Progress in Cardiovascular Diseases

Progress in CARDIOVASCULAR DISEASES

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Diagnostic Methods

The Roentgenology of Pulmonary Manifestations in Mitral Heart Disease and Left Heart Failure

By R. E. Steiner

A CAREFUL STUDY of the radiologic appearances of the lungs and correlation with the clinical data can frequently produce information of the greatest value for the diagnosis and prognosis of disordered lung function due to pulmonary or heart disease. It is possible to accurately correlate the radiologic appearances seen on chest radiographs, and more convincingly on angiographic studies, with hemodynamic findings, the clinical picture and also the pathologic data in mitral heart disease and in left ventricular failure, irrespective of the cause.

Since the pulmonary circulation is so closely related to heart and lung function it is important, from the radiologic point of view, not only to study the pulmonary appearances, but also the heart and to look upon both organs as a single physiologic unit. In the following discussion the main emphasis will be placed on the radiologic appearance of the lungs, but at the same time the heart will be mentioned, in less detail.

Technic

Before I proceed to a discussion of the radiologic appearances it is important to briefly mention some technical points.

Chest radiograph.—For the radiologic study of the pulmonary vascular pattern a chest radiograph of good quality is essential. The film must be adequately penetrated to outline the vascular shadows behind the heart, and its contrast must also be sufficient to produce a clear, sharp outline of the main pulmonary vessels and smaller branches. To achieve this, radiographic factors must be carefully chosen, speed of exposure being the most important to avoid unsharpness due to movement, particularly cardiac pulsation. A high kv. technic with milli-second exposures is probably the most satisfactory combination.

Tomography.—This method of examination is of considerable value for the study of the larger pulmonary vessels. The multi-section technic of tomography should be used since it imparts considerable less radiation to the patient than single-film tomography.

Contrast studies.—Selective pulmonary arteriography is the method of choice for a detailed examination of the pulmonary vasculature. Should pulmonary artery catheterization prove impossible, venous angiocardiography is also satisfactory to outline the pulmonary arteries and veins.

Mitral Heart Disease

Specific lung lesions have been recognized for many years in mitral heart disease and they have been associated with disordered pulmonary function.

In 1936 Parker and Weiss[47] were able to demonstrate pathologic changes

in the walls of small pulmonary arteries and arterioles and also in alveolar walls in patients with severe mitral stenosis. The lumen of the pulmonary arterioles was reduced by muscular hypertrophy, frequently associated with thickening of the intima and some medial scarring. The alveolar walls were edematous, there was thickening of the basement membrane and there was evidence of interstitial edema in interlobular septa. Similar changes were reported by Larrabee, Parker and Edwards,[40] Bayliss, Etheridge and Hyman,[4] Evans and Short[21] and Harrison.[32]

Radiologic Appearance of the Lungs in Mitral Heart Disease

These will be considered under three main headings: (1) changes in the pulmonary vascular pattern involving arteries and veins; (2) hemosiderosis associated with a fine pulmonary nodulation; pulmonary ossification associated with denser and larger pulmonary nodules; and (3) pulmonary edema; this can either be acute and interalveolar or subacute or chronic, and interstitial.

Radiologic appearances of the pulmonary vascular changes in mitral valve disease.—Since the original measurements of pulmonary artery pressures in patients with mitral stenosis by Bloomfield, Lawson, Cournand, Breed and Richards,[6] pulmonary hypertension has been found to be an almost invariable complication of severe mitral stenosis.

Since the vascular changes are so closely related to pulmonary hypertension, one must consider a radiologic classification which will permit a correlation of the radiologic appearances of the pulmonary arteries and veins with the cinical, hemodynamic and pathologic findings.

RADIOLOGICAL CLASSIFICATION OF PULMONARY HYPERTENSION DUE TO INCREASED RESISTANCE. (1) Cardiac or pulmonary lesions associated with an increased precapillary pulmonary resistance will produce a radiologic pulmonary vascular pattern of arterial hypertension. (2) Conditions associated with an increase postcapillary resistance will produce a radiologic pulmonary vascular pattern of venous hypertension.

A. Causes of Precapillary Pulmonary hypertension

Arterial hypertension due to obstruction of the pulmonary blood flow at small pulmonary artery and arteriolar level

a. Secondary to postcapillary hypertension
   1. mitral stenosis
   2. Left ventricular failure

b. Vasoconstrictive
   1. mitral stenosis
   2. congenital heart disease, (atrial septal defect, ventricular septal defect, persistent ductus arteriosus and aortopulmonary septal defect)
   3. anoxic pulmonary heart disease

c. Obliterative
   1. chronic obliterative lung disease (pulmonary fibrosis and parenchymal destruction)
   2. arteritis (polyarteritis nodosa, lupus erythematosus, rheumatic arteritis)
   3. packed pulmonary emboli

4. congenital heart disease (atrial septal defect, ventricular septal defect, persistent ductus arteriosus, aortopulmonary septal defect
5. atheroma (rare)

B. Causes of Postcapillary Pulmonary Hypertension

Venous hypertension is due to obstruction of the pulmonary blood flow at left ventricular, left atrial or pulmonary venous levels

a. Mitral valve disease
b. Left atrial tumour or ball-valve thrombus
c. Pulmonary venous thrombosis
d. Left ventricular failure
e. Constrictive pericarditis

Radiologic Appearance of the Pulmonary Arteries in Mitral Heart Disease

The abnormal radiologic appearances of the pulmonary vascular pattern in mitral heart disease are caused by a rise in the pulmonary arterial pressure, probably secondary to an elevated pulmonary venous pressure. These vascular changes were first observed radiologically in angiographic studies of the pulmonary vasculature by Goodwin, Steiner and Lowe[26] and Actis-Dato, Angelino and Zambellini.[1] In subsequent years a large number of papers appeared in the literature confirming their observations.[7,9,59] Some of these authors used selective pulmonary arteriography in their investigations.

