

Causes of a widened mediastinum: a pictorial review

SA Barnard, CKL Cook

A widened mediastinum is a common finding on the plain chest radiograph. It is often encountered in the acute setting, where correct interpretation is essential to guide immediate management.

There are numerous causes of a widened mediastinum on the plain chest radiograph (CXR). Although some authorities suggest that the mediastinum is widened on the posteroanterior (PA) CXR when measured at more than 8 cm in transverse diameter at the level of the carina, most radiologists' opinion of the mediastinal contour and size is subjective. Indeed, in trauma cases, Burney et al (1983) have confirmed that a subjective assessment of the mediastinal width is most useful.

It should be remembered that the cardiothoracic ratio (CTR) is purely a measurement of heart size. The CTR (in adults normally less than 50%) is assessed by the ratio of the widest breadth of the heart to the widest internal diameter of the thoracic cavity.

Artefactual widening is common. This occurs because the mediastinal structures are anatomically positioned anteriorly, and are therefore magnified on anteroposterior (AP) films, especially when taken supine. The mediastinum often appears widened or equivocal on the supine AP film but is shown to be normal in width on the erect PA film. An erect PA CXR should therefore be obtained wherever possible.

The causes of true mediastinal widening can be divided into vascular and non-vascular. Vascular causes of mediastinal widening can be differentiated from non-vascular causes such as lymph node enlargement by the recognition of a smooth vascular contour and the continuation of this contour with known vascular structures such as the aorta or the subclavian arteries.

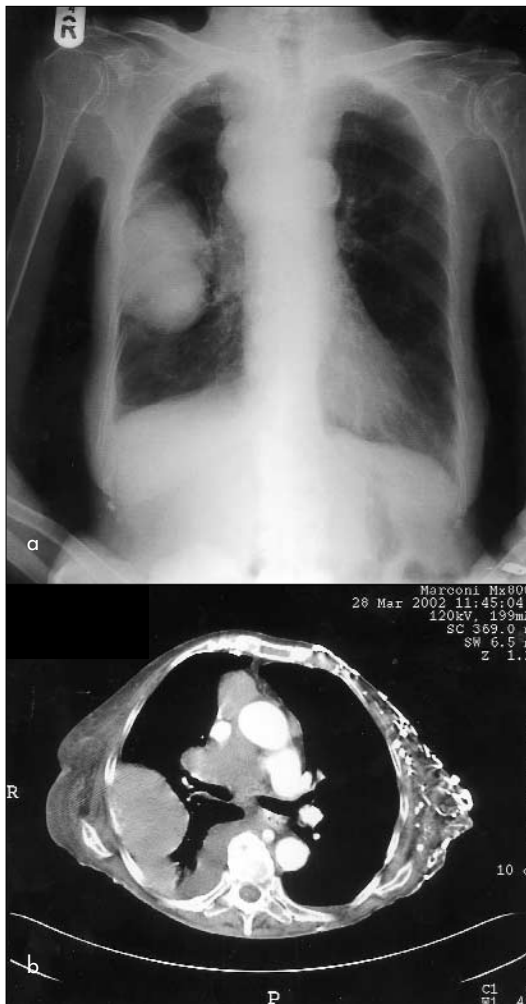
Although there are clues in the history and the plain film findings that may enable a diagnosis to be made, a computed tomography (CT) scan of the thorax and ultimately biopsy are often necessary to refine the diagnosis (Figures 1a and b).

This article will now consider the common pathologies in turn.

VASCULAR CAUSES

When considering the vascular causes of mediastinal widening, it is essential to differentiate between non-traumatic and traumatic causes. The patient's mode of presentation will usually make this distinction obvious.

