A 61 year old female smoker with a background of chronic obstructive airways disease (COPD), FEV1 1.2/FVC 1.85; predicted 2.42/2.86, pulmonary tuberculosis, carcinoma of the breast, and coeliac disease was admitted with a six day history of progressive breathlessness associated with a productive cough. Chest auscultation showed bilateral expiratory wheeze. Admission chest radiograph showed hyperinflated lungs. She was treated for an infective exacerbation of COPD.

Five days later she became acutely unwell. Auscultation of the chest showed severely reduced air entry on the right side; the trachea was central. Urgent portable erect and supine chest radiographs did not confirm a pneumothorax. Observations: sinus tachycardia at 140 beat/min with no acute changes on 12 lead electrocardiogram, respiratory rate of 40 breath/min, decrease in systolic blood pressure to 90 mm Hg, and oxygen saturation below 90%. An intercostal drain was inserted immediately because of the very high index of clinical suspicion and it was felt that definitive treatment was required. This produced subjective and objective improvement with an improvement in oxygen saturation and blood pressure, settling of pulse and respiratory rate, and an increase in air entry on the right side. Subsequent chest radiography showed the tube to be satisfactorily placed and there was no evidence of pneumothorax. The drain was removed two days later when bubbling had ceased, again with no radiological evidence of a pneumothorax. The day after the removal of the drain the patient felt increasingly breathless, in the absence of objective findings on clinical examination. Given the diagnostic limitations of previous chest radiographs, spiral computed tomography was performed to investigate the cause of her breathlessness. This showed a right sided pneumothorax on all cuts of the tomogram (fig 1). It is most probable that the pneumothorax re-accumulated because of a further air leak after the removal of the chest drain. A second intercostal drain was re-inserted; a subsequent tomogram showed that both lungs were fully expanded with severe emphysematous change in the right middle and lower lobe and bullous emphysema on the left at the lung base.

**DISCUSSION**

Spontaneous pneumothorax occurs commonly in two groups of patients: otherwise healthy young subjects who can tolerate a large air leak and older patients with emphysema, in whom even a small pneumothorax may cause severe respiratory distress. Clinical and radiological signs may be difficult to interpret, particularly in the presence of severe COPD, large bullae may mimic pneumothoraces. National guidelines have been published to assist clinical management.1

A radiological diagnosis of pneumothorax can be made only by identifying the visceral pleural line. In the erect person, pneumothorax is first evident near the apex of the chest as air rises to the apex of the hemithorax. In the vast majority of cases, the inspiratory chest radiograph is the only imaging modality required for diagnosis. When pneumothorax is strongly suspected but a pleural line is not identified (possibly obscured by an overlying rib), gas in the pleural space can be detected by either radiography in the erect position in full expiration (the lung density is increased and volume of gas in the pleural space is constant, thus making it easier to detect the pneumothorax) or by radiography in the lateral decubitus position2 (air rises to the highest point and is more clearly visible over the lateral chest wall than over the apex). When patients with suspected pneumothorax have to be examined in the supine position, gas within the pleural space rises to the vicinity of the diaphragm. Depending on the size of the pneumothorax, the result can be an exceptionally deep radiolucent costophrenic sulcus (deep sulcus sign),3 a lucency over the right or left upper quadrants, or a much sharper than normal appearance of the hemidiaphragm with or without the presence of a visceral pleural line visible above it.4 Other findings include visualisation of the anterior costophrenic sulcus, increased sharpness of the cardiac border, collection of air within the minor fissure, and depression of the ipsilateral hemidiaphragm.5

Cross sectional imaging has the advantage over conventional radiography of visualising lung parenchyma and the pulmonary vasculature and is now increasingly accessible at the cost of delivering a higher radiation dose. It has been shown to be superior to frontal chest radiography in making a diagnosis of pneumothorax in a supine patient.6

**Authors’ affiliations**

M K Harkness, A Hashim, D Spence, Department of Medicine, Friarage Hospital, Northallerton, North Yorkshire, UK

