RADIOLOGY OF THE LUNG IN SEVERE MITRAL STENOSIS

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The radiological changes in the lung in heart disease have attracted the attention of cardiologists for many years. Notable contributions have been made by Laubry *et al.* (1929), Roesler (1943), Donzelot and Heim de Balsac (1947), Parkinson (1949), and Zdansky (1953). In mitral stenosis especially, the radiological appearance of the pulmonary vasculature has invited keen attention, but not till the advent of surgery in the treatment of this condition has it been possible to correlate the radiological findings with the severity of the mitral stenosis.

Material and Method

Thirty-three cases of mitral stenosis have been selected in whom the mitral orifice was known to measure one cm. or less in length; so that in each the stenosis was severe. The assessment was made at operation in 30 cases and twice it was later verified at necropsy; in the remaining 3 the valve was examined only at necropsy. Aortic incompetence was diagnosed in 5 cases and mitral incompetence was detected by the surgeon in 13, but never did the electrocardiogram or cardioscopy suggest left ventricular enlargement, so that associated valvular lesions were regarded as slight. Electrocardiographic evidence of right ventricular enlargement was definite in 21, equivocal in 5, and absent in 7 cases. Twelve patients had suffered from right-sided heart failure at some period before operation. Fifteen gave a history of recurrent bronchitis, and two of these had severe emphysema.

Each patient before operation had radiographs that satisfied the criteria of the Medical Research Council X-ray Subcommittee. In 28 of the 33 cases the radiographic examination was made within four weeks of operation or of necropsy.

The films were exposed by a standard technique. The postero-anterior film was taken at a distance of six feet, respiration being halted at the end of a normal, or in some cases a deep, inspiration. The factors employed in the majority of cases were: 300 mA, approximately 65 kV, andone-tenth sec. exposure. Lateral films were taken in all cases, but they contributed little information of value.

Findings

The lung architecture was considered to be abnormal in each case, and the changes that were observed may be described in relation to their anatomical situation under three headings—the hilum, the parenchyma, and the pleura.

The Hilum. In the normal lung, the right and left pulmonary arteries and their subdivisions stand out clearly against the background of a translucent lung (Fig. 1). The margins of the vessels are distinct and on the right side the lumen of the right main bronchus can be seen lying medial to the main descending branch of the pulmonary artery.

In severe mitral stenosis (Fig. 2) the left hilum is frequently concealed by enlargement of the main pulmonary artery and its left branch. The right hilum, on the other hand, is generally visible. It stands out further from the mid line than usual, and is enlarged and clouded. The margins of the main pulmonary artery and its primary branches are ill defined and the bronchial lumen is obscured. Sometimes the hilum merges imperceptibly into the surrounding lung. Great hilar clouding (" hilar congestion ") was observed in 14 of the 33, more moderate clouding in 16, and

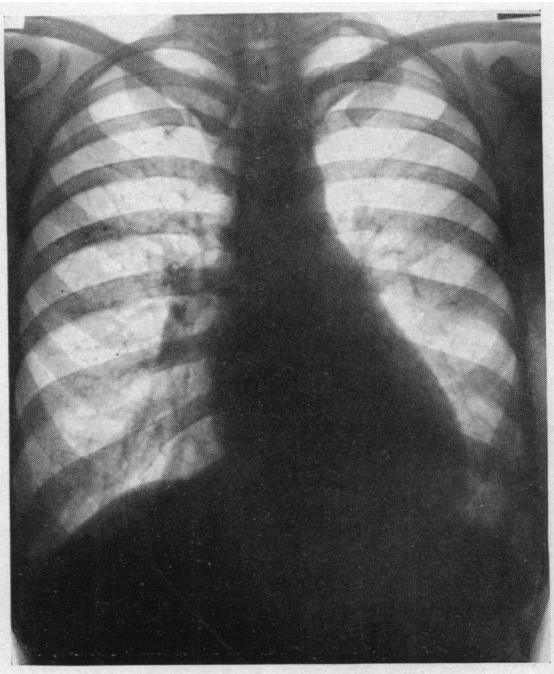

FIG. 1.—Slight mitral stenosis, showing normal right hilum. The mitral valve was 3 cm. long and slightly incompetent.

