RADIOLOGICALLY recognizable anatomic changes in the lung may accompany any of the conditions responsible for pulmonary edema. Most of the roentgen findings reflect the presence of edema fluid either in the alveolar air spaces (alveolar edema) or within the interstitial connective tissue framework of the lung (interstitial edema). When the edema is cardiac in origin, as in patients with mitral stenosis or left ventricular failure, roentgen evidence of pulmonary venous hypertension is usually present and often precedes the findings of pulmonary edema.

The roentgen manifestations of congestive failure depend to a large extent, on the degree of elevation of the pulmonary venous pressure. Pulmonary edema of the alveolar variety is associated with relatively severe venous hypertension and usually does not appear before the diagnosis of failure is obvious to the clinician. Minimal increase in venous pressure often results in a redistribution of the pulmonary blood flow. All that may be seen on the chest films is a subtle hyperfusion of the upper lobes of the lung at the expense of the lower lobes. Between these two extremes lie the manifestations of interstitial pulmonary edema.

Simon\(^1\) classifies as “moderate” pulmonary hypertension with a mean venous pressure ranging from 18 to 25 mm Hg. It is at this level, before the appearance of alveolar edema and the clinical signs of failure that the signs of interstitial pulmonary edema may be expected to appear.\(^1-3\) The latter are detectable only on the basis of the roentgen findings and are much more frequently encountered than is the butterfly appearance of alveolar edema. In our own series of 94 patients admitted to a coronary care unit following myocardial infarction\(^4\) one third had roentgen findings of congestive heart failure without the presence of symptoms or of signs on physical examination. Three quarters of the patients with positive roentgen findings subsequently developed clinical signs of congestive failure.

From the Department of Radiology, Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania.

Address for reprints: Dr. Arnold Chait, Hospital of the University of Pennsylvania, 3400 Spruce Street, Philadelphia, Pennsylvania 19104.
Whereas selective upper lobe vascular dilatation was the most reliable early sign of failure in our series, roentgen signs of interstitial edema were present in about half of the patients in incipient failure; none of these showed evidence of alveolar edema by X-ray.

The interstitial tissue of the lungs casts a shadow of soft-tissue density which is set off by the more radiolucent air in the adjacent alveoli. Actually, the septa of the interstitium are so thin that their shadows are very faint and normally cannot be identified as individual lines. However, when the tissue becomes edematous, its roentgen density increases and several types of shadows appear on the chest film.

1. Septal Lines

These lines represent thickened interlobular septa (figs. 1–4). They are named after Peter Kerley, who first described them and divided them into three types, designated simply as A, B, and C lines. When the lines are the result of pulmonary congestion they are often transitory and disappear without a trace following treatment of the cardiac failure.
They do not bifurcate and they do not follow the normal branching pattern of bronchi and vessels." These lines are most commonly seen in the upper lobes, are slightly curved rather than straight, and are usually longer than B lines, reaching up to 4 cm in length.

**B lines** (figs. 2, 4) are “short, sharp lines seen only at the bases, usually less than an inch long and running transversely outward to touch the pleural margin.” These are the most commonly recognized of the Kerley lines. Although, as originally described, they are usually in the costophrenic angles, they have been reported as high as the apex of the lung. B lines ordinarily do not branch. They may be as wide as 0.2 cm and are rarely longer than 3 cm.

**C lines** (fig. 3) are “fine interlacing lines giving a network appearance.” They are not commonly seen, but when present can appear in any portion of the lung.

The septal lines, and specifically the B lines, are the best-known roentgen signs of interstitial pulmonary edema. However, they are relatively uncommon in patients in a coronary care unit, having occurred in our series in only 12% of patients in clinical failure and in 10% of those with subclinical failure.

### 2. Perivascular Cuffing

This sign (figs. 5, 6) and the two following are reflections of the association of interstitial edema fluid with adjacent bronchi and blood vessels. Medium-sized pulmonary vessels, whether arteries or veins, are normally seen on chest films either end on or longitudinally as sharply defined columns of soft-tissue density, clearly outlined by air in the surrounding lung. A collection of interstitial pulmonary fluid will tend to blur the normally sharp outline of these structures and to cause an apparent widening accompanying the loss of definition. This may also give rise to a generalized nodular appearance throughout the lungs, representing “cuffing” of many medium-sized vessels seen end on.

### 3. Peribronchial Cuffing

Medium-sized bronchi, if visualized at all on a normal chest roentgenogram, are appreciated only when seen end on and then only as...
very thin-walled structures. With the accumulation of fluid in the interstitium surrounding the bronchi, the bronchial walls become increasingly prominent but lose their distinct margination (fig. 5). The interstitial fluid blends with the bronchial wall and the resultant appearance is one of a thick-walled hollow tube.