Correspondence to: Dr M Harkness, Department of Medicine, Friarage Hospital, Northallerton, North Yorkshire DL6 1JG, UK; mharkness@doctors.org.uk

Accepted for publication 23 May 2003
Management of adrenaline (epinephrine) induced digital ischaemia in children after accidental injection from an EpiPen

I Velissariou, S Cottrell, K Berry, B Wilson

The use of adrenaline (epinephrine) containing auto-injector devices as a treatment for severe allergic reactions is now widely accepted and EpiPens are increasingly prescribed for children. It is estimated that 5% of the paediatric population in the United Kingdom have some form of food allergy. In a recent study assessing the extent of nut allergy in school children within the Severn NHS Trust, 26% of allergic children had an EpiPen at school.

In association with increased prescription of these devices, there is a greater incidence of accidental auto-injection into digits, resulting in significant pain and discomfort, because of severe vasoconstriction.

The presentation of three cases over the past six months in our accident and emergency department prompted a literature search to define the most appropriate evidence based management for this situation. We conclude that the intradigital administration of phentolamine is the preferred management.

CASE 1
A 15 year old boy was admitted with a cold and pale right thumb after accidental injection of adrenaline 0.3 mg of 1:1000 from an EpiPen he found on a bus. The injection site was on the palmar aspect of the distal phalanx of the thumb. The boy complained of pain and paraesthesia with a cold, pale thumb having a capillary refill time of five seconds.

Restoration of blood flow was attempted by warm water immersion and application of topical nitroglycerin paste. Peripheral perfusion of the digit was restored six hours later without sequelae.

CASE 2
A 7 year old boy auto-injected his left thumb while playing at home with his own EpiPen. On arrival, the puncture mark on the thumb tip was evident, however, there were no signs of impaired peripheral perfusion. He was subsequently discharged.

CASE 3
A 15 year old boy punctured his left thumb while experimenting with an auto-injecting device that he found in a nearby garden. The description of the device matched that of an EpiPen. On examination, his left thumb was found to be cold and pale, with a capillary refill time of five seconds.

After discussion with the National Poisons Information Service, topical infiltration with 1.5 mg of phentolamine mesilate in 1 ml of lignocaine (lidocaine) 2% was started with immediate response. Peripheral perfusion was restored in less than five minutes and the patient was discharged without sequelae.

In cases 1 and 3, the departmental protocol for needlestick injuries was followed.

DISCUSSION
Accidental digital auto-injection of adrenaline from an EpiPen seems to be increasingly encountered in emergency departments worldwide. It is suggested that the incidence of accidental injection in the United Kingdom, is now 1 per 50 000 EpiPen units. Recognising that this problem is increasing and is important because of the potential morbidity associated with the possible loss of a digit this review was undertaken to examine the published literature investigating this issue. Various methods have been tried to reverse the effect of adrenaline accidentally discharged into a digit. Systemic or topical nitroglycerin and warm water immersion have been attempted, but showed no significant improvement. Topical infiltration with terbutaline was suggested in one case series, however further experience in the use of this drug seems to be needed. Adrenaline can cause severe vasoconstriction because of its α adrenergic effect, therefore the use of an α adrenergic antagonist would seem appropriate.

Phentolamine, a short acting α blocker used mainly to control blood pressure during surgical resection of pheochromocytoma, has been tried. Phentolamine digital block and intra-arterial administration have both proved beneficial in reversing the vasoconstrictive effect of epinephrine induced digital ischaemia, however, a further injection was frequently required to completely restore perfusion. Local infiltration of phenolamine into the puncture site has been used and in most cases the ischaemia fully resolved within an hour.

Local infiltration of phentolamine is easier to perform and is still effective treatment up to 13 hours after the initial digital injection of adrenaline, which is useful if there is a delay in presentation.