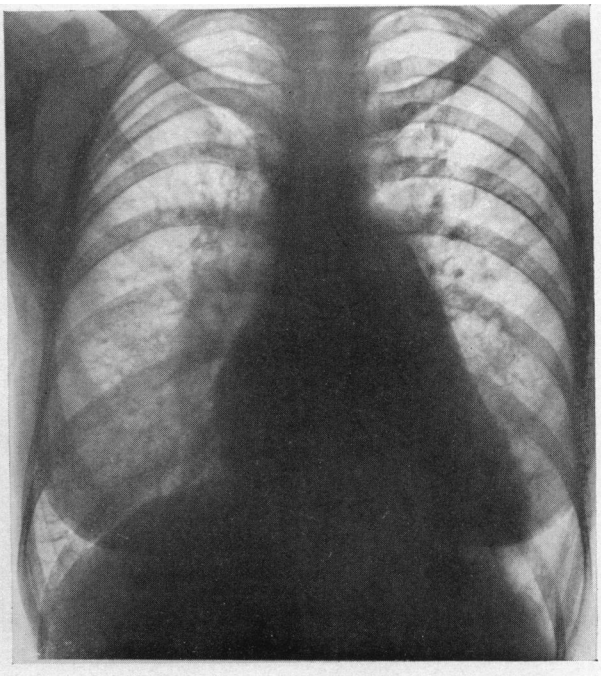

FIG. 2.—Severe mitral stenosis, showing great hilar clouding. The mitral valve was a little less than 1 cm. long.

none in 3 cases. In 23 of the 30 cases with hilar clouding there was also loss of translucency in the surrounding lung. Of the 7 cases where loss of translucency was slight or absent, two had severe emphysema.

The Parenchyma. In health, the lung markings are almost entirely vascular (Fig. 3). The branches of the pulmonary artery can usually be traced to within 1 cm. of the pleura. Often the right inferior pulmonary vein and its tributaries can also be followed and less commonly other parts of the venous system. The periphery of the lung is difficult to analyse with the exception of the costophrenic angles where it is frequently possible to trace vascular aborization to within a few millimetres of the pleura.

In severe mitral stenosis the normal translucency is usually lost (Fig. 4). The margins of the heart and the ribs lose their sharp definition and the outline of the pulmonary arterial branches is obscured. This clouding, greatest in the lower half of the lung, was present in 24 cases. Close scrutiny of the lung structure shows the clouding to be due most commonly to an abnormal profusion of vessels 0·5 to 2 mm. in width. These conceal the larger arteries as fine twigs obscure the branches of a tree. This abnormal vascular pattern was seen, not only in the 24 cases in which loss of translucency was evident from a distance, but also in five cases where the translucency appeared normal or increased; two of these had severe emphysema.

Loss of translucency was frequently associated with the presence of numerous short linear shadows (Fig. 5 and 7). Attention was first drawn to these lesions by Kerley (1951) who noted their presence in silicosis, carcinomatosis lymphangeosa, and hypertension as well as in mitral stenosis. They are distinguished from other shadows of linear appearance by their size, number, distribution, and direction. These lines are 0·3 to 1·0 mm. broad and 5 to 15 mm. long. Their margins are sharp and well defined and they are usually of constant width, though they may be a little broader in the middle than at the ends. They are found most commonly in the costophrenic angles, but when numerous, they extend up the lateral surface of both lungs and may be scattered

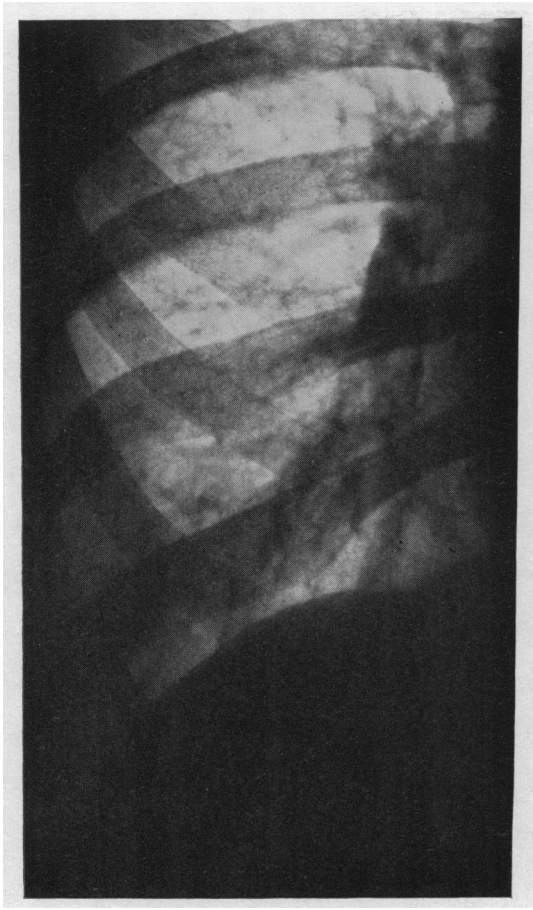

FIG. 3.—Slight mitral stenosis, showing normal vascular markings. The same patient as Fig. 1.

