venous distension

Rarities that might make diagnosis difficult include bilateral TPT and loculated TPT causing cardiorespiratory compromise. The latter may occur if lung is bound to the chest wall by adhesions, (from parenchymal or pleural disease) so that it appears partially expanded on chest rdiography.[21 24 39 56 76 80]

MEDIASTINAL DEVIATION

Review articles of the chest radiological findings in TPT,[24 90] and case reports with chest radiographs[3 10 11 52–54 58] note mediastinal shift to be an inconsistent finding, which can also occur in the absence of TPT.[10 24 52 75 91 92] The most severe mediastinal displacement described was in a 5 year old boy,[53] which is in keeping with Rutherford's[30] comments (and chest radiographs showing) the mobility of the mediastinum in children and immature monkeys in contrast with the immobility in adults and goats. Children may therefore suffer more rapid TPT development because of the more mobile mediastinum.[30]

Even with chest radiological evidence of mediastinal displacement the trachea is often noted to be central—or uncommented upon in the case report.[3 10 11 52–54 58 93] Tracheal displacement is not commented upon in any of the animal experiments. Tracheal displacement was absent in all of the patients in a series of 108 suspected TPTs treated by paramedics with needle decompression[38] and present in only 1% of those receiving needle decompression by flight nurses in another series.[94] Even when tracheal displacement is known to be present the odds ratio for experienced physicians to correctly diagnose this has been shown to be no better than 50:50—that is, the same as tossing a coin.[14] We suggest that tracheal displacement is neither diagnostic of TPT nor does its absence exclude the diagnosis.

CHEST SIGNS

Changing signs of chest mobility and expansion are poorly described features of this disease only being mentioned in one text reviewed[7] of five major titles.[5–9]

Ipsilateral hyper-expansion has been noted rarely in awake case reports although slightly more often in ventilated case reports but is to be expected when considering the pathophysiology. It occurs as a result of the maximal

Box 5 Chest radiological signs of pneumothorax

Ipsilateral

- sharp lung edge running parallel to the chest wall
- lucency
- deep lateral costo-phrenic angle (deep sulcus sign)[86]
- abdominal quadrant hyperlucency

inspiratory efforts required to overcome the developing positive IPP with a transiently negative IPP for inspiration in awake patients. It also occurs as a result of the steadily expanding thoracic volume in both awake and ventilated patients. Ipsilateral chest hyper-expansion is consistently present on chest radiographs taken of TPT (evidenced by individual case reports,[3 52 53 70 75] reviews of chest radiography in TPT,[24 90] and animal experiments[30]) and was well shown in the awake animal experiments.

Ipsilateral chest hypo-mobility may occur as a result of pain—pleuritic or from associated rib fractures. It will also occur in the latter stages as a result of movement limitation secondary to the hyper-expansion.

Contralateral hyper-mobility is expected in awake cases as part of the compensation to generate highly negative IPPs—and this is well shown in the awake animal studies.

It may be that the infrequent description of chest expansion and mobility signs may be attributable to the subtlety in their identification. Elicitation of these signs requires careful, longitudinal examination of the chest from the height of the patient, which is difficult in a critical clinical situation that potentially requires rapid management.

Other potential chest signs include ipsilateral added sounds, such as wheeze or crackles,[3] the well described findings of decreased air entry with or without increased percussion note, sternal resonance, and displaced apex beat.

CARDIAC SIGNS

In awake patients only two case reports had early hypotension, which may have been related to TPT but one of these had significant other trauma as a possible explanation. Four patients developed hypotension between three and 16 hours after their initial insult, which had other potential causative factors (haemothorax, localised ventral TPT, myocardial ischaemia, bradycardia) in all but one. BP was preserved in all other 12 cases with tachycardia being noted in eight and no heart rate data in 10.

There was no evidence of great vessel obstruction in awake patients,[30] who (through respiratory decompensation), may lack the capacity to achieve this level of IPP. Even when mediastinal shift is evident on chest radiogrphy there is evidence of poor correlation with haemodynamic status.[93] Decreased BP or cardiac output are not common in awake patients and if found are likely to be attributable to other pathology or the hypoxic effects of TPT when it is in the advanced stages. TPT induced hypotension and/or rapidly decreasing SpO_2 is almost certainly pre-terminal.

Distension of neck veins is infrequently commented upon in both case reports and animal experiments, will only occur if there is no coexisting hypovolaemia and even then may be obscured by a cervical collar in trauma.

