CHEST RADIOGRAPHS—II

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The common conditions seen in the accident and emergency department that require chest radiography can be divided into two types—those resulting from trauma and those from other causes.

Trauma

Injuries associated with blunt thoracic trauma

Mechanism	Chest wall injury	Possible associated intrathoracic injury
Low energy transfer (direct blow)	Unilateral rib fracture Anterior sternal fracture	Pulmonary contusion Cardiac contusion
High energy transfer (deceleration)	Chest wall may be intact	Ruptured aorta Cardiac contusion
	Bilateral rib fracture	Ruptured diaphragm
	Sternal fracture	Ruptured bronchus
Crush injury	Bilateral rib fractures with or without flail chest	Ruptured bronchus Cardiac contusion
	Ipsilateral rib fractures with or without flail chest	Pulmonary contusion
	Possible contralateral internal rib fracture	

There are three main mechanisms of chest trauma: low energy transfer (low velocity impact—for example, a kick), high energy transfer (high velocity impact—for example, a road traffic accident), and crush injury.
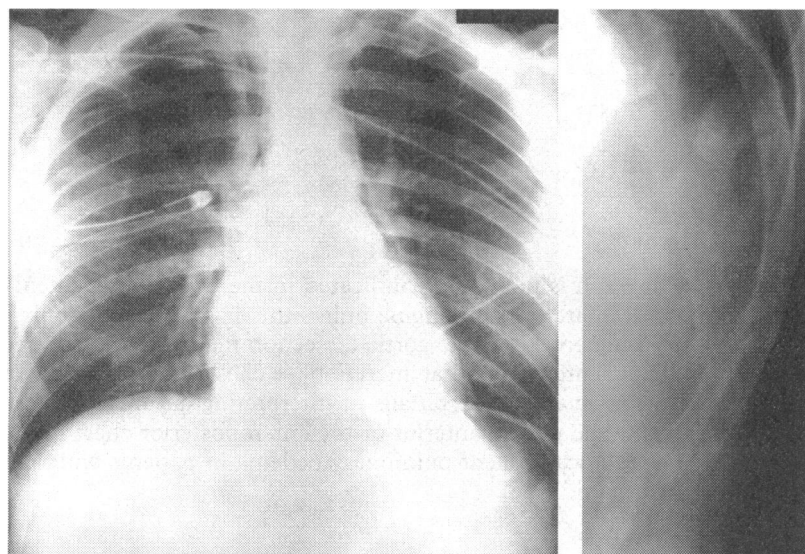
Rib fractures are best diagnosed clinically and are more common in high energy transfer and crush injuries. Remember the chest film is requested to investigate the presence or absence of associated intrathoracic injuries.

Flail segment—This occurs when two or more ribs are fractured in two or more places or when the clavicle and first rib are similarly affected. It is associated with serious intrathoracic injury, especially pulmonary contusion, and may progress to respiratory failure.

FIG 1—Anteroposterior radiograph showing right intercostal drain, pneumomediastinum, and severe surgical emphysema (linear lucent lines in the right pectoral sheath overlying the right hemithorax and in the soft tissues). Rib fractures are seen in the enlarged detail.

Signs of dissecting thoracic aorta

Widening of the superior mediastinum to >8 cm

Depression of the left main bronchus to an angle <40° with the trachea

Tracheal shift to the right

Blurring of the aortic outline

Obliteration of the medial aspect to the left upper lobe (pleural capping)

Opacification of the angle between the aorta and the left pulmonary artery

Lateral displacement of nasogastric tube in oesophagus

Aortic dissection—Ten per cent of patients with this condition survive to reach hospital. They must be identified as soon as possible since a successful outcome depends on immediate identification and treatment. The diagnosis rests on a strong clinical suspicion, plain radiographic features, and ultimately arteriography.

Cardiac contusion is associated with crush and high energy transfer injuries. The chest radiograph may appear normal but there is usually evidence of disruption of the thoracic wall with or without pulmonary contusion.

Pulmonary contusion—Patchy consolidation is the early radiographic feature of this condition but this may underestimate the severity of the injury.