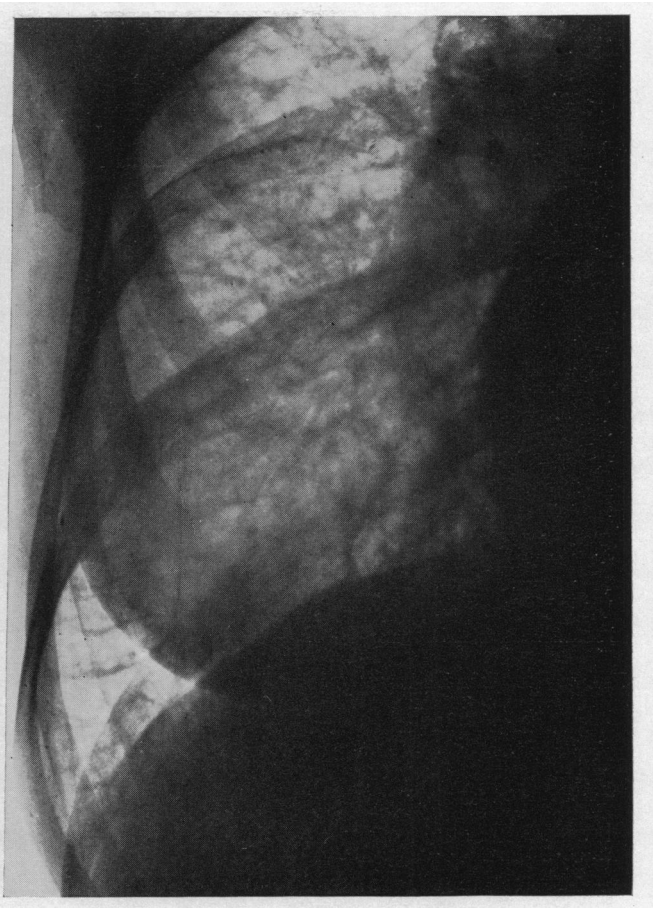

FIG. 4.—Severe mitral stenosis, showing loss of normal translucency and septal lines in the costophrenic angle. The same patient as Fig. 2.

profusely over the whole of the lower and middle zones. In the lateral radiograph the lines may be seen lying beneath the anterior chest wall within the middle lobe.

These lesions lie immediately beneath the pleura and at right angles to it. Thus on the costal surface of the lung they are horizontal, and on the diaphragmatic surface they are vertical. In the centre of the lung where the pleura is viewed end-on instead of tangentially they may present a hexagonal pattern. Since there is no doubt that these lesions correspond to the interlobular septa (see *Discussion*) they may appropriately be termed septal lines or septal shadows.

Septal lines were found in 25 cases. In none were they the sole abnormality, though in several instances where either hilar clouding or loss of translucency were equivocal they provided conclusive evidence of an abnormal lung pattern. When scanty, these lesions may easily be overlooked unless the costophrenic angles and adjacent areas are diligently scrutinized.

Narrowing of the distal half or two-thirds of the pulmonary arterial tree in cases of mitral stenosis with severe pulmonary hypertension has been demonstrated by angiography (Goodwin *et al.*, 1952). This observation has proved of limited application in the study of the plain radiograph for two reasons. In the first place, the arteries to the lower half of the lung, where the attenuation is most pronounced, are often obscured. Secondly, the size of the arteries shows wide variation in health (Lodge, 1946).

