Tomographic Evaluation of Hemodynamic Changes in Mitral Stenosis

With a Statistical Note on Normal Tomographic Findings

By M. J. Dulfano, M.D., and H. Adler, M.D.

The last few years have witnessed a renewed interest in the roentgenologic interpretation of disturbances in pulmonary circulation. Because it is inexpensive and readily available, the plain chest x-ray has been mainly used.\(^1\)-\(^8\) It gives a good view of the major features but, by necessity, represents the superimposition of many structures and thereby limits the observations of details, especially in the hilar and lower parts of the lungs.\(^1\)-\(^3\)

Angiocardiography is undoubtedly the most adequate method for this study but it is expensive, time-consuming, and not without danger for the patient.\(^14\), \(^15\) Tomography utilizes the natural contrast between the blood-filled pulmonary blood vessels and the air-containing lung to good advantage.\(^16\), \(^17\) It constitutes an effective supplement to the use of plain chest x-ray in the assessment of mitral valvular disease.

The intention of our study is to demonstrate that routine tomography can give a very detailed estimation of the size, distribution, and contour of the pulmonary vessels; pathologic changes in the parenchyma, interstitial tissues, and pleura; and the configuration and size of some of the cardiac chambers. All these data can be correlated with the hemodynamic events in the lesser circulation.

Material and Methods

This study is based on 31 patients suffering from mitral valvular disease who were being evaluated for mitral commissurotomy. All except two were clinically diagnosed as having predominant or pure mitral stenosis. All of them underwent cardiac catheterization according to established techniques.\(^18\) The plain chest film and tomography were obtained within 3 weeks after catheterization. One hundred complete chest tomograms of individuals without evidence of heart disease served as controls.

Tomography was performed on a horizontal table with a focus-film distance of 140 cm., with use of a rotation angle of 45° and an exposure time of 0.8 second. The perspective enlargement factor ranges from 14.7 per cent in a layer of 8 cm. to 17.6 per cent in a layer of 11 cm.

Anteroposterior tomograms were routinely made at three levels, 1.5 cm. apart; the middle one was in the hilar region, this ordinarily means the layer 10 to 11 cm. in adults and 7 to 9 cm. in adolescents; one, 1.5 cm. dorsally, and one, 1.5 cm. ventrally from the hilar cut. Lateral tomograms usually required only two layers on each side; in normal adults the layers were 9.5 and 11 cm., and in adolescents 8 and 9.5 cm. from the lateral chest wall in the lateral decubitus position. When necessary, anterior oblique positions were added. The layers, the kilovoltage, and the milliamperes were adjusted to the body structure of the patients.

Normal Anteroposterior Tomograms

In the hilar layer and around it a clear picture is usually given of the descending branch of the right pulmonary artery and the first branches leading to the three lobes. The width of the right descending branch, hereafter called hilar right pulmonary artery (numbered 1 in fig. 1), was easily measured at right angles to the bronchus and to the outer margin of the vessel. Its diameter ranged from 10.0 to 16 mm. The mean value ranged in the various age groups from 11.0 to 14.0 mm. These figures are in accordance with the results obtained by Schwedel and associates in plain chest films.\(^9\)

The left main pulmonary artery (numbered 5 in fig. 1) was quite prominent in this position without overlapping other formations. Its width measured at a right angle to the left main bronchus

\(^*\) There are obvious limitations of measurement of vessels. These include defining the same point because of variations in branching, in size of patients, and in quality of film. Also the normal variations are wide. Nevertheless, the following findings in the normal provide a useful baseline for the assessment of the pathologic changes.

From the Cardiopulmonary Laboratory, Bellinson Hospital, Petah Tikya, and the Radiology Department, Meier Hospital, Kfar Saba. The Sick Fund of the General Federation of Labour, Israel.
ranged from 17 to 31 mm. Its mean values in the various age groups ranged from 20.2 to 25.8 mm. Medium-sized, segmental arteries (numbered 2 in figs. 1 and 3) both in the anteroposterior and lateral tomograms varied from 2.5 to 6.5 mm. in width. The mean values in the various age groups ranged from 3.5 to 4.7 mm. There were no significant differences in the size of the most peripheral vessels; they were usually 1 mm. wide.