Spontaneous reversal of circulation after adrenaline induced ischaemia without long term sequelae has been reported clinically, however most clinicians would be...
Injection of epinephrine from EpiPen into a digit

Assess the puncture site

Adequate peripheral perfusion of the digit Yes → Discharge

No

Local injection of phentolamine

- Dilute 1.5 mg of phentolamine mesilate (phentolamine mesilate amp 10 mg/ml, that is, 1.5 mg equals 0.15 ml) in 1 ml of lignocaine (lidocaine) 2%.
- Inject subcutaneously into the site and stop as soon as the skin becomes pink.
- In adults, you may need to use the whole 1.15 ml (1 ml of lignocaine 2% and 0.15 ml phentolamine), but in children you can titrate to the desired effect.
- Signs of impaired peripheral perfusion are: paraesthesia, pain, pallor, prolonged capillary refill time, and decreased digital temperature.
- If the EpiPen does not belong to the child, then the unit protocol for needlestick injuries should also be followed.

unwilling to risk losing a digit and the current literature suggests that topical infiltration of phentolamine is the most appropriate treatment as it is easy to perform, reverses ischaemia quickly and efficiently, has no reported adverse reactions, and is effective in late presentations.

However, all of the articles available in the British literature refer to adult patients. To date, there has been one case report of accidental injection of epinephrine by a 9 year old girl in Canada, which was treated successfully with topical infiltration of phentolamine.13

Phentolamine mesilate is not routinely available in emergency departments but should be readily accessible as it is shown to be particularly beneficial in relieving symptoms of vasocostriction. Treatment advice using phentolamine provided by the National Poison Centre in the United Kingdom gives doses based on adult practices. There is a need to define a regimen of topical infiltration using phentolamine in children. Associated protocols of treatment should be readily available in emergency departments and disseminated among other health professionals who may encounter similar cases, for example school nurses and general practitioners.

We include a suggested protocol of management for similar cases (fig 1).

Authors’ affiliations
I Velissariou, S Cottrell, K Berry, B Wilson, Accident and Emergency Department, Birmingham, Children’s Hospital, Birmingham, UK

Correspondence to: Dr I Velissariou, Accident and Emergency, Department, Birmingham Children’s Hospital, Steelhouse, Lane, Birmingham B4 6NH, UK; jane_vel@hotmail.com

Accepted for publication 4 March 2003

REFERENCES
Cardiac tamponade presenting to the emergency department after sternal wire disruption

S Cope, J Rodda


An unusual case of cardiac tamponade presenting to the emergency department is reported in a patient with sternal wire disruption after a pectus excavatum repair two years previously. The complication, although rare may have potentially life threatening sequelae and therefore consideration of sternal wire disruption in all patients presenting with chest pain after a previous sternotomy should be made.

A 24 year old man presented to the emergency department with a sudden exacerbation of central chest pain. He had undergone a pectus excavatum surgical repair two years previously and first became aware of pain three weeks before presentation while holidaying abroad. Upon return his general practitioner requested a chest radiograph, which confirmed fracture and displacement of part of the sternotomy wire. A cardiothoracic outpatient clinic was requested.

During this period the patient who was previously well complained of ongoing central sharp chest pain, radiating intermittently to his left shoulder and exacerbated by deep inspiration, coughing, and movement. There was no associated history of collapse haemoptysis, or calf pain. On the day of presentation the pain had become severe.

On examination he was alert and in considerable discomfort. His pulse was 81, blood pressure 145/65, respiratory rate 25, and oxygen saturations 100% on 4 l/min. Chest examination revealed a prominent sternotomy scar but was otherwise unremarkable. Abdominal, neurological, and limb examinations were similarly unremarkable.

An initial ECG showed sinus rhythm with a rate of 80, normal axis and some T wave flattening in leads 3 and Avf.

Attempted cannulation at this time was accompanied by a momentary loss of responsiveness and a decrease of systolic blood pressure to 75 mm Hg, which recovered with head tilt and 250 ml of crystalloid. A repeat ECG was unchanged.

A portable AP chest radiograph (fig 1) confirmed fracture and displacement of part of the left segment of the sternotomy wire with a lateral film (fig 2) indicating the degree of displacement.