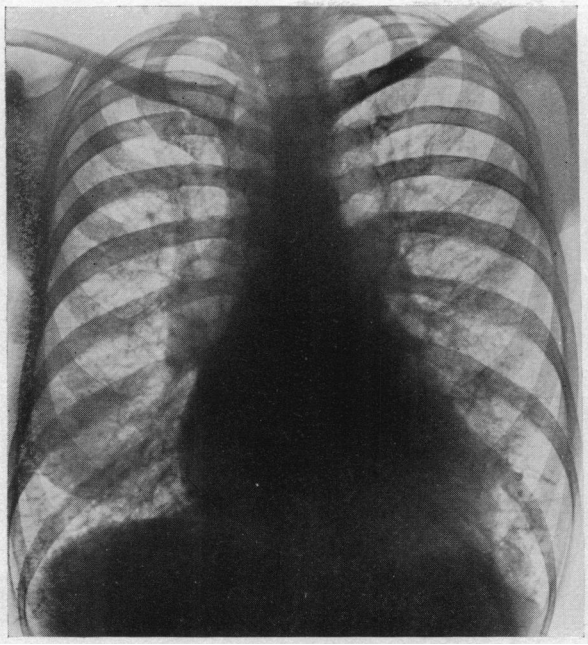

FIG. 5.—Severe mitral stenosis, showing septal lines extending up the lateral surface of both lungs and osseous nodules in the left lower zone. The mitral valve was 1 cm. long (see Fig. 7).

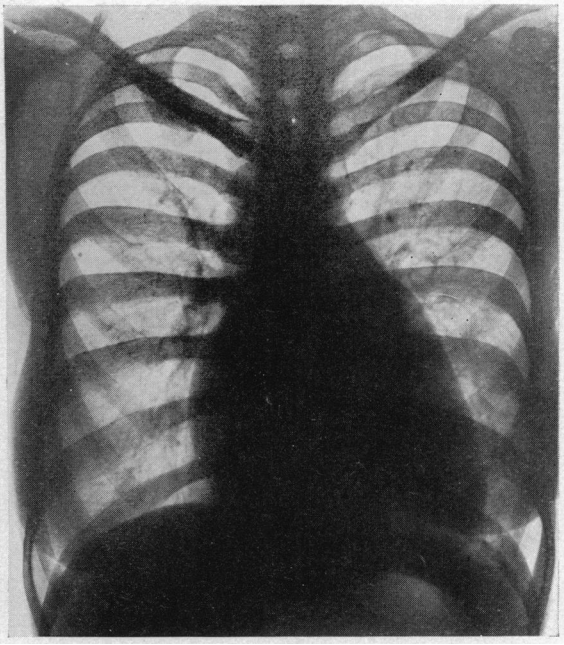

FIG. 6.—Severe mitral stenosis, showing normal translucency and absence of hilar clouding; the intrapulmonary arteries are greatly narrowed. The mitral valve was 1 cm. long (see Fig. 8).

In the present series there were nine patients in whom most of the arteries within the right lung could be seen clearly enough to permit an assessment of calibre. In two of these, all the vessels within the middle and lower lobes were narrowed to a striking degree. Once this feature constituted the sole abnormality of the lung pattern (Fig. 6 and 8). In 4 cases the calibre was regarded as borderline, and in the remaining three the arteries were normal.

The main pulmonary veins were seldom recognized either because they were obscured by loss of translucency within the lung or because they were hidden by the enlarged heart shadow.

Linear streaks radiating out from the hilum into the lower or middle lobe of the right lung, similar to those described by Kerley (1951) in silicosis, were seen twice. Other abnormal features observed in the lung parenchyma included pulmonary infarcts in three cases, a picture suggesting hæmosiderosis in one case, and disseminated osseous nodules in one case.

The Pleura. A large pleural effusion was observed once only. Repeated aspiration was required before operation, but thereafter no further paracentesis was necessary. One other case had a trace of fluid in the costophrenic angle, but it disappeared within a week.

Thickening of the interlobar fissures was noted in 12 cases. Although best appreciated in the lateral view, it was usually evident in the postero-anterior view also; most commonly as an abnormal prominence of the horizontal fissure; less frequently as a thickening of the inconstant pleural fold separating the retrocardiac (inferior accessory) lobe from the remainder of the lower lobe. In the two patients with severe emphysema, there was extensive old pleurisy over both lower lobes.

Severe Mitral Stenosis without Hilar Clouding. There were three patients in whom hilar clouding was absent or equivocal. In one, there was slight loss of translucency and faint septal lines. In another, similar lines were present without loss of translucency, and in the third both were absent. The vascular pattern was abnormal in all three: in one, all the branches of both main pulmonary arteries were considerably narrowed, and in the other two the arteries within the lower halves of both lungs were obscured by a profusion of small vessels.