The pulmonary veins were well visualized in the hilar layers, particularly in the lower part of the right hilus, where they cross the arteries (numbered 3 in fig. 1). The width of the segmental lower lobe veins ranged from 3.5 to 8 mm. The mean values in the various age groups ranged from 4.3 to 5.4 mm. Their point of entry into the left atrium was usually clearly outlined and marked its outer limit (fig. 4). The upper lobe veins run from the apex downward, parallel and externally to the arteries and are better seen on the hilar tomographic layers (numbered 4 in fig. 1). The width of the upper lobe veins ranged from 2.5 to 6 mm. The mean values in the various age groups ranged from 3.7 to 4.4 mm.

Normal Lateral Tomograms
In the right lateral view (fig. 2), the trunk of the right main pulmonary artery is seen end on. Its posterior border stands out, contrasting with the translucency of the stem bronchus, its inferior border contrasting with the lobar bronchus of the middle lobe. The anterior border is usually convex and clearly visible. The mean diameter of the round-like shadow was derived from the horizontal and vertical diameters. It ranged between 20 and 33 mm., and the mean value in the various age groups ranged between 21 and 28.8 mm.

In the left lateral view (fig. 3), the left main pulmonary artery is seen as a well circumscribed band that embraces the round translucency of the left main bronchus, runs dorsally to it and downward to the lower lobe. Its width ranged from 14 to 25 mm. The mean values in the various age groups ranged from 18 to 20.4 mm.

The segmental distribution of the pulmonary vessels is ordinarily recognized in both right and left lateral tomograms, thus giving a better insight into pathologic conditions of the vessels than routine anteroposterior tomography.

Lateral tomography is a valuable method for outlining size and distribution of the pulmonary veins. The inferior veins lie ventral to the arteries running parallel to them (figs. 3 and 7). The common basal venous trunk joins in the lower posterior part of the hilus with \( V_b \), the vein of the apical lower lobe segment which runs in oblique direction downward and ventrally (fig. 2). At entry into the left atrium, the veins are seen as a slight bulblike dilatation (fig. 6) which becomes
progressively prominent with the advancing mitral stenosis (figs. 11 and 14). Topographically they seem to belong to the upper lobe venous system.

Enlargement of the anterior border of the heart shadow on the anterior thoracic wall is a valuable sign in cases of right ventricular hypertrophy. This sign is best visualized in the left lateral tomogram. Left ventricular hypertrophy is generally not so positively identified.

In the left lateral tomogram, left atrial enlargement can be indirectly disclosed by the narrowing and displacement of the left main bronchus, which is always clearly seen in this view (figs. 3, 11, and 13). The right anterior oblique tomogram is of great help in outlining the posterior margin of the enlarged left atrium by observation of the region of entry of the pulmonary veins.

Enlargement of the inferior vena cava may be delineated in the right lateral tomogram.

Intracardiac calcifications may be distinctly discerned in the left lateral tomograms.

**Findings in Patients with Mitral Valve Disease**

The analysis of our pathologic material centered around the following main details:

1. The size of the arteries at the level of the trunk of the right main pulmonary artery, the hilar right pulmonary artery, and the segmental branches. The degree of "tapering off" between the hilar and segmental branches was noted (table 1).1, 3, 10

2. The size of the common upper lobe veins, before their entry into the left atrium (table 1), and their segmental branches.

3. Parenchymal and vascular changes were integrated in three groups of signs: (a). The lung background (diminution of normal lung translucency. (b). The presence of A lines (seen as thin, curvilinear lines radiating to the hilar region,19-21 B lines (horizontal lines running perpendicular to the costal pleura along the peripheral lobular septa. They are better seen in the plain film),20, 22, 23 thickened fissure,20 interlobar or free transudates.12 (c). Changes in the blood vessel contour (tortuosity),14 Each of these groups of signs was arbitrarily awarded a value of one unit. If all the signs were present (3a, b, c) and any was marked, allowance was made for one extra unit so that a limit of four units was set (table 1). It was our opinion that these signs (3a, b, c) represented evidence of impaired venous drainage and are so labeled in this paper (table 1).