The angiographic appearances of the pulmonary arteries and pulmonary artery pressure levels were correlated with the vascular appearances seen on chest radiographs by Davies, Goodwin, Steiner and Van Leuven,[13] Fleischner and Sagall,[23] Van Epps[61] and Boyd, Scott-Park and Smith.[8] All these authors were able to observe a satisfactory correlation. Although Bülow et al.[9] were also able to find a satisfactory correlation between pressure levels and vascular changes, they felt that interpretation and accurate assessment of the pulmonary vascular changes on chest radiographs, particularly at the lung bases, was at times very difficult.

In patients with mitral heart disease and a normal pulmonary artery pressure, the pulmonary vascular pattern on chest radiographs or on contrast studies will be quite normal. There is no dilatation of the main pulmonary artery or of its main branches. The smaller divisional pulmonary artery branches well out in the peripheral lung fields divide equally and regularly, and their caliber is proportional to the larger arterial branches from which they arise (fig. 1A and B).

When the pulmonary arterial pressure is elevated to approximately 60 to 70 mm. of mercury systolic, marked dilatation of the main pulmonary artery will be noted, whereas the small peripheral pulmonary arterial branches appear narrowed, irregular and often tortuous, with ill-defined borders. These changes in caliber and contour occur at levels of the third to fourth divisional branches beyond the main lobar arteries. The arterial narrowing is most marked in the lower and mid zones of the lungs, whereas the vascular pattern in the upper zones remains perfectly normal (figs. 2A and 2B).[13,17] There

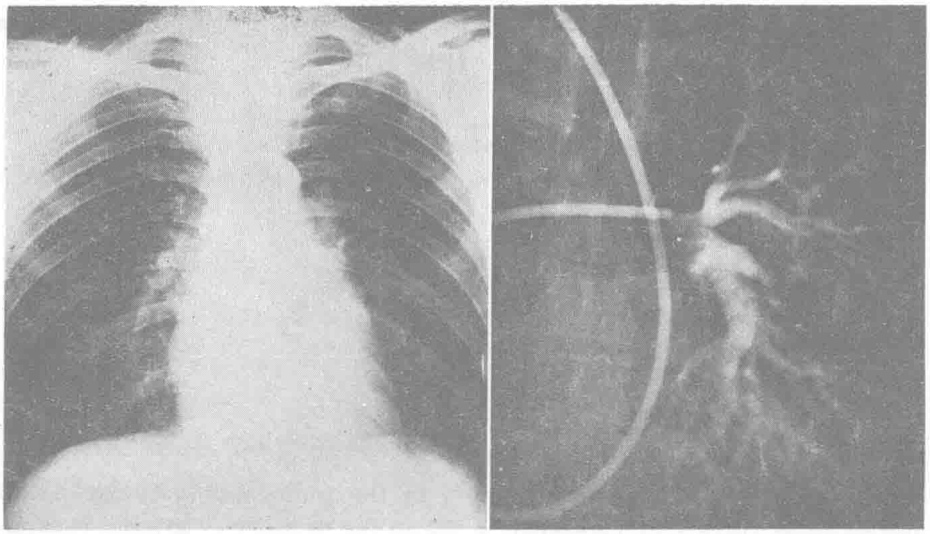


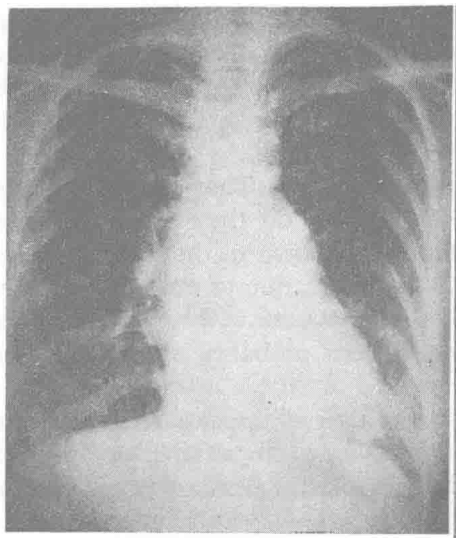
FIG. 1A *(at left).*—Mitral stenosis. Normal pulmonary artery and pulmonary venous pressures. Normal pulmonary vascular pattern.

FIG. 1B *(at right).*—Mitral stenosis. Pulmonary arteriogram. Normal pulmonary arterial pattern.

is close agreement between in vivo and postmortem angiographic studies of the lung in mitral heart disease.[32] This author has shown that in the majority of cases the arterial narrowing was due to muscular elastic hypertrophy secondary to increased vascular tonus and only in a minority of cases was it due to irreversibe atheromatous narrowing. The discrepancy in the appearances of the upper and lower lobe arteries was also clearly demonstrated in postmortem studies and correlated well with pressure levels and in vivo angiograms.[17]

To explain this descrepancy in the appearances of the smaller upper and lower zone pulmonary arteries, Doyle and his colleagues put forward the following suggestion. The left atrial and pulmonary venous pressure is markedly raised in tight mitral stenosis. The pulmonary hydrostatic pressure in the erect posture is higher at the bases of the lungs than at the apices. If the hydrostatic pressure increment is added to the already markedly raised pulmonary venous pressure, a critical level will be reached at the bases of the lung, but not at the apices. This hemodynamic situation may be one of the causes for arterial constriction in the lower and mid zones of the lung, thus diminishing blood flow, and preventing the onset of acute pulmonary edema. The same authors stress the difference in appearance of the pulmonary arterial patterns in congenital heart disease, complicated by the Eisenmenger reaction, for further support of their explanation. In this congenital group, arterial narrowing of the smaller peripheral arteries is uniform throughout the lung fields. No selective arterial response appears to be involved since the pulmonary venous pressure in these patients is either normal or only slightly elevated.