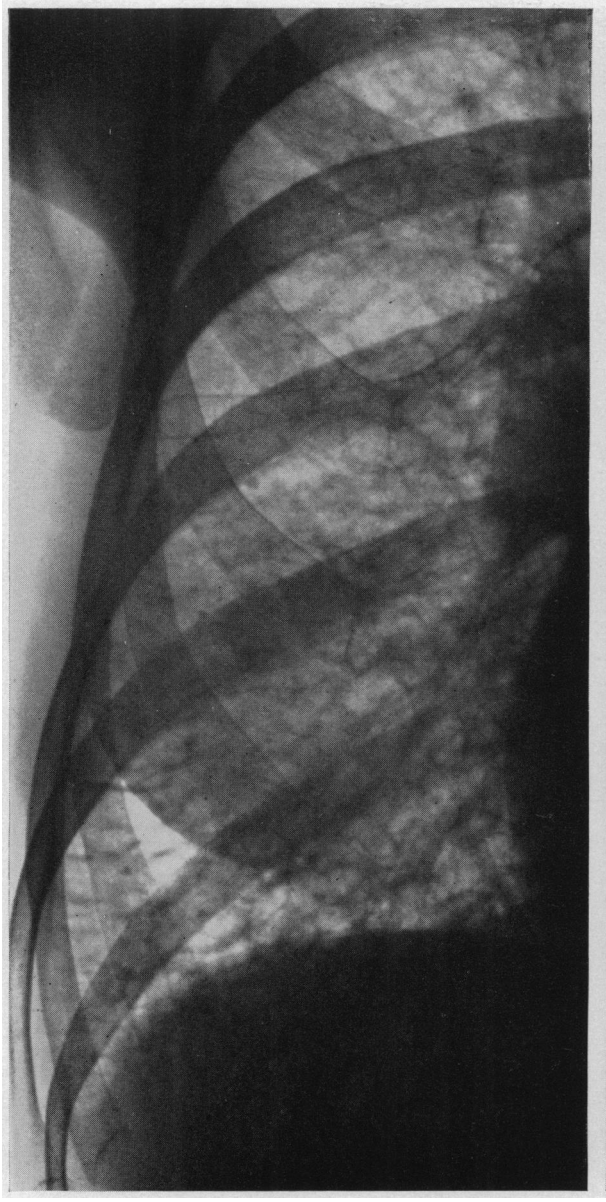

FIG. 7.—Contact print of part of right lung to show septal lines. The same patient as Fig. 5.