He was referred to the cardiothoracic surgeons and an ECG was performed by the cardiologists showing a significant pericardial effusion 2.4 cm posteriorly, 3.6 cm inferiorly, and some evidence of early tamponade of the right atrium. Computed tomography of the chest confirmed pericardial puncture by the sternotomy wire (fig 3) and after a second hypotensive episode the patient was taken to theatre for an urgent thoracotomy.

It was only at this stage, some five hours after presentation that his ECG was noted to have evolved to show ST elevation in the inferolateral leads with an appearance consistent with pericarditis.

The patient went on to make a complete recovery.

DISCUSSION
Cardiac tamponade is a comparatively uncommon presentation to the emergency department, and is usually associated with penetrating trauma. Classic diagnostic teaching refers to “Beck’s triad” with hypotension, muffled heart sounds, and an increase in jugular venous pressure.

It should be acknowledged that while Beck’s triad is widely published and classically described, it is arguably a weak tool in the diagnosis of cardiac tamponade. Few clinicians, for example, would open a chest on the basis of muffled heart sounds as a discriminator.

Figure 1 AP chest radiograph showing fragmented sternotomy wire.

Figure 2 Lateral chest radiograph showing sternotomy wire fragment displacement.
65 year old woman fell on the ground from a standing position and landed on her bottom. She was unable to get up and was brought to the accident and emergency department by ambulance. She complained of pain in her hips. She was tender over her pubic ramus on the left side. Both the hip joints, dorsal and lumbar spine did not reveal any tenderness or deformity. Neurological examination did not show any abnormality. Radiography of the sacrum showed a transverse fracture of the sacrum with severe angulation (fig 2). She was admitted to the orthopaedic ward for pain relief and mobility assistance.

While on the ward she developed urinary retention that was relieved by intermittent catheterisation. After three days, although her mobility improved because of pain relief, her bladder function did not improve and she needed an indwelling catheter. Faecal incontinence was also noted at the same time. A thorough examination of the spine was done that revealed some tenderness in the middle of sacrum.

Motor examination showed weakness of the plantar flexors of ankle and toes. Absence of perianal sensation was noted. Rectal examination showed lax anal sphincter and absent anal reflex. Radiography of the sacrum showed a transverse fracture of the sacrum with severe angulation (fig 2). She was shifted to the specialised centre for rehabilitation. Her motor function improved as a result of improved muscle strength, perianal sensation was still numb, and her bladder function improved, as she was able to hold the urine for short periods, and bowels evacuated spontaneously after retraining four months after the injury.

DISCUSSION

Richarand, a French surgeon, was the first person to report a sacral fracture.1 The position of the sacrum and the strength of the bone make it less vulnerable to fracture. Their occurrence may be isolated or may be associated with other obvious fractures, which easily catch the attention of the treating doctor.2

The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3 The neurological deficit being subtle adds to the delay in diagnosis.3
The incidence of neurological deficit quoted in the literature is 56.7% to 63.6%, followed by bowel and changed sexual function are problems in the order of frequency that occur in a transverse sacral fracture. Neurological deficit is of the lower motor neuron type involving the nerve roots from S1 to S4. Though these fractures can be treated conservatively or by surgical decompression the neurological recovery especially of the pelvic viscera is unpromising and can take as long as 1.5 to 2 years.

In conclusion, a transverse sacral fracture with cauda equina injury is a very rare injury and is commonly missed. The mode of injury, a strong clinical suspicion and examination of perineum and per-rectal examination can give important clues to the underlying fracture. In case of suspected sacral fracture, computed tomography is advisable. Complete recovery is very unpredictable. A missed injury may have serious legal consequences.

Figure 1 Radiograph of the pelvis showing the fracture of the superior and inferior pubic ramus on the left side.