The pathological entities that will be considered are aneurysms (true and false) and aortic dissection. True aortic aneurysms involve dilata-



Dr SA Barnard is Radiology Specialist Registrar, Bristol Royal Infirmary, Bristol and Dr CKL Cook is Consultant Radiologist, Weston General Hospital, Weston-super-Mare BS23 4TQ and Bristol Royal Infirmary, Bristol

Correspondence to: Dr CKL Cook

Figure 1. a. Posteroanterior chest X-ray: There is a large smooth lobulated soft tissue mass in right mid-zone, with associated bone destruction and superior mediastinal lymphadenopathy. The appearances were confirmed to be caused by small cell carcinoma. **b.** Computed tomography through mid-thorax of same patient: The lobulated mid-zone mass seems to be continuous with the mediastinal lymphadenopathy. Bone destruction is again demonstrated. There are multiple collateral vessels opacified with contrast in the left axilla as a result of central great vein occlusion.

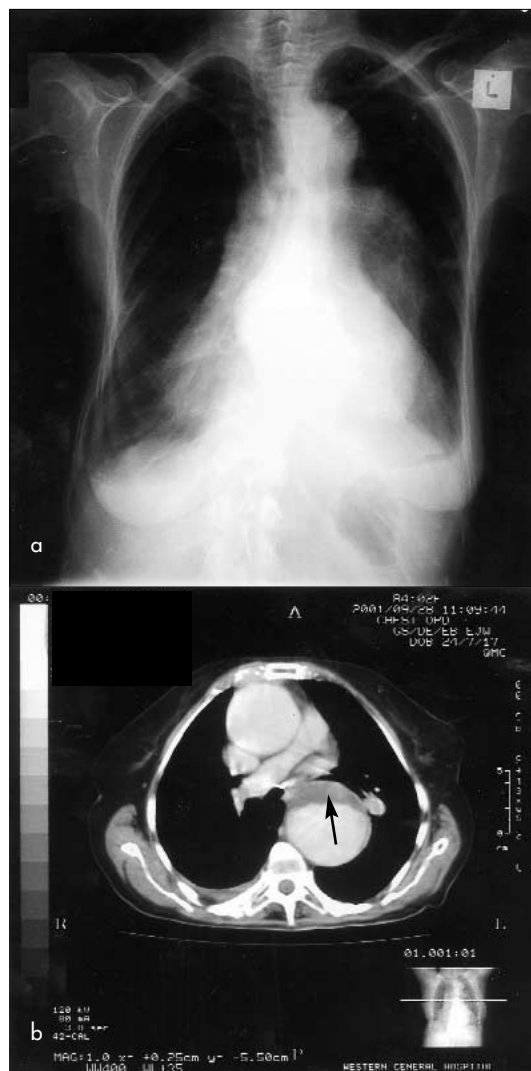
tion of all three layers of the aortic wall whereas false aneurysms (which are usually the result of contained ruptures) are contained only by the outer layer of the aorta (adventitia). Dissection occurs when blood enters the media and separates the intima from the adventitia.

Non-traumatic causes

Non-traumatic vascular causes of mediastinal widening include aneurysms and dissection.

Aneurysms: Aneurysms of the thoracic aorta may include the ascending aorta, the arch or the descending aorta, either individually or in combination. Common aetiologies include atherosclerosis and connective tissue diseases (*Figures 2a*

Figure 2. a. Posteroanterior chest X-ray showing a soft tissue opacity with smooth outline extending from the aortic knuckle to the retro-cardiac position. These are the appearances of thoracic aortic aneurysm. b. Contrast-enhanced computed tomography scan of the same patient. Both ascending and descending aorta are confirmed as aneurysmal. There is moderate thrombus within the descending aorta (black arrow).



