In patients with ischaemic heart disease it is helpful to obtain a plain film of the chest as soon as the patient is fit enough after admission; usually a short distance portable film in the antero-posterior position will have to be taken. This preliminary film will act as a base-line; it need not be repeated, provided convalescence is uneventful. If there is any change in the patient’s condition, further films, even at daily intervals, can be most helpful in influencing management.

The initial film acts as a base-line view for the assessment of heart size and shape and the appearance of the lungs, but it may also exclude non-cardiac causes of chest pain, such as a large hiatus hernia.

In the acute phase of an ischaemic episode, the main features noticed on the film, as far as the lungs are concerned, are the presence or absence of pulmonary venous hypertension, pulmonary oedema or linear or segmental shadows at the lung bases (Tudor et al., 1973). It has been well established that the presence of cardiac enlargement and pulmonary oedema on admission are very significant influences on the prognosis (Norris et al., 1970). When pulmonary shadows are present, progress should be followed by serial films.

A more latent appearance of lung abnormalities that can be associated with myocardial infarction is Dressler’s syndrome; this can be associated with lung shadows occurring some weeks after the initial episode of acute infarction (Dressler, 1959).

**Pulmonary Oedema**

The appearance of pulmonary oedema in ischaemic heart disease is primarily due to an elevated pulmonary venous pressure. As the venous pressure rises fluid extravasates into the interstitial spaces of the lung and from there moves into the alveolar spaces. For this reason we can recognise pathologically and radiologically two types of oedema, interstitial and interalveolar.

**Interstitial Oedema**

The following radiological features are seen in interstitial oedema: horizontal septal lines, the so-called Kerley B lines. They are recognised at the lung edges...
in the lower and mid zones as short perpendicular shadows only a few centimetres in length at right angles to the pleural surface (Fig. 1). Their appearance is due to fluid accumulation in the interlobular septa. These shadows are not due to distended interpulmonary lymphatics; although the lymphatics are dilated, they are not large enough to be visible radiographically. In addition to the short Kerley B lines, or peripheral septal lines, longer septal lines extending from the lung periphery to the hilum can be seen as fine linear shadows of uniform calibre crossing over the normal vascular markings (Fig. 2).

The excessive accumulation of interstitial fluid tends to blur the detailed outline of vascular and bronchial shadows, particularly in the region of the lung hila and also towards the lung base, thus producing a generalised haze (Fig. 2). This appearance makes interpretation of pulmonary vascular markings in the affected areas very difficult. These features are sometimes referred to as peribronchial and perivascular cuffing (Heard et al., 1968). The perivascular accumulation of oedema fluid may also play an important part in elevating pulmonary venous pressure by interfering with the elasticity of the lung, which normally keeps the small vessels open, quite apart from the mechanical compression of the smaller vascular branches by the oedema (West et al., 1965).

Septal lines often precede the appearance of clinical signs of pulmonary oedema; they are the earliest radiological sign of pulmonary oedema and of incipient left heart failure. Septal lines can clear very rapidly as the pulmonary venous pressure drops and the patient’s condition improves; thus their appearance and clearance is one of the most helpful radiological signs in the assessment of left-sided heart failure. In patients who have had previous episodes of left ventricular failure, septal lines occasionally persist due to the deposition of haemosiderin and fibrous tissue within the septa. This persistence of septal lines can be mistaken for interstitial oedema unless an accurate comparison with previous films has been made.

Acute and overwhelming interstitial oedema rapidly leads to the development of interalveolar oedema. This frequently happens in patients with acute left-sided heart failure due to coronary infarction.

**Interalveolar Oedema**

Interalveolar oedema is characterised by confluent shadows of fairly uniform density that can affect the lung fields widely. The distribution of these shadows can be central, unilateral, apical, basal, but most often the posterior and most dependent segments of the lungs are affected. The patient’s position at the time when pulmonary oedema develops probably plays a major part in the distribution of the oedema fluid, partly due to the gravitational effect, but
Fig. 1. Ward unit chest film, localised view of the right base. Note a few septal lines at the edge of the lung: 'interstitial pulmonary oedema'.

Fig. 2. Ward unit chest film, localised view of the right lower zone. There is extensive interstitial pulmonary oedema.
also due to the height of the venous pressure, which is also related to the patient’s position. The shadows of alveolar oedema are very often transient and variable in their appearance and position from day to day.