Figure 2 Radiograph of the sacrum lateral view—showing the transverse sacral fracture involving the body of the S2 sacral body with angulation.

taken in the casualty department are of poor quality. A lateral view should be asked for in suspected cases. Superimposition of the bowel loops on the sacrum, superimposition of the bones especially in the upper sacrum because of kyphosis, osteoporosis in the elderly population, and most importantly the lack of suspicion of the fracture are proposed reasons for delay in diagnosis. Fracture of the transverse process of L5 should raise a strong suspicion of sacral fracture. There was a 33% increase in the observed rate of sacral fracture with the use of computed tomography. Magnetic resonance imaging can show the neural compression and sagittal view may aid in the diagnosis of fracture.

The presence of perineal ecchymosis and a contusion on the base of spine apart from the tenderness is the indirect evidence of sacral fractures. Urethral injury may be present concomitantly. The incidence of neurological deficit quoted in the literature is 56.7% to 63.6%, followed by bowel and changed sexual function are problems in the order of frequency that occur in a transverse sacral fracture. Neurological deficit is of the lower motor neuron type involving the nerve roots from S1 to S4. Though these fractures can be treated conservatively or by surgical decompression the neurological recovery especially of the pelvic viscera is unpromising and can take as long as 1.5 to 2 years.

In conclusion, a transverse sacral fracture with cauda equina injury is a very rare injury and is commonly missed. The mode of injury, a strong clinical suspicion and examination of perineum and per-rectal examination can give important clues to the underlying fracture. In case of suspected sacral fracture, computed tomography is advisable. Complete recovery is very unpredictable. A missed injury may have serious legal consequences.

Authors’ affiliations

C U Dussa, B M Soni, Regional Spinal Injuries Centre, Southport District General Hospital, Southport, UK

Correspondence to: Mr C U Dussa, Flat 9-C, Y-Block, Southport District General Hospital, Town Lane, Kew, Southport PR8 6PN, UK; dussacu1@msn.com

Accepted for publication 25 March 2003

REFERENCES

Blackthorn injury: a report of three interesting cases
H Sharma, A D Meredith

Blackthorn (Prunus spinosus), a member of Rosaceae family is well known for causing infections and tissue reactions of synovial structures. Three interesting cases of cystic blackthorn granuloma, blackthorn synovitis with digital nerve entrapment, and multiple blackthorn syndrome are presented. Removal of foreign body fragments and surrounding reactive tissues resulted in an uneventful recovery with full return of joint and tendon functions.

CASE 1: CYSTIC LUMP CAUSED BY A BLACKTHORN GRANULOMA
A 47 year old man presented with a 2 × 1 cm size mobile, tender, cystic lump over the dorsum of the left hand. He had a minor injury two weeks previously while pruning. The lump was explored and removed in total as he showed no improvement to a prolonged course of anti-staphylococcal antibiotic therapy. Histopathological examination showed a small organised abscess, granulation tissue, chronic inflammatory infiltrates with a piece of blackthorn. His post-operative period was uneventful.

CASE 2: BLACKTHORN SYNOVITIS WITH DIGITAL NERVE ENTRAPMENT
A 52 year old farmer attended the emergency department three days after a history of hedge cutting. He sustained a deep penetrating injury with a history of vigorous attempts for self extraction of the thorn. He presented with red, hot, swollen dorsum of the little finger and a tender fifth metacarpophalangeal (MCP) joint. On exploration, a large piece of blackthorn was found in the subcutaneous tissue plane. On further dissection, two blackthorn pieces were found, embedded in extensor tendons of the little finger. The decision was taken intraoperatively to explore the MCP joint. This revealed four small pieces of blackthorn lodged in the joint, with inflammatory synovitis. All the blackthorn pieces and reactive synovial tissues were removed. He had an uneventful recovery.

DISCUSSION
Blackthorn injury can give rise to a wide variety of manifestations ranging from mechanical dermatitis, cellulitis, abscess, foreign body granuloma, peritendinitis, tendinitis, pericapsulitis, synovitis to acute septic arthritis. Human synovial tissue is very prone to react to organic substances like blackthorns. Removal of the blackthorn fragments causes prompt resolution of the inflammation.3,5 Granulomatous reaction is a well known manifestation caused by blackthorn.9 Expectant treatment by conservative medical therapy did not improve the thorn granuloma as showed in the first case.