4. Subpleural Thickening

Fluid in the interstitium may extend into the subpleural interlobular septa and present on film as apparent pleural thickening (fig. 4). Radiographically, this may be impossible to differentiate from a true interlobar fluid collection.

5. Perihilar Haze

This sign (figs. 4, 6) results from loss of the sharp definition of the large central pulmonary vessels and is best observed on the frontal film in the right hilar area. Although the increased density of the hilar shadow is due in part to the engorgement of the perihilar veins that

Figure 3

C lines of Kerley. (Left) The arrows indicate a diffuse network of curvilinear streaks throughout the lung, haphazardly arranged, slender, and occasionally branching, representing Kerley C lines. B lines are noted in the lung base. No rales were heard. (Right) Five days later, following treatment, virtually all trace of these lines has disappeared, and the chest film is normal.
A lines of Kerley, B lines of Kerley, subpleural thickening, and perihilar haze. (Left) During a clinically undetectable episode of congestive failure, Kerley B lines are seen in the base as short, sharp, nonbranching, dense radiopaque streaks (vertical straight arrows). Subpleural edema is indicated in the area of the horizontal fissure by the broad solid arrows. (This is virtually impossible to distinguish from a small accumulation of fluid within the fissure). The right descending pulmonary artery branch (slender curved arrows) is indistinctly marginated because of fluid in the interstitium adjacent to this vessel destroying the vessel-air interface. Kerley A lines are indicated by the broad open arrows in the upper lobe. These are somewhat longer than the B lines, radiate from the hilum, and are often curvilinear. These are less frequently seen than are the B lines. (Right) After treatment of congestive failure, the subpleural collection has diminished in prominence (broad closed arrow). The Kerley A and B lines have disappeared. The descending pulmonary artery branch (slender curved arrows) is now sharply marginated. The time interval between figure 4, left and figure 4, right is 7 days, but a similar reversion to normal may be seen on films made only hours apart.
Figure 5
Perivascular and peribronchial cuffing. (Left) During an episode of congestive failure, a medium-sized upper lobe pulmonary artery branch is seen end on, larger in diameter than during its normal state, and indistinctly marginated (broad open arrow). A medium-sized bronchus is seen just below this vessel (slender arrows). It has an apparent thick wall and is poorly marginated, due to the presence of peribronchial interstitial edema. (The foreign body overlying the left upper lobe is a pin in the patient's gown.) (Right) Following bed rest, the left upper lobe vessel is still seen (open arrow) but is smaller in size and well marginated, and the bronchus is all but invisible. Edema fluid has left the interstitium.
Figure 6

Generalized loss of translucency, perihilar haze, and vascular cuffing. (Top) During an episode of congestive failure with interstitial edema, there is a generalized increase in lung density, and the perihilar and basilar portions of the lung, particularly, appear veiled and hazy. Vascular (Continued on next page)
cuffing is particularly well demonstrated in a vessel seen end on in the left upper lobe (open arrows). The vascular margins are unsharp, and the vessel appears larger than normal. A diffuse, poorly defined nodularity due to perivascular edema about innumerable smaller vessels seen end on is present throughout both lungs. A large descending right hilar artery (curved arrows) is indistinctly seen because of loss of the normal vessel-lung interface. (Bottom) Following treatment, the left upper lobe vessel (open arrows) and the right hilar artery (curved arrows) are now seen to be sharply defined and of smaller caliber. This decrease in caliber is in part real, but in part also due to the disappearance of adjacent interstitial fluid. The generalized nodularity is no longer seen.

accompanies left heart failure, the blurring of the normally margined vessels indicates, in addition, a swelling of the interstitium due to the accumulation of edema fluid. When alveolar edema is present, the shadows of the blood vessels are usually totally obscured.

6. Generalized Loss of Translucency

This sign (fig. 6) consists of a slight, generalized increase in the radiologic density of the lungs, particularly at the bases, due to the interstitial accumulation of fluid. This finding may be difficult to differentiate from that of early alveolar edema.

The recognition of all of the above signs of interstitial pulmonary edema is dependent upon meticulous and reproducible roentgen technic. Whether films are made in the X-ray department or at bedside, exposures must be rapid (0.05 sec or faster) so that the structures are not blurred by respiratory motion and do not mimic perivascular and peribronchial cuffing or perihilar haze. Motion can totally obliterate Kerley A and C lines and probably B lines as well. A generalized loss of translucency can be simulated by an underexposed film and obscured by an overexposed one.

References


Interstitial Pulmonary Edema
ARNOLD CHAIT

Circulation. 1972;45:1323-1330
doi: 10.1161/01.CIR.45.6.1323

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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