4. The pattern of blood vessel filling throughout the lung, based essentially on Simon's work.13 Four stages were adopted: (a). Normal distribution, i.e., equal throughout the lung. (b). Less filling in the lower lobes of the lung and increased filling in the whole of the upper lobes (including the apex of the lower lobe, which usually behaves functionally like the upper lobe. (c). Diminished filling in the lower lobes and increased filling only

*Figure 3*

**Mitral stenosis, early stage:** 1, normal left main pulmonary artery; 2, segmental artery; 3, segmental vein, normal blood vessel and filling throughout; 4, slight displacement of left main bronchus.

*Figure 4*

**Mitral stenosis, early stage:** normal width and vessel filling distribution throughout lung; 1, slight enlargement of left main pulmonary artery; 2, slight enlargement of left atrium.
Table 1
Individual Tomographic Correlation with the Hemodynamic Findings

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<th>Blood vessel pattern</th>
<th>Common ULV mm.</th>
<th>PCP mean mm. Hg</th>
<th>RPA (trunk)</th>
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Common ULV: Width of the upper lobe vein at the level of the left atrium. PCP: Mean pulmonary 'capillary' pressure. Arteries, RPA: Trunk of right pulmonary artery diameter. ‘Taper off’: Millimeter difference in width between hilar right pulmonary artery and segmental arteries.

*Pulmonary artery pressures and vascular resistances are not considered because of the 'myocardial factor.'

in some segments of the upper lobes. (d). Diminished filling throughout the lung without regional differences (table 1).

5. We took notice of several cardiac features: enlargement of the right ventricle and left atrium, calcifications, and shape of the heart in the left lateral tomogram.

Results

Venous Circulation

Drainage Impairment

Two units of drainage impairment marked the boundary between the milder and severer cases (table 1). Patients with less than two units belonged to clinical group II to early III (New York Heart Association Classification); their mean pulmonary 'capillary' pressure was less than 20 mm., cardiac index above 3 L. per minute per M.² and total pulmonary resistance below 400 dynes sec. cm⁻¹.⁵.¹⁸

Increased signs of drainage impairment correlated well with increased disruption of the normal pattern of blood vessel filling in

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Figure 5
Mitral stenosis, early stage: 1, normal hilar and, 2, segmental branches of pulmonary artery; 3, normal right inferior vein with; 4, segmental branches. Note that the left upper segmental lobe vein, 5, is normal and of less width than the inferior segmental lobe veins.

Figure 6
Mitral stenosis, early stage (right lateral tomodogram): 1, the trunk of the right pulmonary artery is minimally enlarged, normal blood vessel filling distribution; 2, slightly enlarged right pulmonary veins at the level of the left atrium.

the lung (table 1). The individual tomographic sign that correlated better with the degree of mitral impairment to flow (as expressed by the pulmonary "capillary" pressure) was the size of the common upper lobe vein as seen in the region of the left atrium in the right or left lateral tomograms. A common upper lobe vein less than 17 mm. in diameter meant pulmonary "capillary" pressure of less than 20 mm. Hg mean pressure (table 1).

Pattern of Blood Vessel Filling
Stage A was comprised of patients in clinical group II and early III whose pulmonary "capillary" pressure was less than 18 mm. Hg mean pressure, cardiac index more than 3 L. per minute per M.², and total pulmonary resistance of less than 300 dynes sec. cm⁻⁵. Stage D meant pulmonary "capillary" pressure usually above 25 mm., cardiac index less than 2.5 L. per minute per M.², and total pulmonary resistance more than 1000 dynes sec. cm⁻⁵. Stages B and C were intermediate (table 1). The results were in fair accordance with the analysis of signs of drainage impairment.

Arterial Circulation
The more dynamic behavior of the arterial circulation in mitral stenosis renders the roentgenologic correlation more difficult than on the venous side. The mean diameter of the trunk of the right pulmonary artery was measured in the right lateral tomograms. A diameter of 30 mm. was found in patients whose pulmonary artery systolic and mean pressure were in the region of 50 and 30 mm. Hg, respectively.

The correlation was better when we compared the millimeter difference in width of the hilar right pulmonary artery minus the segmental artery branches ("tapering off")
in the anteroposterior tomogram (table 1). The boundary between milder and more severe cases was at the width difference of 10 mm. When this difference was below 10 mm, the pulmonary artery systolic pressure was less than 50 mm. Hg, the mean pressure was less than 30 mm. Hg, and the pulmonary arteriolar resistance was less than 200 dynes sec. cm.⁻¹ (table 1).