The decreased cardiac output in ventilated patients is consistent, early, and progressive. It is probably attributable to a combination of hypoxaemia,[3 95] diminished blood flow through the collapsed lung,[30 96 97] reduced venous return, and possibly great vessel/ventricular compression.[47 49] Hypotension is pre-terminal[3 30 49] and if TPT is not recognised and

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treated urgently it is especially likely in ventilated patients to lead to sudden cardiac arrest.[1 10]

CHEST RADIOGRAPHY IN TENSION PNEUMOTHORAX

Box 6 lists the chest radiological findings of tension pneumothorax. It should be noted that merely looking for mediastinal shift may not give conclusive differentiation of a TPT from a simple pneumothorax as this may occur in both conditions.[24 34] In rare cases a loculated anterior TPT may exist that will only be visible on a lateral chest radiography or computed tomography.[56 76 98 99]

The use of chest radiography to diagnose TPT has been associated with a fourfold increase in mortality based on the evidence from two studies in ventilated patients who waited between 30 minutes and eight hours for the chest radiograph.[27 87] This high mortality was almost certainly attributable to the prolonged delay when the diagnosis was not made clinically or the chest radiograph was misinterpreted. The high conversion rate from pneumothorax to TPT in ventilated patients[27] further alerted clinicians to diagnose the condition earlier and treat before chest radiograph. The concept of; "the chest radiograph that should never have been taken," has since been emphasised.[3 5 35 100]

However, other evidence does not support this blanket statement. Chest radiography has been used (apparently without adverse outcome) to confirm TPT diagnosis in stable patients with equivocal diagnostic signs,[52 53 70 75] so avoiding unnecessary morbidity and potentially offering alternative diagnoses.[101] Chest radiography has also diagnosed unexpected TPT in unstable ventilated patients when a lack of other signs has prevented earlier diagnosis.[63 65 66 71 77] It has been suggested that in awake patients in the emergency department a chest radiograph is obtained before tube thoracostomy thus sparing some patients the morbidity and discomfort of a potentially unnecessary procedure.[18 102] In a retrospective analysis of 176 emergency department presentations of pneumothorax none of the 30 patients identified with mediastinal shift on chest radiography, had decompensated while awaiting tube thoracostomy, and two further patients in this series with haemodynamic compromise were decompressed before chest radiography.[93]

Ultimately whether or not radiological confirmation of the diagnosis takes place must be a balanced risk of the time taken to obtain the test, and the expected clinical course. We suggest that in ventilated patients decompression should still precede chest radiography where the diagnosis of TPT is suspected. However, in awake patients we suggest that a chest radiograph precedes decompression unless the patient is unstable and potentially in the advanced stages of tension—or an immediate chest radiograph is unavailable. If a chest radiograph is not immediately available box 7 lists our recommendations for immediate decompression, (which are in partial agreement with previous indications for prehospital needle decompression).[38] When the chest radiograph is taken a practitioner capable of urgent decompression should stay with the patient.

Box 6 Chest radiological findings in tension pneumothorax[24 80 90]

Ipsilateral hyper-expansion

- hemi-diaphragmatic depression
- increased rib separation
- increased thoracic volume

Mediastinal pressure

- ipsilateral flattening of heart border
- contralateral mediastinal deviation

Box 7 Recommendations for immediate chest decompression in awake patients with tension pneumothorax as the suspected cause

Chest radiograph not immediately available and:

- $SpO_2<92\%$ on O_2
- Systolic BP<90 mm Hg
- Respiratory rate<10
- Decreased level of consciousness on O_2
- Cardiac arrest
  - bilateral finger or tube thoracostomy
  - not needle thoracocentesis

NEEDLE DECOMPRESSION

Emergency needle decompression followed by tube thoracostomy is widely advocated[5 103] but it must be appreciated that it has an associated morbidity (box 8).

It may also be ineffective with decompression only being adequately achieved on tube thoracostomy.[15–22] Barton[94] found that of 106 patients treated with tube thoracostomy by prehospital flight nurses 38% had been attributable to failure of clinical improvement with needle decompression. Box 9 lists the potential causes of needle decompression failure.

No studies exist showing the sensitivity of needle decompression[21] and yet it is regularly taught as a "rule out" investigation. This assumption of needle thoracocentesis having a 100% negative predictive value is clearly dangerous in the treatment of severely compromised patients, especially those with a potentially reversible cause of cardiac arrest.[1]

Despite the problems with needle decompression, it is a technique used commonly in the prehospital environment,[32 37 38 82] has proved safe and therapeutic,[18 36 37 94] and has been shown to lead to shorter on-scene times compared with tube thoracostomy.[94] However, some feel that the number of needle decompressions far exceeds the incidence of TPT and should be limited—especially in the prehospital environment.[18 32 102] An examination of needle decompression in 19 trauma patients, admitted to an American level 1 trauma centre found that only four patients had evidence of