Ruptured major airway—This should be suspected in the presence of any of the following: pneumomediastinum, surgical emphysema in the neck, haemoptysis, collapse of a lung or lobe, pneumothorax with major air leak.
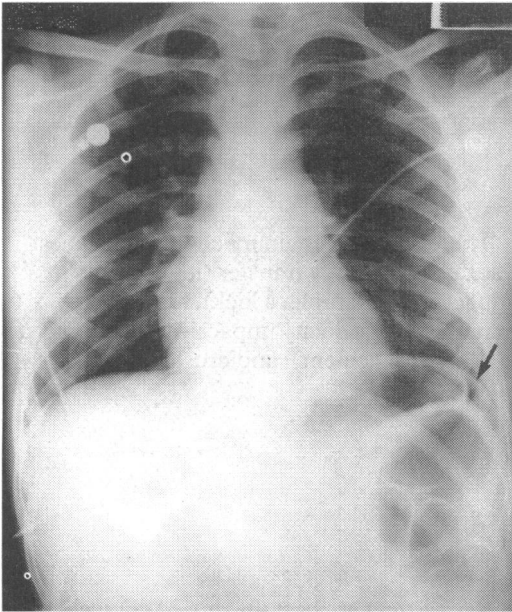
Ruptured diaphragm—This is more common on the left and should be suspected when there is a history of high energy transfer or crush injury with a bowel or stomach shadow in the thoracic cavity or an ill defined hemidiaphragm.

Penetrating chest trauma—The plain posteroanterior chest radiograph is the most important investigation. Most penetrating wounds damage only the chest wall and underlying lung. Even the more serious injuries—for example, cardiac tamponade, transected aorta, lacerated diaphragm, and major airway injury—can be diagnosed by the mechanism of injury, the clinical findings, and a posteroanterior or anteroposterior chest radiograph. Remember that penetrating injuries to the neck and abdomen can affect the contents of the thoracic cavity. More specifically, globular enlargement of the cardiac silhouette may not be apparent in the plain film even in the presence of life threatening cardiac tamponade.

FIG 2—Small pneumoperitoneum caused by a stab wound to the left lower chest (arrow).

Non-trauma

Chest pain

A chest radiograph is not usually indicated in the early management of acute myocardial infarction and angina unless the diagnosis is in doubt. A patient with spontaneous thoracic aortic dissection may present with chest pain suggestive of acute myocardial infarction—a chest film may help but it is important to be aware of the pitfalls of interpreting an anteroposterior film. Occasionally the posteroanterior or the anteroposterior chest film, or both, may show clinically silent pulmonary oedema in patients with acute myocardial infarction.

Patients with severe acute central chest pain after vomiting who have a pneumomediastinum or increasing left sided pleural effusion on radiography may have a ruptured oesophagus. In acute pulmonary embolus the posteroanterior chest radiograph can show a variety of abnormalities but more commonly it appears normal. The combination of a breathless patient with or without chest pain and a normal posteroanterior chest radiograph does not exclude a pulmonary embolus. A common finding in a chest film is a hiatus hernia; this is not usually the cause of the patient's pain.
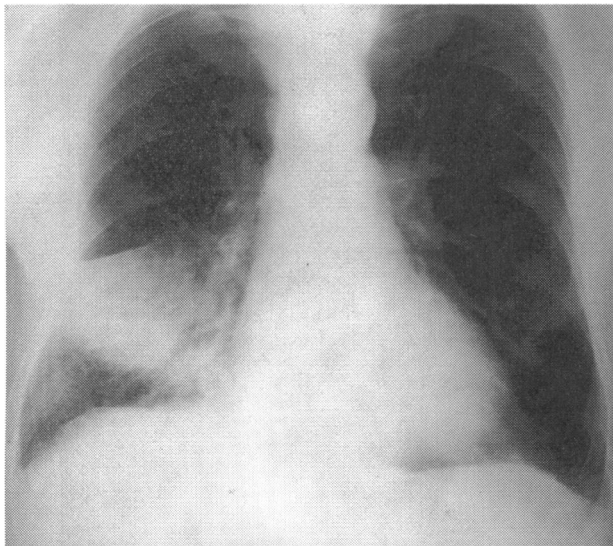
FIG 3—Right middle lobe pneumonia with a silhouette sign reducing the definition of the right heart border. There is slight volume loss in the middle lobe.
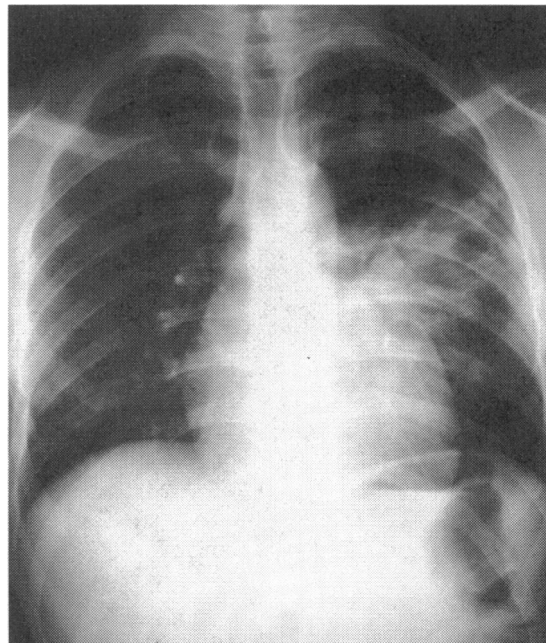