With moderate elevation of the pulmonary arterial pressure to levels below



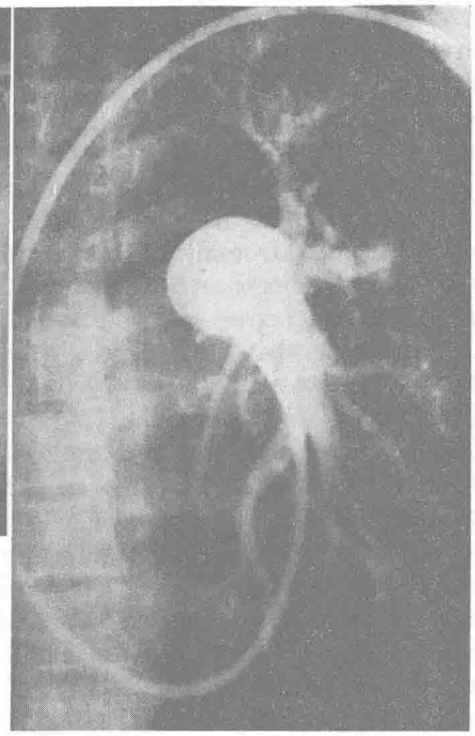


FIG. 2A *(at left).*—Mitral stenosis. Severe pulmonary hypertension (Pulmonary artery pressure, 105/45 mm. of Hg.) Abnormal pulmonary vascular pattern.

FIG. 2B *(at right).*—Mitral stenosis. Severe pulmonary hypertension. Pulmonary arteriogram. Normal peripheral upper lobe arteries. Markedly narrowed peripheral lower lobe arteries.

60 mm. of mercury systolic the vascular changes described above also occur. They are much less marked and are confined to the lung bases. In this group of patients interpretation and correlation of the vascular changes in the pulmonary artery pressures is difficult and not very reliable.

Main pulmonary artery enlargement was originally described by Zdansky,[64,65] and put into perspective in mitral heart disease by Parkinson.[48] In a follow-up study of 351 patients Olesen[46] noted prominent hilar shadows in patients with severe mitral disease. Jacobson, Schwartz and Sussmann[37] found that correlation of main pulmonary artery size and pulmonary artery pressure was not altogether satisfactory. On the other hand, Davies et al.[13] showed that significant elevation of the pulmonary artery pressure to levels of approximately 60 to 70 mm. of mercury systolic was associated with considerable dilatation of the main pulmonary artery, whereas a normal pressure was associated with a normal size.

It is impossibe radiographically to distinguish between pulmonary arterial narrowing due to vascular spasm and narrowing due to secondary arterial disease such as atheroma or pulmonary artery thrombosis. Massive pulmonary artery thrombosis of a large branch generally occurs secondary to peripheral thrombosis or due to primary embolism. Associated heart disease, for instance, mitral stenosis, is quite common.[3] These authors reported six cases of mitral heart disease out of a total of 53 patients with pulmonary artery thrombosis. Gross dilatation of one main pulmonary artery branch or a lobar

artery, with very sudden narrowing of the branches distal to it and peripheral oligemia in the affected segment, may suggest the diagnosis of pulmonary artery thrombosis. Occasionally calcified atheroma is visible at the edge of the distended artery; this will produce a linear opacity outlining the border of the vascular shadow.

The arterial changes in mitral stenosis are not static. They usually progress as stenosis increases in severity. Only occasionally do they seem to regress after successful valvotomy.[27] These authors have shown in their postoperative studies that radiologic arterial changes in the lungs, due to pulmonary hypertension, did not regress in two-thirds of their patients with good or fair result, suggesting that some of the changes were probably organic and not entirely due to vascular spasm.

There is no doubt that the study of the arterial appearances is a most valuable method to estimate by radiologic means pulmonary artery pressure levels and also to help in the pre- and postoperative assessment of patients with mitral valve disease.

The pulmonary veins.—Pulmonary veins are not easily seen on chest radiographs. Only the large veins, particularly those of the right lower lobe, are visible on most films. The left lower lobe veins are completely obscured by the heart shadow; upper lobe veins are only occasionally seen. Tomography and contrast studies are the methods of choice for the study and demonstration of the pulmonary veins.

In patients with a normal or only slightly raised pulmonary venous pressure, the pulmonary veins appear of normal caliber. Venous constriction in some cases with a high pulmonary venous pressure has been observed on plain chest radiography by Steinbach, Keats and Sheline.[57] Considerable dilatation of the upper lobe veins was described by Simon.[55] In patients with markedly elevated venous pressure, with a pulmonary capillary pressure level from 35 to 40 mm. of mercury, Steiner[60] was able to show on angiographic studies slight dilatation of the upper lobe veins whereas the lower lobe veins were either of normal caliber or slightly narrowed. At pulmonary capillary pressure levels of 15 to 20 to 25 mm. of mercury, the upper lobe veins were of normal caliber. At normal pressure levels, pulmonary veins in all lobes were of normal caliber. These observations of venous changes in tight mitral stenosis with high venous pressures were confirmed by Arvidson and Odman.[2] In postmortem studies of the pulmonary veins Harrison[32] was able to show marked thickening of their walls in the lower lobes whereas in the upper lobes they were of normal thickness, thus somewhat comparable to the arterial changes.