This variability of appearance makes interpretation of these lung shadows difficult. Heard et al. (1968) showed that changes in the lung and pleura in left ventricular failure are quite complex. Pathologically they include interstitial and interalveolar non-fibrinous oedema and, occasionally, interalveolar fibrinous oedema with some interalveolar fibrosis. In addition, siderophages can be present in the alveoli, and there may be pulmonary infarcts, haemorrhages and very often small pleural effusions. The correlation between the appearance on the chest film, seen often days before demise, and post-mortem studies is not satisfactory since the day-to-day changes are so variable. Consequently, that which is seen at post mortem is usually due to terminal events and bears little relationship to the in vivo X-ray findings.

An occasional appearance of symmetrically distributed alveolar oedema close to the lung hila and mid zones, the so called ‘bats-wing’ shadows (Fig. 3) is sometimes seen, but is not very common. This central distribution is partly due to the considerable accumulation of interstitial fluid in the larger interstitial spaces, close to the lung hila and in the central core of the lung, and partly due to an accumulation of interalveolar oedema in the posterior aspects of the lungs (Heard et al., 1968). A further reason why peripheral oedema fluid may be cleared faster from the lungs than central oedema could be the more satisfactory lymphatic drainage at the lung periphery, together with the more extensive respiratory lung compression of the periphery.
PULMONARY VASCULAR APPEARANCES DUE TO PULMONARY VENOUS HYPERTENSION

If the pulmonary venous pressure is only slightly elevated, no observable changes will be noticed on the plain chest film. If the pulmonary venous pressure rises to a significant level, i.e. 20 to 25 mm Hg, the upper lobe pulmonary veins tend to dilate, whereas the lower lobe veins tend to constrict. There is a correlation between the level of the pulmonary venous pressure and the degree of upper lobe venous dilatation denoting blood flow diversion from the lower zones to the upper zones of the lung (West, 1965). The pulmonary arterial pressure rises secondarily to the elevated pulmonary venous pressure, leading to dilatation of the main pulmonary artery and main branches of the first and second divisions. The smaller peripheral branches will at first remain of normal calibre but, as the pulmonary arterial pressure rises further, the lower lobe smaller arteries will contract, whereas the upper and mid-zone smaller peripheral arteries remain normal. This effect of vasoconstriction at the base further enhances diversion of blood from the lower to the upper zones. This response of the pulmonary vasculature is in direct contrast to the normal physiology in man, where blood flow to the upper zones is smaller than blood flow to the lower zones of the lung (Hughes et al., 1968). These vascular appearances can be well demonstrated on pulmonary arteriograms (Steiner, 1964). The vascular changes are most marked in patients with a chronically elevated pulmonary artery and venous pressure, such as in mitral heart disease, but less well established in patients with ischaemic heart disease.

PLEURAL EFFUSIONS

In patients with left-sided heart failure pleural effusions are common, more frequently on the right than on the left side. The volume of pleural fluid can be considerable but can clear rapidly with a drop in the pulmonary venous pressure and improved cardiac function. Occasionally, slight pleural thickening will persist after the fluid has been absorbed, leaving obliterated costophrenic angles.

ABNORMAL BASAL LUNG SHADOWS AFTER ACUTE MYOCARDIAL INFARCTION

It has been shown by Tudor et al. (1973) that in 20 per cent of patients with acute myocardial infarction abnormal lung shadows develop at the lung bases within the first few days after the acute episode. These shadows are often linear or of segmental distribution, associated with minimal pleural reaction. It is for this reason that these shadows have been considered to represent pulmo-
nary infarcts secondary to peripheral venous thrombosis; this is understandable since venous thrombosis in patients with acute myocardial infarction is not at all uncommon (Maurer et al., 1971). Tudor et al. (1973) further studied the significance of these shadows and attempted to determine the incidence, aetiology and relationship between their appearances and the presence of venous thrombosis in the legs. They showed that there is no significant correlation between the incidence and timing of venous thrombosis in the legs with the timing of the appearances of these lung shadows. They usually appear on the first or second day after admission, whereas venous thrombosis happens very much later. The shadows clear rapidly, within 10 to 14 days, and are found with equal frequency in patients with and without isotope evidence of venous thrombosis. It is therefore apparent that these shadows are not necessarily related to pulmonary infarction from emboli derived from veins in the calf.