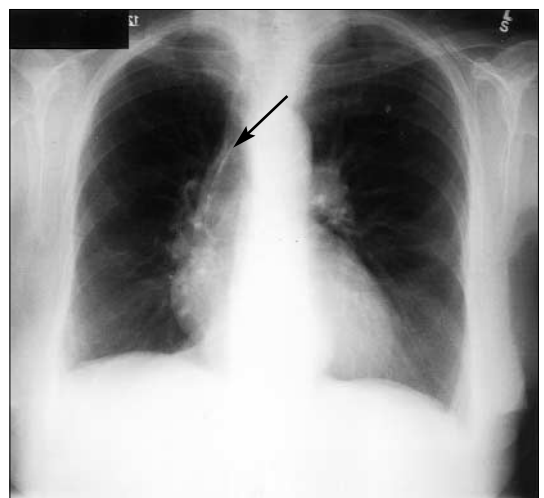
and b). Aneurysms can be confused with aortic ectasia (unfolding), although the aortic diameter should not be dilated in the latter.

Aneurysms of the ascending aorta usually begin at the aortic root. Plain CXR findings of an ascending aortic aneurysm include prominence of the right mediastinal border and cardiac enlargement if the left ventricle dilates as a consequence of aortic valve incompetence. Most cases are idiopathic, although Marfan's and Ehlers–Danlos syndromes do predispose. Syphilis was once a common cause of ascending aortic aneurysms but is now uncommon in developed countries. The aortic wall is often calcified in syphilitic aneurysms (*Figure 3*). Similar appearances may be seen in connective tissue disorders. Complications of thoracic aortic aneurysms include aortic valve incompetence, aortic dissection and rupture.

Medical treatment consists of treating hypertension and other risk factors. According to Coady et al (1997) surgery is recommended when the ascending aortic diameter reaches 55 mm, although patients with Marfan's syndrome may require surgery earlier because they have a higher risk of rupture. Elective surgery is much safer than emergency surgery and will usually include the replacement of the aortic valve together with synthetic graft replacement of the ascending aorta.

Aneurysms of the aortic arch and descending aorta are usually the result of atheromatous disease. Patients may present with chest pain. Local pressure effects include hoarseness, stridor and occasionally superior vena cava obstruction.

Figure 3. Posteroanterior chest X-ray. There is a smoothly outlined soft tissue density on the right side of the mediastinum. This is continuous with the aortic knuckle and contains calcification within it (black arrow). The appearances are those of ascending aortic aneurysm caused by syphilis.



Plain CXR findings include enlargement and tortuosity of the aortic outline. Contrast-enhanced helical CT scanning is the preferred method of investigation. Surgery for descending aortic aneurysms carries a higher risk of complications (especially paraplegia resulting from the interruptions of spinal arteries) and so the threshold for surgery is higher (65 mm; Coady et al, 1997).

Aortic dissection: Aortic dissection is a significant cause of mortality in the 50–70-year-old age group. Patients with a history of Marfan's syndrome, Ehlers–Danlos syndrome and hypertension have an increased risk of aortic dissection. Acute aortic dissection typically presents with acute chest pain radiating to the back. It is an especially important to differentiate this from myocardial infarction since thrombolysis may lead to increased morbidity and mortality.

Until the advent of helical (spiral) CT, invasive aortography was the only reliable method of diagnosing aortic dissection. However, contrast-enhanced helical CT of the thorax is now generally accepted as the method of choice in investigating a patient with suspected aortic dissection, particularly as this is non-invasive (Moore et al, 2002) and more widely available. Although an intimal flap may be demonstrated on imaging, it is not essential to make the diagnosis. The Stanford classification is now used to describe the site of the dissection (Dailey et al, 1970). Stanford type A dissections involving the ascending aorta are more common (70%) and are more likely to result in early mortality as a result of their propensity for rupture into the pericardium, causing tamponade, acute aortic incompetence and dissection of the coronary or brachiocephalic arteries. Type B dissections begin distal to the origin of the left subclavian artery and are generally treated conservatively.

Traumatic vascular causes

Rapid deceleration is the typical cause of acute traumatic aortic injuries (TAI). This is often as a result of road traffic accidents (Koali et al, 1991). Most patients with TAI will die before reaching hospital, and of those that do survive up to 40% will die within the first 24 hours after admission if they are not actively treated.