Pulmonary Edema in Mitral Heart Disease

This can be either (1) acute and interalveolar or (2) subacute or chronic and interstitial.

Interalveolar edema.—This complication occurs fairly commonly in tight mitral stenosis. It was reported in 8 per cent of all patients in Wood's series.[63] Pulmonary edema will produce diffuse, ill-defined shadows in the lungs, which may become confluent. The shadows are often widespread,

affecting both lungs, or they may be localized to one lung or even to one lobe. Occasionally the distribution of the edema is central and symmetric, confined to the central core of the lungs, the so-called "bats wing" shadows of Hodson[35] and Jackson.[36] Occasionally a rather unusual distribution can be seen in mitral heart disease in which the edema is localized to the apical segment of the lower lobes and to the posterior basal segments of the upper lobes. The distribution is the reverse of that of arterial hypertrophy and arterial narrowing. This uncommon distribution, seen on chest radiographs, was confirmed on postmortem studies by Harrison.[32]

The exact cause for the variable distribution of acute pulmonary edema and its mechanism is not yet fully understood. Many factors are involved, such as the plasma osmotic pressure, pulmonary capillary pressure, the condition of the alveolar basement membrane and probably also the hydrostatic pressure and posture of the patient, preceding and during the attack. The mechanism will be further discussed below under pulmonary edema in acute ventricular failure.

The shadows of acute pulmonary edema can appear rapidly within hours of the onset and they can disappear equally rapidly with adequate treatment. Since small interlobar and small pleural effusions are a quite frequent accompaniment, the shadows due to pulmonary edema may be partly obscured, particularly if they are basal in distribution.

Interstitial pulmonary edema.—This is the more common type and is frequently seen in mitral heart disease. Interstitial pulmonary edema produces a distinct radiologic picture associated with pulmonary venous hypertension.

The most important radiologic signs of interstitial edema are the so-called "septal lines." They are dense, horizontal, linear shadows at the lung edges with a predominantly basal distribution. These lines were first described by Kerley,[38] who ascribed them to dilated pulmonary lymphatics and called them "B" lines. The septal lines can vary in length from 1 to 3 cm.[22] They are often accompanied by straight, or slightly curved, longer, dense lines, and tend to radiate from the periphery towards the lung hilum. These longer lines were also described by Kerley,[38,39] who called them "A" lines; they too are due to distended interlobular septa (fig. 3A and B).

A number of radiologic and pathologic studies have produced convincing evidence that these lines are due to the distention of interlobular septa with edema fluid, but not to distended pulmonary lymphatics, although lymphatics can be found within the septa.[22,28-30,53] Anatomically the peripheral interlobular septa are arranged perpendicular to the pleural surface of the lung, which is the reason for the radiographic appearances of the basal septal lines. Since edema fluid accumulates within the interstitial tissue only and leaves the bordering alveolar clear, the distended septa are not obscured and cast the characteristic radiographic shadow.[30]

Several observers have correlated pulmonary venous pressure levels with the appearance of septal lines and found that below a critical level of approximately 20 mm. of mercury, pulmonary capillary wedge pressure septal lines rarely occur.[11,29,30]

A drop in the left atrial pressure and pulmonary venous pressure, following

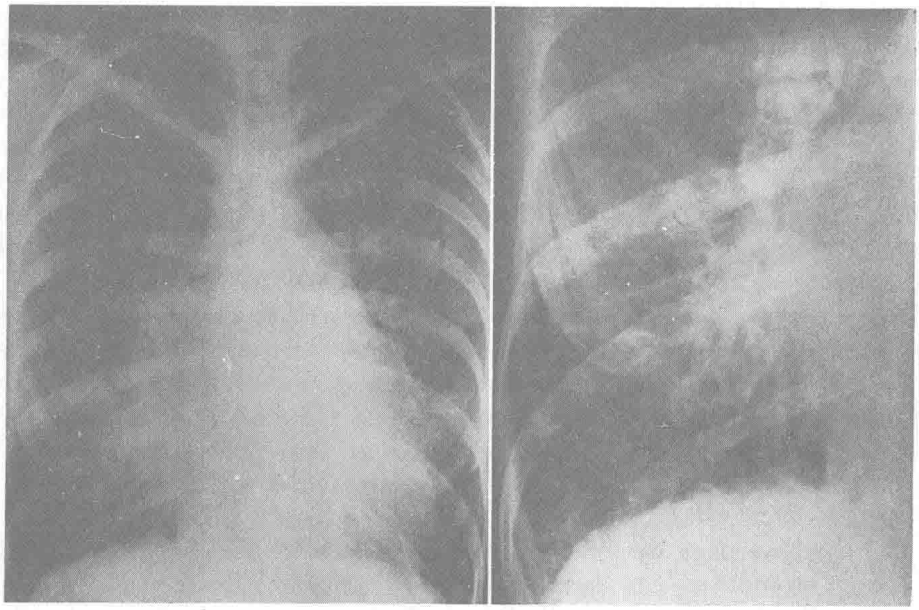


FIG. 3A *(at left).*—Mitral stenosis. Severe pulmonary hypertension. Marked interstitial pulmonary edema. Septal lines.