**Tomography-Hemodynamic Correlation**

A clear picture of tomographic-hemodynamic correlation could not be obtained by the use of individual tomographic signs. The integration of several tomographic signs, however, allowed us to define the progression of the hemodynamic derangement and to classify them into 3 stages: early, intermediate, and advanced (table 2).

**Early Stage.** Minimal signs of impaired venous drainage (0 to 1) unit, normal pattern or minimal disturbance of the blood filling pattern (stages A-B), minimal stasis of common upper lobe vein (mean 13.5 mm.), slight engorgement of the trunk of the main right pulmonary artery (in right lateral tomogram—mean 29.6 mm.), and slight increase in the degree of "tapering off" (in the anteroposterior tomogram, the width difference between the hilar right pulmonary artery minus the segmental branches had a mean value of 7.7 mm.) (figs. 4-7).

These patients belonged to clinical group II and early III. Their mean pulmonary "capillary" pressure and pulmonary artery pressure were 13.4 and 18.8 mm. Hg, respectively. Their mean pulmonary arteriolar resistance and total pulmonary resistance was 101 and 318 dynes sec. cm.⁻¹, respectively. The cardiac index remained normal with a mean value of 3.5 L. per minute per M.².

**Intermediate Stage.** Impaired venous drainage of 2 to 3 units, abnormal pattern of blood filling (stages B-C), increased stasis of the common upper lobe vein (mean 21.8 mm.), and greater engorgement of the trunk of the right pulmonary artery (mean 36.9 mm.). The mean degree of arterial "tapering off" in the anteroposterior view was 14.6 mm. (figs. 8-11).

All the patients belonged to clinical group III. Their mean pulmonary "capillary" pressure and pulmonary artery pressure was increased to 24 and 40 mm. Hg, respectively. Their mean pulmonary arteriolar resistance and total pulmonary resistance were 193 and 600 dynes sec. cm.⁻¹, respectively. The cardiac index decreased to the lower limits of normal and had a mean value of 3.0 L. per minute per M.².

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**Table 2**

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See table 1 for key to abbreviations.
Advanced Stage. Maximal signs of impaired venous drainage (3 to 4 units) and disruption of the blood filling pattern (stages C-D). All the patients had B or A lines, marked tortuosity of the blood vessels, the filling was poor throughout the lung, interlobar and free transudates were prominent. The common upper lobe vein and trunk of right pulmonary artery were maximally dilated (mean 25 and 42.6 mm., respectively). At this stage, artery size may be misleading if the right ventricular muscle is functionally inefficient. In such cases, observation of the venous circulation will still show the true advanced stage of the disease (figs. 12-14).

In this stage all the patients belonged to clinical group IV. Their mean pulmonary “capillary” pressure and pulmonary artery pressures were 27.2 and 50 mm. Hg, respectively. Their mean pulmonary arteriolar resistance and total pulmonary resistance were extremely elevated to the level of 507 and 1350 dynes sec. cm.−5, respectively. The cardiac index was always below normal with a mean value of 2.0 L. per minute per M.2.

Heart Shape. As an incidental observation, we noticed in the left lateral tomogram that the heart shape exhibited a characteristic of cases of pure mitral stenosis. It resembled a pear standing on its narrower pole. This shape is apparently caused by the dilatation of the upper parts of the heart (the right ventricle and left atrium) without a corresponding dilatation of the lower half, particularly of the left ventricle (fig. 7). It may also be related to the usual clockwise rotation of the heart in mitral stenosis. The patients who also had an enlarged left ventricle due to added mitral insufficiency or aortic valve disease did not develop this shape.