Foreign body synovitis attributable to joint penetration by a blackthorn may be a cause of monoarticular arthritis, which may easily be overlooked because of its uncommon nature and difficulty in diagnosing it.7 Thorough exploration should be performed. Arthrotomy (opening up the joint), if considered necessary on table, should be performed by a consultant, or at least under consultant supervision, to avoid any recurrent problems as shown in the second case.

You should be aware of the migration of blackthorns in the coronal, sagittal, and deeper tissue planes on exploration as highlighted in the third case.

Authors’ affiliations
H Sharma, Department of Trauma and Orthopaedics, Southern General Hospital, Glasgow, UK
A D Meredith, Department of Trauma and Orthopaedics, Bronglais General Hospital, Aberystwyth, UK

Correspondence to: Mr H Sharma, Department of Trauma and Orthopaedics, Southern General Hospital, Glasgow G51 4TF, UK; himanshusharma1@hotmail.com

Accepted for publication 12 February 2003

REFERENCES
Bochdalek hernia masquerading as a tension pneumothorax
A M Dalton, R S Hodgson, C Crossley

A rare case of congenital diaphragmatic defect presenting with clinical signs of an acute tension pneumothorax is described. The clinical findings were eventually attributable to a herniation of abdominal contents into the chest (Bochdalek hernia). Attempted decompression of the chest by needle thoracocentesis and subsequent introduction of a chest drain caused gastric perforation, requiring repair at laparotomy. It is suggested that if needle thoracocentesis does not result in immediate clinical improvement, or if there is abdominal pain, a portable chest radiograph should be performed before tube thoracostomy to exclude Bochdalek hernia. All emergency department staff should be taught to recognise the radiological appearance of a Bochdalek hernia.

Diaphragmatic hernias through the foramen of Bochdalek were first described in 1848 and result from the failure of fusion of the lateral (costal) with the posterior (crural) components of the diaphragm. It occurs in one in every 2500 live births and is more common on the left.

It is well described in children, but rarely presents in adulthood.

CASE REPORT
A 43 year old, previously fit man presented to accident and emergency complaining of severe abdominal pain and shortness of breath.

Examination revealed a tachypnoea of 34 breath/min, pulse rate of 137 beat/min, and a blood pressure of 117/85. His oxygen saturation was maintained at 94%. Chest expansion was equal bilaterally with a central trachea but decreased breath sounds on the left. The abdomen was soft to palpation, but tender in the left upper quadrant.

A diagnosis of simple pneumothorax was made and a portable chest radiograph performed.

Shortly afterwards, the patient became hypotensive (blood pressure of 105/65 mm Hg), cyanosed and sweaty, with a respiratory rate of 40 breath/min and pulse rate of 140 beat/min. The chest appeared hyper-expanded on the left, with decreased breath sounds and reduced movements on that side. The trachea was deviated to the right.

The chest radiograph (see fig 1) revealed a large radiolucent area in the left hemithorax. The appearance was thought to confirm the diagnosis of tension pneumothorax.

Needle thoracostomy was performed in the second intercostal space anteriorly and a hiss of escaping air was noted.

A chest drain was introduced and air was seen to bubble through an underwater drain. There was an improvement in pulse and respiratory rate and an increase in oxygen saturation to 95%.

Within a few minutes, the chest tube had stopped draining air and the patient deteriorated again. The tube was replaced, but still no air drained.

On further analysis of the chest radiograph, herniation of abdominal contents into the left hemithorax was diagnosed. The chest drain was removed and the patient prepared for theatre. Operative findings included a large Bochdalek hernia posterolaterally with herniation of stomach, transverse colon, and spleen into the chest. There was a large perforation in the gastric fundus, presumably caused by the insertion of a chest drain.

The abdominal organs were replaced and the gastric fundus oversewn. The diaphragmatic defect was repaired. The patient made a full recovery.