There are no reliable clinical findings in TAI. Early consideration of TAI is important and should not be overlooked in the presence of other distracting injuries. The CXR is a reliable method of excluding TAI, but it is non-specific. A good quality normal erect CXR has a negative predictive value of 98%; this drops to about 96% when a supine CXR has been obtained (Mirvis

et al, 1987). TAI is manifested on CXR by mediastinal widening, abnormal aortic contour, deviation of the trachea to the right and widening of the left paraspinal line in the absence of vertebral fracture. Other less reliable signs include a left apical cap and widening of the right paraspinal line.

Contrast-enhanced CT has developed as the investigation of choice in cases of suspected TAI. As in the evaluation of aortic dissection, its major advantages are its acute and widespread availability, and its non-invasiveness. CT also demonstrates mediastinal haematoma and other non-aortic pathologies.

NON-VASCULAR CAUSES

The non-vascular causes of mediastinal enlargement include lymphadenopathy, thyroid or thymus enlargement, teratoma, and occasionally other rare causes.

Mediastinal lymphadenopathy

Mediastinal lymphadenopathy is a common cause of mediastinal widening. The right paratracheal group are easily visualized because of the widening of the right paratracheal stripe. Enlarged aortopulmonary nodes will cause a bulge between the aortic arch and the left pulmonary artery. Subcarinal lymphadenopathy may cause splaying of the carina. The pattern of other lymph node enlargement is often less specific and will lead to less focal mediastinal widening. Lymphadenopathy is best demonstrated on CT, where the short axis of the lymph node should be measured. Mediastinal lymph nodes are considered abnormal if the short axis diameter exceeds 10 mm (Glazer et al, 1985).

Ultimately, tissue biopsy may be required to finalize the diagnosis in these non-vascular causes.

This article will now consider the more common causes of mediastinal lymphadenopathy.

Sarcoid: Sarcoid is a common cause of mediastinal lymphadenopathy and hilar lymphadenopathy. Involvement at the hila is characteristically bilateral and symmetrical (*Figure 4*).

The hilar nodes are almost always enlarged and they can be distinguished from mediastinal nodes by the lucent line that separates them medially from the mediastinum. The majority of patients will also have enlarged right paratracheal, aortopulmonary and subcarinal lymph nodes (Sider and Horton, 1990). These may later calcify (*Figure 5*).

Lymphoma and leukaemia: Lymphoma (usually Hodgkin's rather than non-Hodgkin's lymphoma) and leukaemia commonly cause anterior

and paratracheal lymphadenopathy. The lymphadenopathy is usually bilateral (*Figure 6*), but may be asymmetrical. These appearances can resolve rapidly after treatment.

Infection: Infectious causes of mediastinal lymphadenopathy include tuberculosis and histoplasmosis. Any lymph node group may be affected and there may be coexistent pulmonary consoli-

dation. Treated infection is a common cause of calcified lymph nodes, but these may also be caused by silicosis or treated lymphoma.

Recent events have reminded us that anthrax is an unusual cause of mediastinal widening (Earls et al, 2002).

Primary and secondary malignancies: Metastatic carcinoma may be manifested by mediastinal lymph nodes. Bronchogenic carcinoma is a common primary, and indeed such a tumour may mimic mediastinal lymph nodes if it is positioned centrally (*Figure 7*). Other possible

Figure 4. Posteroanterior chest X-ray: there is bilateral symmetrical hilar lymphadenopathy. These are the appearances of sarcoid.



Figure 5. Anteroposterior chest X-ray: There is linear mid and upper zone reticular shadowing in keeping with fibrosis. Multiple calcified lymph nodes are seen within the mediastinum. The appearances are those of sarcoidosis.