The early appearance of these shadows and their transient nature suggests that they are much more likely to be due to local pulmonary abnormalities. In the early stages of acute myocardial infarction, diaphragmatic splinting with shallow respiration is usual, coughing can be painful, and ventilation of the lung bases may be inadequate. These factors may well produce accumulation of secretions with subsequent plugging of small bronchi at the lung bases, giving rise to the appearance of local areas of consolidation with segmental pulmonary atelectasis. This sequence of events is the likely cause of basal lung shadows. With the patient’s improvement respiration becomes deeper, areas of collapsed lung expand and the chest radiograph returns to normal (Fig. 4a,b,c).

Although the appearances of these radiological abnormalities may raise the possibility of pulmonary infarction, they should not be used as evidence for instituting anticoagulant therapy, but rather emphasise the necessity for breathing exercises from the beginning of the illness.

Very occasionally, pulmonary infarcts may be present in a patient with acute myocardial infarction; usually the shadows due to such an infarct at the lung bases will occur a few days after admission and may be indistinguishable from the shadows described above. On the other hand, the clinical findings of chest pain, possibly haemoptysis and the occurrence of deep venous thrombosis in the calves and thigh may clinch the diagnosis. If isotope scanning is available, it may help to establish an accurate diagnosis. In the presence of basal lung lesions, multiple areas of inadequate perfusion of the lungs, not necessarily coincident with the lung shadows demonstrated on the radiograph, will support the diagnosis of widespread pulmonary vascular occlusion due to embolism.
Dressler's syndrome occurs usually some weeks after acute myocardial infarction. It may be associated with recurrence of chest pain, a rise in temperature and haemoptysis. The syndrome is not related to deep venous thrombosis or pulmonary embolism and it does not respond to anticoagulant therapy, but responds rapidly to steroid treatment.

The typical radiological appearances seen on the plain film are often bilateral, basal and ill-defined shadows due to a pneumonitis. The appearances of small pleural effusions at both bases, associated with a pleuritis and an increase in cardiac size due to a pericardial effusion, are also noted. The importance of the radiological recognition of this syndrome is to differentiate it from other more serious complications, such as massive pulmonary infarction, that may require pulmonary arteriography or scanning and sub-
sequent treatment with anticoagulants, or lung infection due to pneumonia. In this situation the differential diagnosis can be very difficult. The lung shadows in Dressler's syndrome tend to vary from day to day whereas those of pneumonia are much more slowly resolving and there will be no evidence of a pericardial effusion. On the other hand, pleural effusions are common in both conditions (Fig. 5a, b, c).

Once the diagnosis of Dressler's syndrome has been established on clinical and radiological grounds and the patient's treatment with steroids has begun,

Fig. 5. Ward unit chest films
(a) there is minimal pulmonary vascular congestion
(b) there are effusions at both bases obscuring the heart shadow and some consolidation and collapse in the right lower zone
(c) the lungs have expanded and the effusions have cleared.
rapid resolution of the pleural and pericardial effusions and of the lung shadows is common. Usually, it is the response of the patient to treatment that finally clinches the diagnosis.

Another difficulty in differential diagnosis is a further episode of myocardial infarction complicated by left ventricular failure. In this situation the basal shadows can be misinterpreted as pulmonary oedema associated with pleural effusions and the increase in heart size due to further dilatation of the left ventricle. On radiological grounds the absence of classical oedema as well as the absence of pulmonary vascular changes due to the elevation of pulmonary arterial and venous pressures may help one to arrive at the correct diagnosis.

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References

Dressler, W. (1959) Archives of Internal Medicine, 103, 28.
Heard, B. E., Steiner, R. E., Herdan, A. and Gleason, D. C. (1968) British Journal of Radiology, 41, 161.
Hughes, J. M. B., Glazier, J. B., Maloney, J. E. and West, J. B. (1968) Respiratory Physiology, 4, 58.
Maurer, B. J., Wray, R. and Shillingford, J. P. (1971) Lancet, 2, 1385.
Norris, R. M., Caughey, D. E., Deeming, L. W., Mercer, C. J. and Scott, P. J. (1970) Lancet, 2, 485.
Steiner, R. E. (1964) American Journal of Roentgenology, 91, 249.
Tudor, J., Maurer, B. J., Wray, R. and Steiner, R. E. (1973) Clinical Radiology, 24, 365.
West, J. B., Dollery, C. T. and Heard, B. E. (1965) Circulation Research, 17, 191.
West, J. B. (1965) Ventilation/Blood Flow and Gas Exchange. London: Blackwell Scientific Publications.