FIG 4—Consolidation in the left upper and mid-zones with cavitation in a patient with pulmonary tuberculosis.
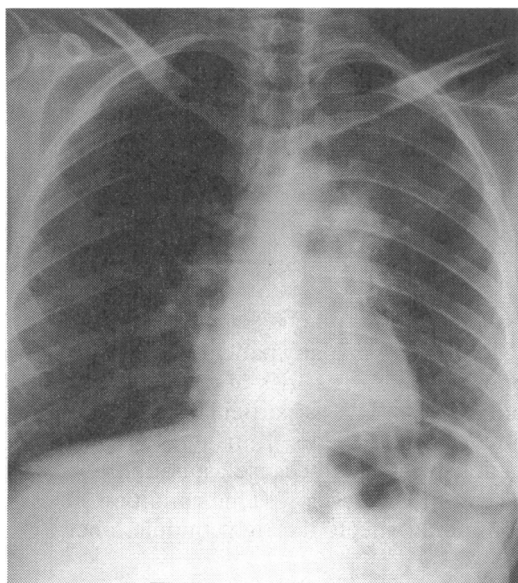

FIG 5—Left upper lobe collapse with the radiological signs of volume loss (raised left hemidiaphragm and raised left hilum) and loss of definition of the superior mediastinum on the left side.

Chest infections

The chest radiograph can confirm a clinical diagnosis of pneumonia or pleural effusion. The film usually appears normal in patients with acute asthma or exacerbated chronic obstructive pulmonary disease, but it is required to exclude conditions implicated in their aetiology or complications which will affect the patient's management—for example, pneumonic consolidation, pneumomediastinum, or pneumothorax. A chest radiograph is vital when an immunocompromised patient is thought to have an infection.

Unexplained breathlessness

Look for a pneumothorax, evidence of heart failure, or unsuspected collapse of lobes or segments (figs 5 and 6). If the radiograph appears normal remember the possibility of neuromuscular disorders, pulmonary embolism, anaemia, central airway lesions (including radiolucent foreign bodies), and the hyperventilation syndrome.

Heart failure

The chest radiograph will usually confirm clinical suspicion of heart failure or show its severity. Radiological signs of heart failure precede clinical signs (fig 7).

Haemoptysis

Look for evidence of lung cancer or tuberculosis (fig 4). A large tumour of the trachea or main bronchus may produce no radiological change until a lung suddenly collapses. Listen for stridor or monophonic wheeze. Don't forget pulmonary embolism.
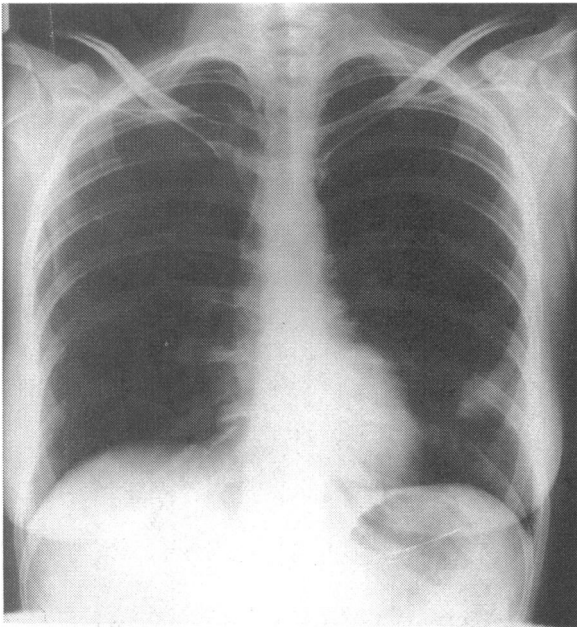
FIG 6—Abnormality in the transradiancy of the left lung field with a large irregular opacification in the left mid-zone. In this case it represented a pulmonary embolus.
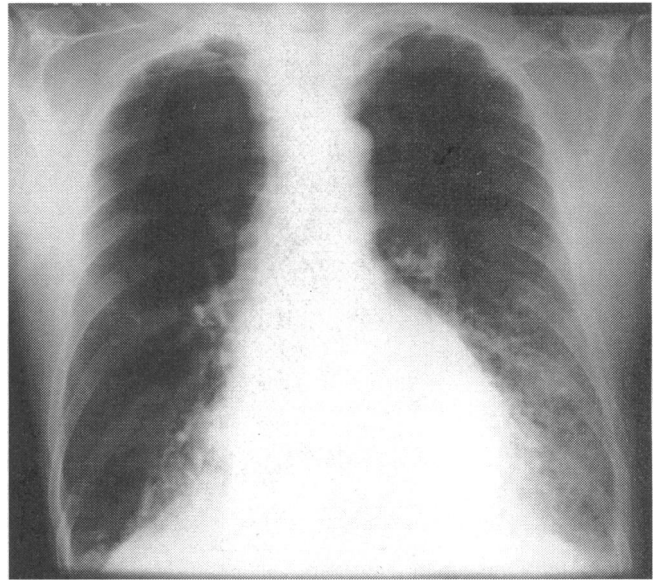