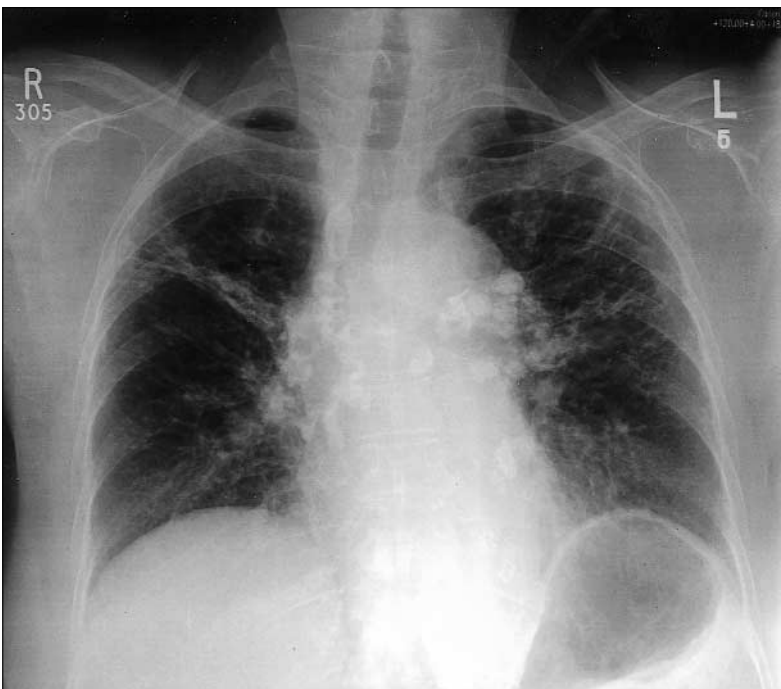


Figure 6. Posteroanterior chest X-ray: there is bilateral symmetrical superior mediastinal lymphadenopathy and associated hilar lymph node enlargement. The appearances are those of lymphoma.

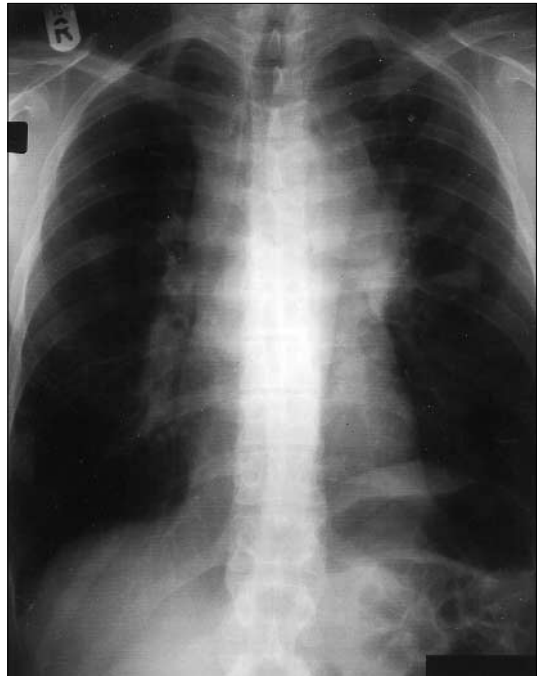


Figure 7. Plain chest X-ray: this shows a poorly defined mass adjacent to the aortic arch in the left upper zones. Its irregular margin suggests this is malignant. This was confirmed as a rather centrally positioned primary bronchogenic tumour.



sites for a primary include the genitourinary tract, head and neck tumours, and breast.

There are a number of other causes of mediastinal widening not caused by lymphadenopathy. These are discussed individually below.

Thyroid and thymus glands

Thyroid goitre: A thyroid goitre may extend down into the mediastinum from the neck. Thyroid enlargement is often asymmetrical and can cause focal deviation and narrowing of the trachea (*Figure 8*). CT of a thyroid goitre will show a well-defined mass in the para- or retrotracheal region which is continuous with the thyroid in the neck. Thyroid tissue is of higher attenuation than muscle on both unenhanced and enhanced scans.