DISCUSSION
Tension pneumothorax is a life threatening condition. It should be treated by immediate decompression by needle thoracocentesis followed by tube thoracostomy. The diagnosis is a clinical one and treatment should not be delayed by chest radiography.

In this case the clinical signs, while being consistent with a tension pneumothorax, were caused by herniation of a gas filled stomach into the left hemithorax. Chest radiography before chest decompression revealed the gastrothorax, but was misinterpreted. Subsequent needle thoracostomy resulted in a slight improvement, presumably because of decompression of the stomach. The radiograph (fig 1) showed lung markings in the left upper zone, separated from the large lucent area by a curved radio-opaque line representing the stomach wall. These changes are not consistent with a diagnosis of tension pneumothorax, in
which the peripheries of the lung field will be devoid of lung marking and the lung margin should be visible.

Herniation of abdominal contents should have been diagnosed and a nasogastric tube passed to decompress the stomach. Laparotomy and diaphragmatic repair should then have been carried out without the need to repair the stomach perforation.

Differentiating tension pneumothorax from bowel herniation may not be straightforward. In one study of 26 children, 16 patients (62%) were misdiagnosed clinically and radiologically. In another case report a 29 month old child presented with acute respiratory failure. Chest radiography showed a gas arteriography, which (as in this case) was misinterpreted as a tension pneumothorax and the stomach perforated by tube thoracostomy.

In conclusion, patients presenting with the signs of tension pneumothorax should have immediate decompression by needle thoracocentesis. In the presence of abdominal pain, or a failure to improve significantly after needle decompression, a portable chest radiograph should be performed before tube thoracostomy to exclude Bochdalek hernia. All emergency department staff should be taught to recognise the radiological appearance of a Bochdalek hernia.

Tension pneumothorax is normally associated with trauma, 1,2 and ventilated patients. 3–5 It is a rare diagnosis that should not be missed and may be overlooked in other settings. It may not present with all the "classic" signs leading to a potential delay in treatment. 4

CASE REPORT
While serving on a nuclear submarine a 23 year old sailor presented acutely unwell after exercise. He was an extremely fit sportsman of average height and build who was a smoker with no significant medical history.

He was dyspnoeic, and complaining of sudden left sided pleuritic chest pain. On examination he was sweaty and tachycardic with deteriorating consciousness, becoming rapidly weaker and confused. There was reduced expansion and absent breath sounds on the left side, his trachea was deviated to the right, neck veins were prominent, and apex beat was not palpable. Of note, there was no hyper-resonance on the affected side.

He was placed on high flow oxygen and positioned to maintain his airway. Because of large pectoral muscle mass, attempts at needle thoracocentesis via the second intercostal space proved difficult, and only provided transient benefit. On piercing the parietal pleura with blunt dissection through the fifth intercostal space, there was a reassuring release of air and the patient promptly improved. A 24 French gauge intercostal drain was then inserted.

No imaging facilities are available on board, therefore progress was assessed by clinical examination only. The drain remained in situ with a persistent air leak without re-expansion of the lung until day 4, when the patient coughed violently resulting in re-inflation of his lung. After a further 24 hours with no recurrence, the drain was removed and the patient remained on light duties for the remaining duration of the patrol (four weeks), with an uneventful recovery.

Subsequently the patient was referred for video assisted thorascopic surgery, where no significant pleural or pulmonary defect was found, and he had a pleural abrasion on the affected side. He recovered quickly back to full fitness, and was counselled to give up smoking. He has been passed fit for further service on submarines.

A pneumothorax is the collection of air in the potential space between the parietal and visceral pleura. A tension pneumothorax is the presence of intrapleural air under positive pressure throughout the entire respiratory cycle. Without prompt diagnosis and treatment this condition is usually fatal. Severe cardiorespiratory embarrassment occurs with a prompt fall in PaO2 and decline in stroke volume and cardiac output because of impaired venous return from raised intrathoracic pressure. 5 In this case the patient already had a considerable oxygen debt from his vigorous exercise immediately before the event, which no doubt contributed to his rapid compromise.