Box 8 Morbidity associated with needle decompression[18 94 102 104 105]

- Unnecessary pain/discomfort for the patient
  - if procedure was not required
- Pneumothorax
  - with potential to tension later—especially if ventilated
- Cardiac tamponade
- Life threatening haemorrhage
- Loculated intrapleural haematoma
- Atelectasis
- Pneumonia

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Box 9 Potential causes of needle decompression failure[17 18 20 21 94]

- Obstruction by:
  - blood
  - tissue
  - kinking
- Missing a localised tension pneumothorax
- Inability to drain a large air leak
- Requirement for repeated needle decompression

pleural air leak and only two of these had tension physiology with just one being adequately decompressed by needle.[18]

Needle decompression in the 2nd/3rd intercostal space (ICS), mid-clavicular line (MCL) is easy to access, but entails penetration of pectoral muscles and a variable quantity of subcutaneous tissue with or without oedema and subcutaneous emphysema. A standard 14 gauge (4.5 cm) cannula may not be long enough to penetrate parietal pleura,[15 16 19 22] with up to one third of trauma patients having a chest wall thickness greater than 5 cm in the 2nd ICS MCL.[106] This will lead to treatment failure and diagnostic confusion, although using the trocar instead (7 cm) may negate this problem and prevent kinking.[22] The use of the 4th or 5th ICS in the mid-axillary line may be safer[105] and has been recommended by ATLS[5] as it contains less fat and avoids large muscles. Unfortunately this site may have an increased risk of lung damage in the supine patient, as gas collects at the highest point and adhesions are most likely in more dependent parts of the lung.[107]

A syringe filled with sterile saline attached to the cannula may help confirm pleural penetration[91 108] and an Asherman chest seal may help stabilise the cannula preventing displacement or kinking.[109] Some authors advocate the attachment of a flutter valve to the cannula[103] but if attached wrongly this may cause re-tension.[110]

TUBE THORACOSTOMY

The rapid potential deterioration of TPT means that some suggest blind tube thoracostomy upon disease suspicion or after needle decompression.[5 6] This is arguably the treatment of choice, ensuring maximal pleural cavity evacuation and lung re-expansion.[94] It has been shown to be safe and effective in trained physician led prehospital trauma care.[36 82 94] In Coats' series[36] 57 patients with TPT who had received physician led blind needle decompression or tube thoracostomy in prehospital trauma care showed an improvement in BP and $Sp0_2$, with one case of infection and one episode of tube misplacement, which was recognised and corrected on scene. Schmidt[82] noted no cases of re-tension after tube thoracostomy in his series of 76 patients. This approach inevitably has an over-diagnosis rate with 15 of 76 patients in whom tube thoracostomy was performed, having no haemopneumothorax or pneumothorax on arrival at the trauma centre.[82]

The reported complication rate of tube thoracostomy varies from 4% to 30% with only 1% occurring from insertion.[111–114] Complications (box 10) are probably less common now that use of the trocar has been abandoned in place of blunt dissection,[115] although fatalities may still occur.[116]

Many tube thoracostomy complications are related to the tube itself—re-tension, haematoma, infection, subcutaneous placement.[94] Simple thoracostomy without tube placement is an effective alternative, (in ventilated patients) that avoids

Box 10 Complications of tube thoracostomy[117 118]

- Death
- Haemorrhage (most commonly intercostal artery damage)
- Bronchopleural fistula
- Subcutaneous tube placement
- Intraperitoneal tube placement
- Infection
- Damage to
  - Lung parenchyma
  - Mediastinal contents
  - Neurovascular bundles
- Myocardium

these complications. It also decreases time to decompression, decreases on scene time in prehospital care, and enables repeat 360° finger sweep in the event of patient deterioration.[81] The latter will permit palpation of the lung to check for continued re-expansion and confirm the patency of pleural communication. The procedure simply entails a thoracostomy in the 4th or 5th ICS mid-axillary line, ensuring good finger access to the pleural cavity to permit decompression.

SUMMARY

Any pleural injury communicating with the atmosphere via a one way valve that opens on inspiration and closes on expiration will lead to an expanding pneumothorax. Defining the point of tension without IPP measurements is more difficult and it might be argued that "expanding pneumothorax" is a more appropriate term. For clinical purposes however we would suggest that a pneumothorax is considered to be under tension when it results in "significant respiratory or haemodynamic compromise (the latter especially in ventilated patients) that reverses on decompression alone"—that is, without chest drain placement. A continual egress of air throughout the respiratory cycle will add to the diagnostic certainty—unlike a brief hiss during one phase of the respiratory cycle.