FIG. 3B *(at right).*—Same patient as in figure 3A. Localized view of right lower zone. Septal lines. Generalized haziness due to interstitial pulmonary edema.

successful valvotomy or medical treatment, can lead to rapid disappearance of interstitial pulmonary edema and aso of septal lines.[29] Persistence of septal lines after adequate treatment can be due to accumulation of hemosiderin in the septa.[22] Septal lines can occur in other conditions in which the pulmonary venous pressure is raised, as for instance in left ventricular failure (this will be discussed later) and rarely in atrial septal defects. Lymphatic obstruction is yet another cause for their appearance, as for example, in malignant disease associated with lymphangitis carcinomatosa, or reticulosis with mediastinal and hilar glandular enlargement and lymphatic obstruction. Rarely do septal lines occur in pneumoconiosis,[28] or in other forms of pulmonary heart disease.

Another manifestation of interstitial pulmonary edema is the subserous accumulation of edema fluid which may extend along the interlobar fissures and can give rise to considerable pleural thickening. This fluid retention will be responsible for some loss of radiologic translucency in the affected areas and will produce a generalized haziness and ill definition of the lung texture. Further spread of the interstitial edema into the central areas of the lung will further add to the haziness of the chest radiograph. This loss of definition is one of the main reasons why interpretation of the pulmonary vascular markings at the lung bases can be so difficult.[9,53]

Summary of Radiologic Signs of Pulmonary Arterial Hypertension in Mitral Stenosis

(1) Enlargement of the main pulmonary artery; (2) normal peripheral upper zone arteries, constricted lower and occasionally also mid-zone arteries.

Pulmonary Venous Hypertension

(1) Normal or dilated upper zone veins; (2) normal or constricted lower zone veins; (3) septal lines; (4) thickened interlobar fissures and haziness of the lower zones of lung.

Correlation of the Radiologic Signs of Pulmonary Hypertension with the Appearance of the Heart Shadow in Mitral Disease

The correlation of left atrial size with the severity of pulmonary hypertension is not good.[27,44] In tight mitral stenosis with a high pulmonary artery pressure and vascular changes in the lungs, slight left atrial enlargement is usually seen; occasionally, however, the atrium can be very large or even of aneurysmal proportions.

The radiologic signs of pulmonary venous hypertension can be present in predominant mitral stenosis or in predominant mitral imcompetence. The radiologic signs of marked pulmonary arterial hypertension are not seen in predominant incompetence, and always indicate tight mitral stenosis. Patients with tight mitral stenosis with the radiologic signs of severe pulmonary venous hypertension usually do well after successful valvotomy. These signs are a good indication for operation.[29]

No change in the signs of venous hypertension, following operation, can mean an unsuccessful split of the valve, or possibly hemosiderin deposits within the septa. Improvement in the signs after operation, with later recurrence, may indicate restenosis of the valve.

When tricuspid insufficiency develops, the pulmonary vascular changes, due to a high venous and pulmonary artery pressure, can regress markedly and this may result in a relatively normal lung vascular pattern.

Hemosiderosis

Hemosiderin agregates in the lung parenchyma will produce shadows on chest radiographs, provided the deposits are large enough and fairly widely distributed. The shadows appear as a fine nodulation, which is most marked in the mid-zones and lower zones of the lungs, frequently leaving the upper zones free (fig. 4). Very slight changes of hemosiderosis may be so indistinct that a definite radiologic diagnosis cannot be made.

Following extensive studies of secondary pulmonary hemosiderosis, Scott, Park and Lendrum[54] and Lendrum, Scott and Park[42] concluded that this condition resulted from multiple, minute, intrapulmonary hemorrhages which had occurred at the site of varicose bronchopulmonary capillary anastomosis. These hemorrhages had ruptured into the terminal bronchioles; this led later to an accumulation of hemosiderin within the lung, the deposits being partly interalveolar and partly interstitial.

The incidence of hemosiderosis in mitral heart disease is about 10 to 15 per cent in all cases.[27] Some correlation between hemosiderosis and pulmonary hypertension was recorded by Steiner and Goodwin[59] and Goodwin et al.[27]; Wood[63] could find no such correlation.

Once pulmonary hemosiderosis is established, it does not regress; the radiographic shadows persist, often remaining static for many years, but on

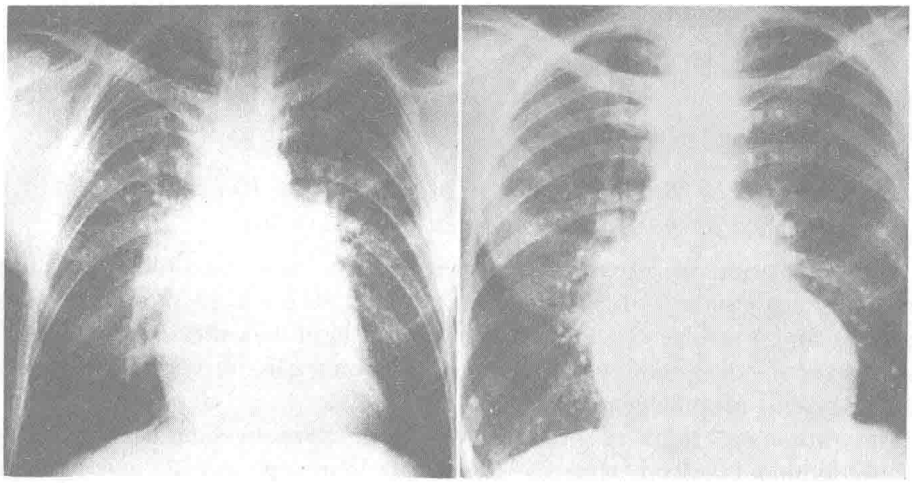


FIG. 4 *(at left).*—Mitral stenosis. Normal pulmonary artery pressure. Marked hemosiderosis.