The thyroid enlargement is usually the result of multinodular colloid goitre but occasionally adenoma or carcinoma can exist in an enlarged thyroid extending into the thorax. It is not possible to diagnose malignancy in the thyroid on CT unless there is local spread beyond the gland.

Thymus gland: The thymus gland usually lies anterior to the ascending aorta but may lie as low as the cardiophrenic angles. It is large in neonates and infants but atrophies with age and certainly should not be visible on the plain CXR beyond childhood. Enlargement of the thymus may be the result of thymoma, lymphoma, thymic carcinoma, germ cell tumour or teratoma, and thymolipoma.

Thymoma: Thymomas are smooth in outline. They may be large and adjacent to the aortic outline (*Figures 9a and b*). The majority of thymomas are benign but 10–40% are malignant. Of patients with myasthenia gravis, 30–40% will suffer from myasthenia gravis, and 10% of patients with myasthenia gravis will have a thymoma. Thymomas are also associated with red cell aplasia and hypogammaglobulinaemia. They are rare before the third decade.

Teratoma or germ cell tumour

The teratoma or germ cell tumour spectrum ranges from benign to malignant and includes benign cystic teratoma (also called dermoid cyst) and the malignant germ cell tumours: seminoma, teratocarcinoma, embryonal carcinoma and choriocarcinoma. Benign cystic teratomas are the most common. Malignant germ cell tumours produce an asymmetrical lobular mass and grow rapidly, metastasizing early to lungs, bone and pleura. They secrete beta human chorionic gonadotrophin and alpha fetoprotein, which can be detected in the blood.



Figure 8. Posteroanterior chest X-ray: showing smooth outlined soft tissue mass within the superior mediastinum. The trachea seems to extend through this region. The soft tissue mass represents the lobes of a large retrosternal goitre.

Mediastinal cysts

Foregut cysts arise from the embryonal foregut and include bronchogenic, enteric and neurenteric cysts. Bronchogenic cysts are the most common.

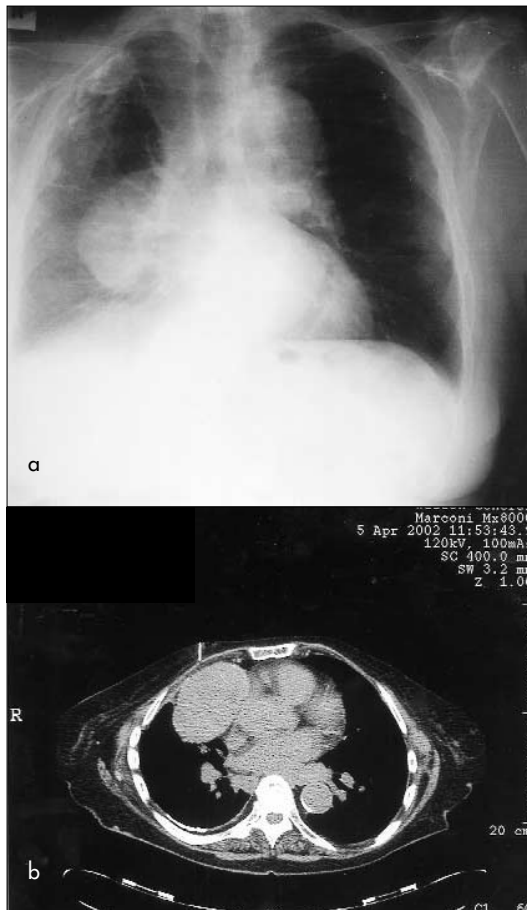


Figure 9. a. Posteroanterior chest X-ray: there is a large smooth well-defined soft tissue mass arising from the right heart border. This was histologically confirmed as thymoma. Also note some unfolding of the descending aorta. The calcified region in the right upper zone is caused by an old tuberculous empyema (an incidental finding). b. Computed tomography-guided biopsy of the same case. This confirms the right-sided mass, and a needle is seen in its periphery passing into this region.