FIG 7—Cardiomegaly with severe pulmonary oedema. Peribronchial cuffing, well defined Kerley's B lines in the right lower zone, and bilateral alveolar shadowing are visible. Hyperinflation suggests pre-existing chronic obstructive pulmonary disease.
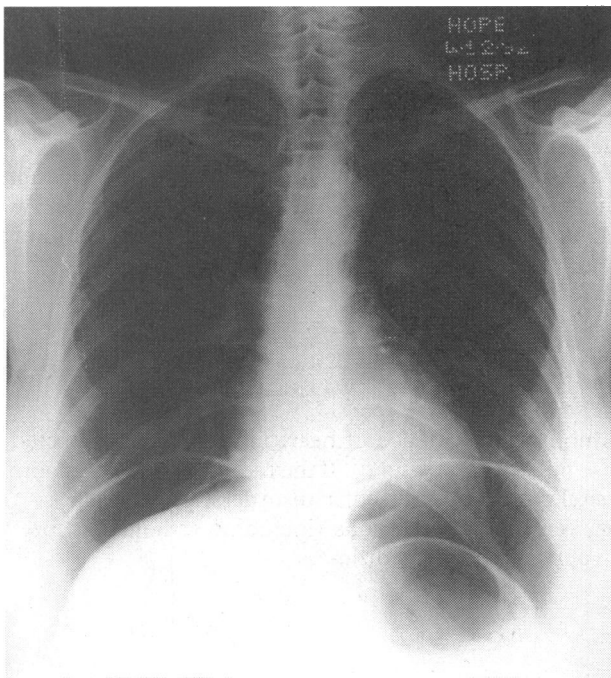

FIG 8—Large pneumoperitoneum with air under both diaphragms.

Abdominal pain

Try to get an erect posteroanterior or anteroposterior chest film to look for free gas below the diaphragm (fig 8). Remember that cases of lower lobe pneumonia may present with abdominal pain.

Neurological presentations and coma

Remember that any patient with dysphagia or a poor gag reflex is at risk of developing aspiration pneumonia. This is particularly relevant in unconscious patients. Initially there may be few radiological signs because aspiration pneumonia takes time to develop. This condition most commonly affects the right middle lobe.

Inhaled foreign body

Metallic or bony foreign bodies may be seen in the routine radiograph. The lateral radiograph will show whether a coin is in the oesophagus or in the trachea. Inhaled organic material such as food is usually radiolucent, but a lobe or segment may have collapsed if the inhalation occurred some time previously. In acute aspiration air may be trapped in the affected lobe or segment, which may show up as hyperinflation with mediastinal shift to the normal side on the expiration film.

Useful radiological rules and signs

Pulmonary oedema
The radiographic changes are often worse than the clinical condition of the patient as fluid initially collects in the interstitium

The silhouette sign

Interfaces between lung and soft tissue structures will have clear margins on a chest radiograph provided that the interfaces are smooth and tangential to the x ray beam. If air in the lung at the interface is removed (for example, consolidation) the radiographic boundary will disappear (fig 3). This is known as the silhouette sign and it can be used to localise and identify both normal and abnormal structures.
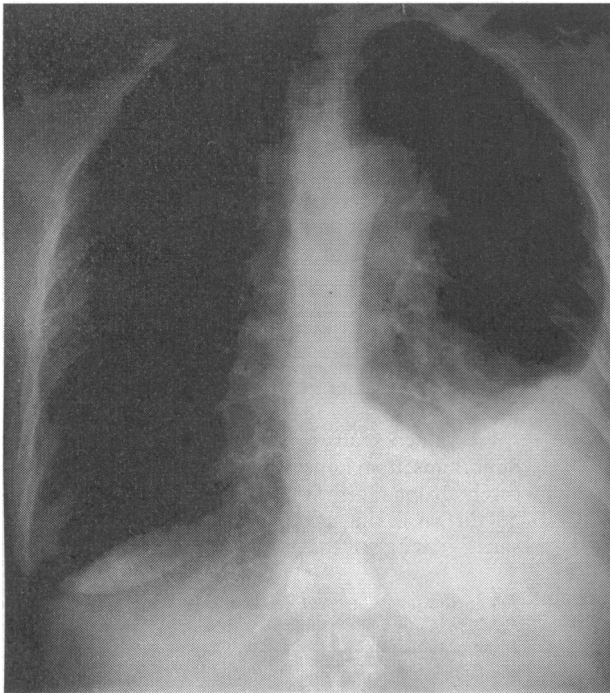
FIG 9—Left sided pleural effusion, a large mass lesion projected over the aortic knuckle, and a left mastectomy. This represents metastatic carcinoma of the breast.