FIG. 5 *(at right).*—Mitral stenosis. Moderately elevated pulmonary artery pressure. Pulmonary ossification. No evidence of hemosiderosis.

long-term follow-up examinations, considerable progression and extension of the shadows is occasionally noted.

Differential diagnosis of hemosiderosis.—Other pulmonary conditions producing a fine nodular pattern must be considered in the differential diagnosis, as for example miliary tuberculosis, some forms of pneumoconiosis and idopathic hemosiderosis. The association of a mitral configuration of the heart with a fine pulmonary nodulation will in most instances establish the diagnosis.

Pulmonary ossification.—Pulmonary ossification was first described by Salinger.[51] This author also associated this phenomenon with mitral valve disease. A large number of case reports has appeared in the literature since the first description of this unusual manifestation.[19,31,39,42] The literature, etiology and pathology were extensively reviewed by Whitaker, Black and Warrack[62] and Fleming and Robinson.[24]

The nodules are true lamellar bone and probably develop in areas of long-standing interstitial pulmonary fibrosis and as a sequel to long-standing interstitial pulmonary edema. Hemosiderin was absent or very slight in some cases reported, so that a causal relationship between hemosiderosis and pulmonary ossification, suggested by Lawson[41] and Ellman and Gee,[10] is most unlikely.

The ossified nodules cast dense shadows in the peripheral lung fields. There is considerable variation in size, from a pinpoint to a fairly large nodule a few millimeters in diameter. The opacities are round or oval, sometimes have an irregular border, but always with a sharp definition (fig. 5). The nodules are usually distributed over the bases and in the mid-zones of the lungs. That nodules can appear in a relatively short time, during periods of observation of a few months, has been recorded. Some observers have also been able to show that pulmonary ossification is nearly always associated with tight mitral stenosis and a high pulmonary artery pressure.[24,62]

Differential diagnosis of mitral heart disease associated with pulmonary hypertension.—Two conditions must be considered, since they may mimic mitral heart disease very closely: (1) left atrial tumor and (2) left atrial ball-valve thrombus.

Significant radiologic features will be seen in these two uncommon conditions only if the tumor mass or thrombus is large enough to obstruct the mitral valve orifice sufficiently to produce a rise in the left atrial and in the pulmonary venous pressures. Steinberg, Dotter and Glenn[58] and Steiner[60] have shown that the radiologic appearances of the heart and the appearance of the pulmonary vascular pattern can be very similar to those seen in tight mitral stenosis with a raised pulmonary venous and pulmonary artery pressure. The severity of the vascular changes depends on the severity of the valve obstruction.

On routine chest radiographs it is impossible to differentiate between mitral heart disease and the other two conditions. The clinical findings, however, may be very suggestive of the diagnosis of left atrial tumor or possibly a ball-valve thrombus. Angiocardiography is the method of choice in the diagnosis, since by this method a mass in the left atrial cavity will be demonstrable as a constant filling defect.[25] Occasionally marginal calcification in a large atrial thrombus may occur, and this will then be visible radiologically as linear shadows at the edge of the left atrium.

Left Ventricular Failure

A distinct radiologic picture will be seen in left ventricular failure, irrespective of its etiology. Among the important causes of left ventricular failure one must consider hypertension, ischemic heart disease and aortic valve disease. The less common causes are thyrotoxic heart disease, myxedema and severe and prolonged anemia.

Radiologic Appearances of the Lungs

The radiologic appearances of the lungs are best considered from two aspects: (1) acute left heart failure, which results in acute pulmonary edema; (2) chronic left heart failure, which presents a very diverse radiologic picture.

Pulmonary edema.—To date the exact mechanism of pulmonary edema is not fully understood. A number of different factors is involved; the predominant one largely depends upon the underlying etiology. Drinker[18] and Cameron[10] in their discussion of the pathologic physiology of pulmonary edema considered the following important factors responsible for the formation of pulmonary edema: (1) the efficiency of the pulmonary venous drainage, (2) the pulmonary capillary pressure, (3) capillary permeability and the colloid plasma osmotic pressure and (4) the hydrostatic pressure.

Pulmonary edema in cardiac failure is mainly due to increased pulmonary capillary pressure, and to inadequate pulmonary venous drainage. The pressure must be sufficiently high to overcome the normal plasma colloid osmotic pressure of approximately 25 mm. of mercury. This simple relationship, however, does not always apply since Drinker[18] in experimental studies

and Hayward[33] on clinical evidence were able to demonstrate higher pulmonary capillary pressures than 25 mm. of mercury without a significant accumulation of intra-alveolar edema fluid. Thickening of the alveolar and capillary walls[47] may be partly responsible for this discrepancy. It was suggested by Grainger[30] that the capillary permeability may partly increase due to anoxia and so permit protein-rich edema fluid[18] to accumulate in the alveolar spaces. Grainger also suggests that overloading of the pulmonary venous drainage system by excessive fluid production in heart failure will also lead to edema formation.

Radiologic appearances of acute pulmonary edema.—It is not always possible to study patients adequately since they may be too ill to be x-rayed in the department, and only ward unit films may be available for interpretation. Acute pulmonary edema in mitral stenosis has already been discussed.