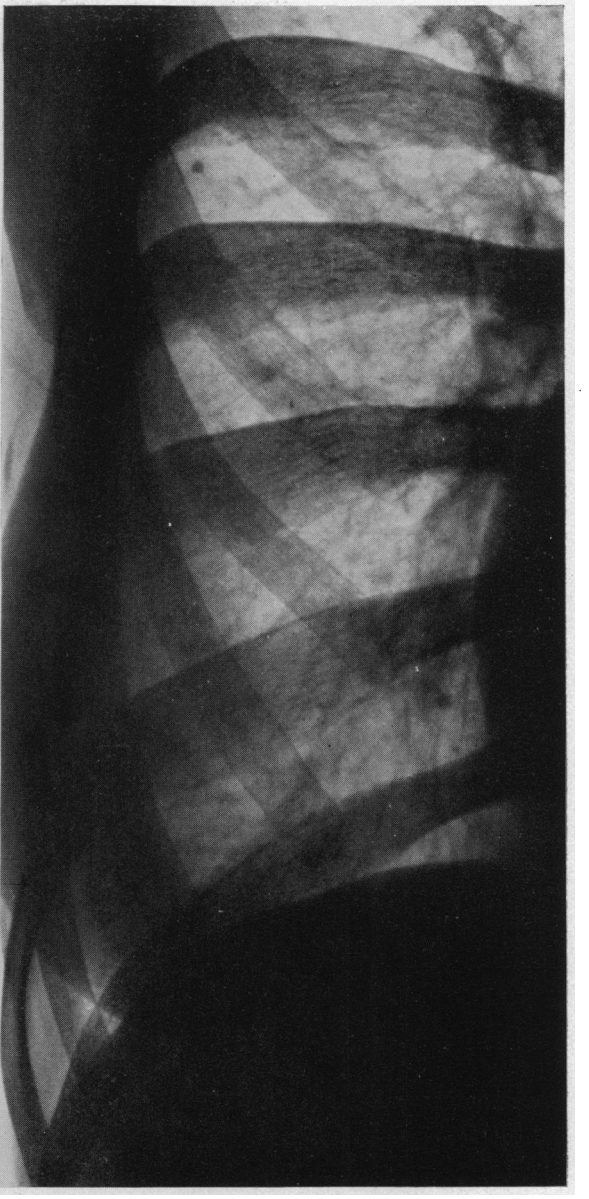

FIG. 8.—Contact print of part of right lung to show narrowed arteries. The same patient as Fig. 6.

Clinically, these patients were indistinguishable from those with pronounced radiological changes. One had severe pulmonary hypertension. Another suffered from recurrent right heart failure although the electrocardiogram showed no evidence of right ventricular hypertrophy. The third had never suffered from congestive failure; the right ventricular pressure at rest was scarcely raised and the cardiogram showed only slight right ventricular hypertrophy. In none was any evidence of tricuspid or pericardial disease found.

Serial Radiography. Two patients were under observation for over three years before surgical treatment and in both the lung architecture was abnormal from the start. During the period of

observation, hilar clouding increased and fresh septal lines appeared. In one there was a sudden temporary deterioration due to acute pulmonary œdema. Two years later, during a period of rest in bed and digitalis medication before operation, the septal lines decreased though the hilar clouding and loss of translucency persisted.

No definite change in lung pattern was detected in patients followed for periods of less than one year. Rest in bed with or without salt depletion produced slight or moderate improvement in the radiograph, irrespective of the presence of right heart failure. In one case a small effusion cleared. In several instances pleural thickening disappeared and septal shadows became less conspicuous though in none was there a reversion to anything approaching a normal pattern. Thus, the pulmonary changes in severe mitral stenosis are persistent but not fixed. Medical treatment may lead to temporary improvement, but the general trend is one of slow deterioration.

Discussion

An abnormal lung pattern was found in each of the 33 cases of severe mitral stenosis, but in three, hilar clouding, loss of translucency and septal lines were either absent or equivocal and the only definite abnormality was in the vascular pattern.

An adequate radiograph is essential if slight changes are to be detected. Loss of definition in the hilum and the arterial tree may be produced by pulsation, so the exposure should be as short as possible. With modern apparatus 0·05 sec. is frequently possible and it should never be necessary to exceed 0·1 sec. No conclusion should be drawn from any radiograph in which the outline of the upper ribs, the diaphragm, and one or other cardiac border is not sharp.

The salient features of the lung radiograph in mitral stenosis have long been recognized, but Kerley's description of the linear shadows has provided an additional sign, the importance of which may be measured by the fact that it was noted in no less than three-quarters of the cases. Once the characteristic appearance of the septal line has become familiar—its thread-like form, horizontal direction, and predeliction for the costophrenic angles—it is easily recognized. Scanty lesions in obese patients may be missed if the film is not taken in full inspiration or is under-exposed. Where the costophrenic angles are concealed by the breasts, it may be helpful to take a film with the breasts raised, or alternatively to use a grid or take an antero-posterior radiograph.

Small vessels are sometimes well seen running laterally in the costophrenic angles, and it is important not to mistake these for septal lines. Differentiation is usually possible if attention is paid to the following points (Fig. 9).

(1) The margins of vessels are blurred while septal lines are sharp and well defined.

(2) Small vessels can be seen joining a larger vessel proximally. Septal lines seldom join a larger vessel and if they do so, the difference in character between the well-defined line and the " softer " vessel is evident.

(3) Vessels taper toward the periphery whereas the linear shadows are of constant width or else widen slightly in the middle.

(4) Vessels are part of a ramifying system, junctions being indicated by dots formed by branches viewed end-on. Septal lines do not branch and therefore dots are absent.