Signs of consolidation

(1) Shadowing with ill defined margins due to piecemeal alveolar involvement. If the process comes up against a pleural surface the margin may be clearly defined (as in lobar pneumonia).

(2) No volume loss.

(3) Air bronchogram: normally intrapulmonary airways are invisible unless "end on" to the x ray beam, but if they pass through a zone of consolidation they become visible.

(4) Vascular changes: blood vessels are normally seen because their soft tissue density contrasts against air-containing lung but with consolidation they become obscured.

Signs of pleural effusion

These signs are different in the erect and supine films.

Erect film—Homogenous opacification of the chest; obliteration of the costophrenic angle and the hemidiaphragm; the upper margin is concave to the lung and is higher laterally.

Supine film—Reduced transradiancy of the hemithorax due to dorsal pooling. The above signs are often absent.

Summary

Name, age, and date

Position of any invasive equipment

Mediastinum, including hila and size and shape of heart

Diaphragms, including costophrenic angles

Lung field abnormalities, including apices

Bones

Extrathoracic soft tissues

Signs of lobar collapse

(1) Crowding of vessels and airways within the lobe.

(2) Raised hemidiaphragm.

(3) Shift of mediastinum, hilum, fissures, and other structures.

(4) Compensatory hyperinflation.

(5) Ribs close together.

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The ABC of Emergency Radiology has been edited by David Nicholson and Peter Driscoll.

ANY QUESTIONS

Can men who have had a vasectomy develop epididymitis?

The simple answer is yes. The exact route by which infective organisms gain access to the epididymis is not clear. As long ago as 1953 it was suggested that reflux of urine from a full bladder could occur through the normal ejaculatory duct check valve on the floor of the prostatic urethra, allowing infected urine to pass along the lumen of the vas deferens to the epididymis.[1] Such vasoepididymal reflux, however, has never been documented under normal conditions. It can occur after prostatectomy when the ejaculatory duct check valve has been damaged and urine has been reported to leak from the cut end of the vas.[2] Prophylactic vasoligation during prostatectomy, however, is no longer performed routinely, and epididymitis after prostatectomy has become a rare event, largely because of improved sterilisation of endoscopic instruments and treatment of known urinary infection before operation.

Under normal conditions vasoepididymal reflux cannot be shown even under the provocation of lifting a heavy weight with a full bladder. Direct endoscopic injection of fluid into the ejaculatory duct rarely produces reflux of urine, and, even under very high pressure, flow cannot be achieved beyond the junction of the vas deferens with the duct of the epididymis.[3] The presence of secretions and products of spermatogenesis, together with peristaltic activity down the vas deferens towards the seminal vesicle, may also act as a physical barrier to intraluminal transmission of infection.

It seems most likely, therefore, that infection reaches the epididymis via the lymphatics and lymphatic spaces which follow the vasal arteries; hence occlusion of the vas deferens does not protect against epididymitis.

For reasons which are not entirely clear, epididymitis after vasectomy can be notoriously difficult to treat, and prolonged courses of broad spectrum antibiotics may be necessary to obtain complete resolution. In some patients, despite adequate treatment, inflammation and epididymal pain may persist, probably because of rupture of epididymal tubules and leakage of sperm with infective organisms into the epididymis to form small, chronically infected sperm granulomas.—NIGEL BULLOCK, *consultant urologist, Cambridge*

1 Badenoch AW. Vaso-epididymal reflux syndrome. *Proc R Soc Med* 1953;46:847-9.
2 Hanley HG. Urinary fistula following scrotal vasectomy. *Br J Urol* 1945;17:34-5.
3 Wolin LH. On the aetiology of epididymitis. *Br J Urol* 1971;105:531-3.