Alveolar accumulation of the edema fluid usually gives rise to widespread, ill-defined shadows; these shadows may be confluent, simulating large areas of pulmonary consolidation with ill-defined borders. Occassionally the shadows may be more localized and discrete, producing a rather nodular appearance. The distribution of the shadows is mostly bilateral and symmetric and to some extent influenced by gravity; occasionally, however, only one lobe may be affected. Frequently the edema fluid tends to accumulate at the lung bases, or along the lobar borders, close to the interlobar fissures; this type of distribution will be seen in patients who have been ambulant, or sitting up in bed during an attack of acute pulmonary edema.

Distribution of edema is not wholly regulated by hydrostatic mechanisms, as shown by Doniach,[15] Hodson[35] and Jackson,[36] who described the radiologic appearance of the "bat's wing shadow" associated with central pulmonary edema. Day, Sisson and Vogt[14] were the first to observe central pulmonary edema. The radiologic features were fully described by Nessa and Riegler,[45] who also coined the term "butterfly shadow."

On posteroanterior chest films central edema or the "bat's wing shadow" appear as symmetric, confluent opacities extending from both hilae into the central core of the lung, leaving the periphery or cortex perfectly translucent (fig. 6). A clear border may sometimes be seen along the lobar edges, bordering the fissures. On the lateral radiograph the central shadows are also very clearly defined and well separated from the translucent peripheral zones surrounding them.

Shadows due to acute pulmonary edema appear very rapidly, often within hours after the onset of an attack, and they may equally rapidly disappear. The clearance rate often depends on the speed of recovery of left-sided failure and also on the consistency of the exudate. The more albuminous the exudate the quicker it will reabsorb. If the edema fluid is more fibrinous it may persist over long periods, even days or weeks. Incomplete resolution with subsequent carnification of intra-alveolar exudate was recorded by Doniach, Morrison and Steiner.[16] These authors observed persistent lung shadows in patients who were treated with ganglion-blocking agents for hypertension and who probably had many minor attacks of pulmonary edema. Similar findings were observed by Perry, O'Neal and Thomas.[49]

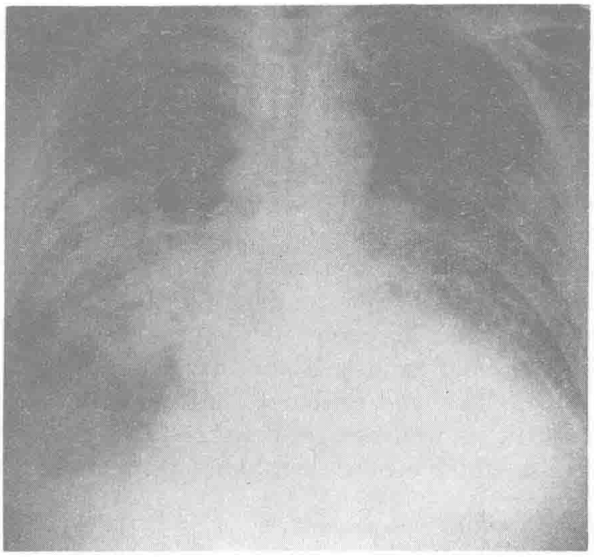


FIG. 6.—Acute left ventricular failure. Central pulmonary edema.

Differential diagnosis of acute pulmonary edema.—This may give rise to some difficulty in interpretation, since the radiologic appearances are so varied. Lobar pneumonic consolidation or bronchopneumonia are the two most important conditions to be differentiated. Correlation of the speed of appearance of the shadows and the appearance of the heart shadow can help in the differential diagnosis. Pulmonary shadows, due to lobar or bronchopneumonia, appear much later than those due to acute pulmonary edema. Frequently pulmonary edema is complicated by super-added pulmonary infection, which will not necessarily influence the radiologic appearance of the pulmonary shadows, but will influence the clearance rate, prolonging it, although there may be considerable clinical improvement in the degree of left-sided heart failure.

Uremic edema of the lungs.—This unusual type of edema occurs in left heart failure associated with marked renal impairment. The radiologic and pathologic features were reviewed by Doniach.[15] This author considered that pulmonary edema was partly due to the high venous and pulmonary capillary pressures, as well as to an alteration in the capillary permeability, probably due to the uremia. The radiologic features of uremic edema are indistinguishable from those seen in acute pulmonary edema, already described above.

In uremic edema there may be a marked discrepancy between the extent of the lesion seen on the films and the relative paucity of physical signs. The absence of physical signs is probably partly due to a rather fibrinous consistency of the edema fluid and to its central distribution.

The mechanism for the occasional central distribution of acute pulmonary edema, and particularly of uremic edema, is still obscure. It has been suggested by Herrnheiser and Hinson[34] that there exists an anatomic and functional difference between the lung cortex, or central core of the lung, and the lung periphery, or medulla. In postmortem angiographic studies, these

authors demonstrated a different anatomic arrangement of the smaller pulmonary arteries and arterioles and bronchi, in relationship to the larger arteries and bronchi in the central core of the lung and in the peripheral lung field. Some of the observations of Prichard, Daniel and Ardran[50] seem to confirm this anatomic difference. In acute animal experiments involving the injection of contrast material into the main pulmonary artery, these authors were able to demonstrate distinctive flow patterns. The bulk of contrast material appeared to be concentrated in the central core of the lung, and only small amounts reached the periphery. In addition the lung periphery became ischemic after the injection, the ischemia extending to the lobar borders along the interlobar fissures. It appears, therefore, that some anatomic factors such as distribution and caliber variations of the smaller pulmonary arteries and arterioles, as well as variations in the blood flow, may play a part in the mechanism of the central distribution of pulmonary edema.