Primary spontaneous tension pneumothorax in a submariner at sea

F J H Brims

---

Authors’ affiliations
A M Dalton, C Crossley, Emergency Department, Hemel Hempstead Hospital, Hertfordshire, UK
R S Hodgson, Department of Gastroenterology, Queen Elizabeth 11 Hospital, Welwyn Garden City, Hertfordshire, UK

Funding: none.

Conflicts of interest: none declared.

Correspondence to: Mr A M Dalton, Emergency Department, Hemel Hempstead Hospital, Hertfordshire HP2 4AD, UK, daltonmark@yahoo.com

REFERENCES

The incidence of spontaneous tension pneumothorax is rare: a search of Medline shows only five previous case reports, but between 1% to 3% of unrecognised pneumothoraces may tension without treatment. Primary pneumothorax describes those with no underlying clinical lung disease and is more common in young men aged 15–34 and in smokers. Incidence is estimated at 18–28/100 000 per year for men and 1.2–6/100 000 per year for women. Recurrence rates in the absence of underlying lung disease or further treatment have been estimated at between 19% to 54% and are increased by continuation of smoking, and height.

A tension pneumothorax is an absolute indication for an intercostal chest drain. Furthermore, the British Thoracic Society now recommend using a cannula with a minimum of 4.5 cm in length to overcome the problems of pectoral muscle mass.

In around 80%–90% of cases with primary spontaneous pneumothorax a cause can be found with either computed tomodraphy or thoracoscopy, these are often subclinical blebs, bullae, or cysts. This submariner had a chest radiograph that was normal three years before this event as part of routine screening for pressurised submarine escape training. Indications for video assisted thoroscopic surgery include occupational risk; during the procedure a patient can undergo resection of pleural blebs and bullae as well as pleurectomy or pleural abrasion.

In this case changes in ambient pressure cannot be implicated as a submarine is at atmospheric pressure (or thereabouts) at all times and no significant rapid changes in pressure were recorded. Neither variations in ambient atmospheric pressure nor exercise are associated with increased incidence of spontaneous pneumothorax.

COMMENT

Tension pneumothorax is a clinical diagnosis and this case highlights the absolute importance of rapid recognition and treatment of such a serious condition. Previous case reports have suggested that some clinicians are not aware that tension pneumothorax can occur in the absence of trauma or artificial ventilation. In any suspected pneumothorax evidence of mediastinal shift should be carefully looked for using tracheal position and apex beat, although these can be inconsistent findings.

Author’s affiliation
F J H Brims, Adult Intensive Care Unit, Royal Brompton Hospital, Sydney Street, London SW3 6NP, UK; fbrims@doctors.org.uk

Accepted for publication 31 October 2003

REFERENCES
fractured ribs on initial or subsequent chest films. The pneumothorax was treated by the insertion of a chest drain under local anaesthesia. The chest drain was removed five days after the injury and the patient was discharged home with the right arm rested in a sling. Two months after the accident, the clavicular fracture had united and the chest radiograph was normal.

**DISCUSSION**

The clavicle is one of the most commonly fractured bones, accounting for up to 4% of all fractures. These fractures are comparatively easy to manage and typically heal with routine immobilisation. Anatomically, the apex of the lung lies behind and above the medial one third of the clavicle, with the anterior scalene muscle, brachial plexus, and subclavian vessel interferences. However, the incidence of complications associated with isolated clavicle fractures, including vascular, brachial plexus, and pneumothorax, are low.³

Most clavicular fractures result from a fall on an ipsilateral shoulder. Other mechanisms of injury include direct blows and falls on an ipsilateral outstretched hand. It is interesting to note that three of five reported cases in the literatures of pneumothorax complicating clavicular fractures were caused by direct injury of low velocity.⁴,⁵

Careful history and physical examination with particular attention to the neurovascular and chest examination are vital.³ Close inspection of the radiographs for such a potential complication are mandatory in all clavicular fractures and cannot be overstated.

**REFERENCES**