Septal lines correspond both in size and distribution with the interlobular septa, a fact that may readily be appreciated by an examination of lung slices prepared by the technique of Gough and Wentworth (1949). These septa consist of ingrowths from the subserous areolar tissue that invests the lung. They are traversed by tributaries of the pulmonary venous system and also by the lymphatic channels that link the deep lymphatic system with the subpleural network. The prominence of the interlobular septa in mitral stenosis must therefore be due to enlargement of one or more of these structures. The fact that in some cases septal shadows persist unchanged after relief of the stenosis is difficult to reconcile with the view that they are due to lymphatic or venous engorgement. An acceptable explanation must take into account both the fluctuation in size that is observed in some cases and the persistence following surgical treatment in others. We

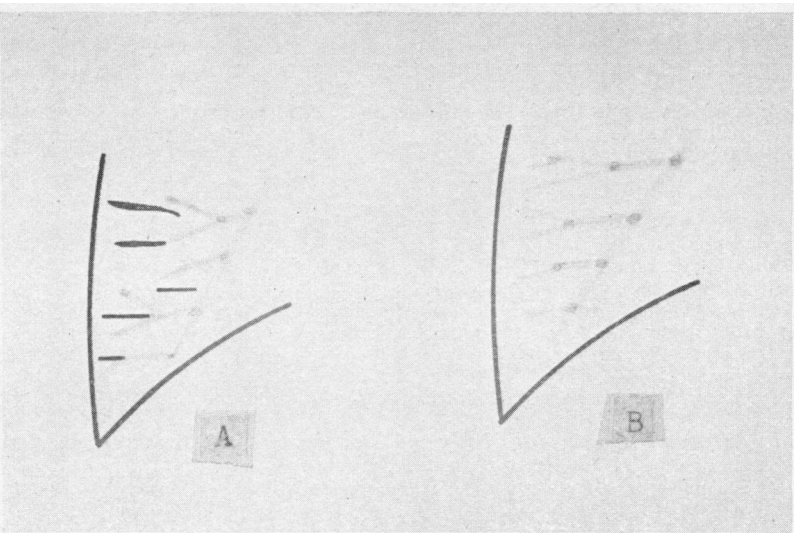

FIG. 9.—Septal lines contrasted with blood vessels in the costophrenic angle (diagrammatic). (A) Septal lines. (B) Blood vessels.

think that the enlargement of the septa is due to swelling of the areolar tissue, at first from œdema and later from fibrosis.

It is perhaps necessary to emphasize that although the radiological pattern as a whole may prove to be peculiar to mitral stenosis, no single feature is diagnostic of it. A similar appearance of the hilum is seen in left ventricular failure and in emphysema and bronchitis. Prominent septal lines are found most frequently in mitral stenosis, but they are also seen in some types of pneumoconiosis and in carcinomatosis lymphangeosa. Slender lines are not uncommon in hypertensive heart failure. Loss of translucency is seen in heart failure from a variety of causes, and attenuation of the peripheral pulmonary arteries has been noted in primary pulmonary hypertension. It is not claimed that the presence of these radiographic abnormalities indicates mitral stenosis, but rather that their absence is strong evidence against severe mitral stenosis.

There are many gaps in our understanding of the pulmonary changes in mitral stenosis. Perhaps the most obvious is our inability to account for the wide variation in intensity of these changes in patients with an identical degree of mitral stenosis. Another aspect on which our information is inadequate concerns the effect of these changes on respiratory function. Again, what is the morbid anatomy underlying hilar clouding, arterial narrowing, and the profusion of small vessels? Which lesions are reversible and which are irreversible? The answers to these questions may have an important bearing on the selection of patients with mitral stenosis for surgical treatment.

SUMMARY AND CONCLUSION

The radiographic appearances of the lungs were studied in 33 patients with rheumatic mitral stenosis in whom, at operation (or necropsy), the maximum diameter of the mitral valve was 1 cm. or less. Twelve had suffered from right heart failure.

The lung architecture was abnormal in each patient, though the degree of abnormality varied widely, being slight in some and considerable in others. Hilar clouding was present in 30 and loss of translucency in 24. Pleural involvement was evident in 15, two of whom had a right-sided effusion. The vascular pattern was abnormal in 30 patients. Most commonly the intrapulmonary arteries were obscured by a profusion of small vessels, but occasionally these arteries were well-defined and severely attenuated. Septal lines were found in 25 cases and though never the sole

abnormality they were sometimes the most impressive. Serial radiography showed the changes to be persistent and slowly progressive.

A normal lung architecture is inconsistent with a diagnosis of severe mitral stenosis.

The patients were under the care of Dr. William Evans or Dr. Wallace Brigden, to both of whom, as well as to Dr. Peter Kerley, I am indebted for helpful criticism. Mitral valvotomy was performed either by Mr. Vernon Thompson or Mr. Geoffrey Flavell.

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