Chronic Left Heart Failure

A most varied and frequently changing radiologic picture may be present in left heart failure. Short in 1956[54] has very accurately analyzed the various appearances which may be seen and also has correlated the x-ray shadows with the clinical state of the patient and the underlying lung pathology.[54]

Hilar clouding associated with hilar enlargement.—This is probably the most constant and frequent appearance seen in left-sided heart failure. The vessels at the root of the lung appear distended, their contours are not too clearly outlined, and they cannot always be clearly identified as vascular shadows. There is a generalized haziness extending from the hilum into the lung. It is suggested by Grainger[30] that hilar clouding and haziness is primarily due to interstitial edema in the hilar connective tissue (fig. 7A and B).

Pleural transudates.—Hydrothorax is a common accompaniment of left heart failure as opposed to right heart failure, in which it is rare. Effusions are usually bilateral and basal, but they can be unilateral, and it appears that the left side is more frequently affected than the right.[5] The amount of fluid can vary considerably from a very small collection in the costophrenic angles or posteriorly in the paravertebral sulcus to a large transudate which can extend well up into the axilla. If the effusion is massive it is often associated with mediastinal displacement away from the effusion. Transudates can also accumulate in the interlobar fissures, in which they may become encysted, or they may appear as a fine lamellar effusion along the chest wall. Small or encysted effusions are at times difficult to see on posteroanterior chest radiographs, but may be clearly visible on oblique or lateral projections or on fluoroscopy.

Interstitial pulmonary edema.—Interstitial edema due to a raised pulmonary venous pressure is common in left heart failure. Interlobular septal lines appear as in mitral heart disease, but they are usually much less widespread and less marked (fig. 7A and B). A slight haziness at the lung bases may be noticed due to further spread of the interstitial edema. It may extend subpleurally along the fissures, widening them appreciably. The signs of

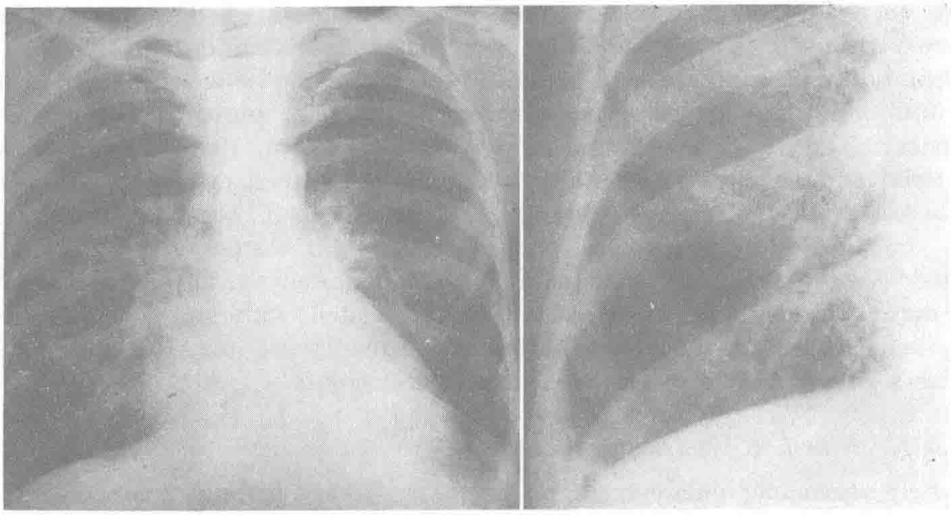


FIG. 7A *(at left).*—Chronic left ventricular failure. Dilated hilar vessels. Small transudate at the left base. Septal lines at the right base.

FIG. 7B *(at right).*—Localized view, right lower zone. (Same patient as figure 7A). Septal lines. Slight haziness due to interstitial edema.

interstitial basal edema are not always as well seen in left ventricular failure as in mitral disease, since pleural transudates obscure the lungs.

It has been pointed out by Short[54] and Grainger[30] that interstitial edema will often be visible radiologically long before clinical signs of left ventricular failure are recognizable. The appearance of interstitial edema should therefore be considered as a warning of incipient left heart failure.

Dilatation of the main pulmonary arteries and pulmonary veins.—As previously stated, fullness of the root shadows is frequently noted. Assessment of the pulmonary arterial and venous vascular pattern in the peripheral lung field is not very satisfactory in left ventricular failure. Occassional dilatation of the main lobar arteries and veins can be seen, but there is no distinct alteration in caliber of the smaller peripheral branches.

In severe cases of left heart failure an elevated pulmonary artery pressure has been recorded by Lewis, Houssay, Haynes and Dexter.[43] The pulmonary artery pressure levels are not as high, however, as in mitral heart disease, nor does pulmonary hypertension persist for long periods. These facts may explain the absence of visible radiologic vascular changes in the smaller peripheral arteries and veins. Muscular hypertrophy of the pulmonary arteries in hypertension and in aortic valve disease has been demonstrated histologically by Smith, Burchell and Edwards,[56] further confirming the presence of an elevated pulmonary artery pressure in some cases of left heart failure.

The radiologic signs of left ventricular failure are often quite transitory, particularly when the response to treatment is satisfactory. The pulmonary vascular changes and interstitial edema are largely a reflection of a labile pulmonary venous pressure. A distinct difference in the appearances of the vascular changes from those seen in mitral heart disease is not surpris-