CONCLUSIONS

There are numerous causes of mediastinal widening on the CXR. Artefactual widening should first be excluded by an erect PA CXR if possible. There are some CXR features that may suggest a particular diagnosis or differential diagnosis. These features should be sought in conjunction with the history, examination and other investigations to direct further management. Where the mediastinal widening is caused by lymphadenopathy, the pattern of lymph node involvement and the clinical history may give some clue as to the aetiology. However, as the images in this review show the appearances on CXR are often non-specific and CT (and ultimately biopsy) may be needed to confirm the diagnosis. **HM**

KEY POINTS

- The anteroposterior chest X-ray may indicate artefactual widening. An erect posteroanterior chest X-ray should be obtained whenever possible.
- The chest X-ray findings should be taken in the context of the clinical history, particularly if there is a history of trauma.
- Smooth mediastinal widening which is continuous with vascular structures is most likely the result of aneurysm or ectasia.
- A lobulated outline may be the result of lymphadenopathy, and its position and symmetry may give a clue as to its aetiology.
- Computed tomography and biopsy may be necessary to confirm the diagnosis.

Conflict of interest: none.

- Burney RE, Gundry SR, Mackenzie, JR, Wilton GP, Whitehouse WM, Wu SC (1983) Comparison of mediastinal width, mediastinal-thoracic and cardiac ratios, and 'mediastinal widening' in detection of traumatic aortic rupture. *Ann Emerg Med* **12**(11): 668–71
- Coady MA, Rizzo JA, Hammond GL, Mandapati D, Darr U, Kopf GS, Elefteriades JA (1997) What is the appropriate size criterion for resection of thoracic aortic aneurysms? *J Thorac Cardiovasc Surg* **113**(3): 476–91
- Dailey PO, Trueblood HW, Stinson EB et al (1970) Management of acute aortic dissections. *Ann Thorac Surg* **10**: 237–46
- Earls JP, Cerva D Jr, Berman E et al (2002) Inhalational anthrax after bioterrorism exposure: spectrum of imaging findings in two surviving patients. *Radiology* **222**(2): 305–12
- Glazer GM, Gross BH, Quint LE, Francis IR, Bookstein FL, Orringer MB (1985) Normal mediastinal lymph nodes: number and size according to American Thoracic Society mapping. *AJR Am J Roentgenol* **144**(2): 261–5
- Koali S, Jamieson WR, Leia-Stephens M, Miyagishima RT, Janusz MT, Tyers GF (1991) Traumatic rupture of the thoracic aorta. A 20-year review: 1969–1989. *Circulation* **84**(5 Suppl): III40–6
- Mirvis SE, Bidwell JK, Buddemeyer EU, Diaconis JN, Pais SO, Whitley JE, Goldstein LD (1987) Value of chest radiography in excluding traumatic aortic rupture. *Radiology* **163**: 487–93
- Moore AG, Eagle KA, Bruckman D et al (2002) Choice of computed tomography, transesophageal echocardiography, magnetic resonance imaging, and aortography in acute aortic dissection: International Registry of Acute Aortic Dissection (IRAD). *Am J Cardiol* **89**(10): 1235–8
- Sider L, Horton ES Jr (1990) Hilar and mediastinal adenopathy in sarcoidosis as detected by computed tomography. *J Thorac Imaging* **5**(2): 77–80

Further reading

- Grainger RG, Allison DJ, Adam A, Dixon AK (2001) *Grainger and Allison's Diagnostic Radiology: A Textbook of Medical Imaging*. 4th edn. Churchill Livingstone, London
- Weatherall DJ, Ledingham JGG, Warrell D (1996) *Oxford Textbook of Medicine*. 3rd edn. Oxford University Press, Oxford