The true incidence of TPT is unknown but it is more common in ventilated than awake patients and possibly most common in ventilated patients with visceral pleural injury from chest trauma.[5]

The differences in the pathophysiology and presentation of TPT between awake and ventilated patients have been mentioned by other authors[3 50] but deserve increased emphasis. TPT should not be taught as if it was a single entity. The natural history of the disease is of progressive hypoxia with a variety of cardiorespiratory compensatory mechanisms, which are well seen in awake patients but are blunted by ventilation and sedation.

In ventilated patients TPT presents rapidly with consistent signs of respiratory and cardiac compromise. In contrast awake patients show a greater variability of presentations, which are generally more progressive, with slower decompensation. In both groups the general signs are more consistent than the lateralising signs and we suggest that the clinician attempts first to diagnose the condition and then to lateralise it. In trauma if the diagnosis of TPT is in doubt these lateralising signs may also help to differentiate TPT from other causes of severe respiratory embarrassment such as flail chest and pulmonary contusion. We feel that tracheal deviation and neck vein distension should be de-emphasised, if not abandoned altogether, as taught signs

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of the disease. The clinician should also be aware of the pre-terminal signs in either group of patients and the fact that a ventilated patient is more likely to suffer a cardiac arrest in contrast with the awake patient who is more likely to suffer a respiratory arrest. We suggest the signs and symptoms listed in boxes 11 and 12 are adapted as the way of diagnosing TPT in both of these groups.

Once diagnosed treatment should begin with immediate high flow oxygen to maximise oxygenation and provide an oxygen reservoir for further resuscitation. Upright positioning may improve the patients condition and be beneficial for survival,[11 30] but should not be done in multi-trauma or other cases of suspected spinal injury.

Needle decompression still has a place and is potentially a life saver, but its indiscriminate use should be discouraged. Its potential for failure, the reasons for this, and alternative approaches should be emphasised in teaching. In awake patients it should be performed when there is specific evidence of decompensation (box 6) although immediate tube thoracostomy is preferred. Otherwise we would encourage that the clinician obtains a chest radiograph to confirm the diagnosis and lateralise the disease while being prepared to perform needle decompression should the patient decompensate.

In ventilated ICU patients needle decompression has a place in the immediate treatment of suspected TPT but where possible immediate tube thoracostomy should be encouraged as the treatment of choice. In a ventilated patient with significant chest trauma and undiagnosed shock or cardiac arrest (for example, in the prehospital or immediate resuscitation room phase) we would advocate formal thoracostomy (finger or tube) as an immediate treatment. However, it should also be noted that in unskilled hands the morbidity of thoracostomy is not insignificant (box 10).

Tension pneumothorax is a potential killer and the possibility of it should always be borne in mind. Its diagnosis however may be difficult even in the hands of experienced clinicians and the decisions regarding the best treatment option and its timing require critical decisions to be made. It is hoped that this review will help with that process and we encourage the submission and publication of further detailed case reports to build up the knowledge base—especially in the more variable disease that occurs in awake patients.

Box 11 Diagnosis of tension pneumothorax in ventilated patients

Rapid disease progression

Early reliable

- Decrease in SpO_2—immediate
- Decrease in Q
- Decrease in BP
- Tachycardia

Others

- Increased ventilation pressure
- Surgical emphysema

Disease lateralisation

- Ipsilateral
  - Hyper-resonance
  - Decreased breath sounds
  - Chest hyper-expansion
  - Chest hypo-mobility
  - Added sounds

Inconsistent

- Cyanosis
- Distended neck veins
- Tracheal deviation

Monitor dependent[32 50 95]

- Increasing CVP
- Increasing pulmonary arterial pressure
- ECG data
- Decreased mixed venous oxygen saturations

Box 12 Diagnosis of tension pneumothorax in awake patients

Reliable and early

- Pleuritic chest pain
- Air hunger
- Respiratory distress
- Tachypnoea
- Tachycardia
- Falling SpO_2
- Agitation

Disease lateralisation—ipsilateral

- Hyper-expansion
- Hypo-mobility
- Hyper-resonance
- Decreased breath sounds
- Added sounds—crackles/wheeze

Disease lateralisation—contralateral

- Hyper-mobility

Pre-terminal[3 49 54]

- Decreasing respiratory rate
- Hypotension
- Decreasing SpO_2
- Decreasing level consciousness

Inconsistent

- Tracheal deviation
- Distended neck veins

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CONTRIBUTORS

Mr Leigh-Smith initiated the review, performed the original literature search, prepared the first draft, and collated the two authors drafts. Dr Harris performed a separate literature search and produced an independent draft. Both authors contributed to the final draft. Mr Leigh-Smith is the guarantor of this paper.

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