Discussion

The pulmonary circulation is a most sensitive indicator of the hemodynamic derangement caused by mitral valvular disease. This can best be ascertained by studying the appearance of the pulmonary veins and the arteries separately. Throughout the course of the disease, the veins respond to changes in pressure by active constriction in the lower lobe and initial compensatory dilatation in the
upper lobes as described by Simon. Later this condition is maintained by organic changes. The arteries bear this increased pressure in two fashions: at the beginning, the pressure increase is equivalent to that in the vein in order to keep the same pressure gradient for ensuring forward flow; above the critical level of 25 mm. Hg the pulmonary artery pressure rises in a disproportionate amount to the venous pressure.24

Thus, venous pressure becomes a variable dependent on two factors: the degree of stenosis of the mitral valve and the amount of blood flow passing through the barrier of arteriolar resistance. On the other hand, pulmonary artery pressure and size fluctuate according to the pulmonary arteriolar resistance and the effectiveness of the right ventricular muscular pump.

These many variables, which can only be ascertained by cardiac catheterization, account for some of the failures of roentgenologic estimation of the hemodynamics in mitral stenosis, primarily because most of the attention has been given to the arterial tree.2, 5, 6, 8, 10, 21, 25 Therefore, we tried to correlate all the roentgenologic signs with the hemodynamic findings and, after giving them numerical value, tried to obtain a total picture. In this analysis, the greatest attention was given to the pulmonary veins and their drainage. This drainage in mitral stenosis is effected in several ways: (1) through the pulmonary veins; (2) by lymphatics; (3) by pulmonary-bronchial venous anastomosis.26, 27

The first way can be followed quite well tomographically by measuring the size of the veins in their confluence into the left atria, especially in the lateral tomograms (figs. 6, 11, and 14).

The second way was appraised by the presence of A and B lines and by the presence of thickened fissures, intralobar transudates, and free transudates in the costophrenic angles, which we believe are different expressions of the same hemodynamic phenomenon (figs. 8 and 12).20, 23 The third way cannot be ascertained roentgenologically.

Tomography is a very helpful method in differentiating true parenchymal nodules from
sections of blood vessels that have become tortuous; their pathway is ill defined and in a plain chest film, due to superimposition of many layers, they may be confused with nodules. We repeatedly found this feature of tortuous vessels in the more severe cases of mitral stenosis, confirming early angiographic observations (figs. 12 and 14). The survey of our plain films did not allow us to make this differentiation clearly in every case.

The physiologic analysis of the pulmonary drainage, when considered from all aspects, brought us to coin the phrase "impaired venous drainage," which is an expression not only of narrowing of the mitral valve, but also of its effects upon the pulmonary parenchyma. Impaired venous drainage proved to be a much better index than the currently used correlations of left atrial pressure with individual roentgenologic signs and represents a better expression of so-called degree of venous congestion.

Another approach to the hemodynamic effects of mitral disease is the study of the pattern of the filling of blood vessels throughout the lung. Simon gave a most lucid interpretation of this pattern with special reference to the veins, but because of the limitations of the chest x-ray, he was only able to grade the changes in the upper lobes though his observations embraced all the vessels. Our study confirms his results but we were able to go further, since tomography permitted us
to visualize the veins in all the lobes of the lung more clearly. The following facts were seen in regard to the medium-sized veins:

(a) In the very mild cases, the lower lobe veins, as in normal individuals, may still be larger than the veins in the upper lobe (figs. 1 and 5). (b) With advance of the mitral obstruction, there appears the "shunting away" of blood toward the upper lobe veins, which become engorged (figs. 8, 9, and 10). This phenomenon, at a relatively early stage, was considered by Simon to be due to early vasoconstriction of lower lobe arteries, resulting from the higher hydrostatic pressure in the lower lobes in the standing position. This finding in the lying position of the tomographic technic, confirmed that organic changes in the lower lobe arteries and veins become superimposed on the vasoconstriction fairly early. (c) In the advanced stages, this upper lobe engorgement does not recede "in toto." Usually, the anterior segments are the last to maintain it (fig. 11). (d) The veins of the apex of the lower lobe (Vo) behave functionally like the upper lobe veins. (e) Within lung segments, the arteries and veins appeared generally to be of the same caliber (figs. 10 and 11).

The measurement of the size of the arteries alone proved to be much less contributory to the assessment of the severity of mitral disease. Absolute values of pressure and resistance were correlated only within broad limits with the roentgenologic findings and our results were in general agreement with previous observations. Lack of correlation between pulmonary artery size and pulmonary artery pressure, especially in severe cases, may be due not only to intrinsic alterations in vessel walls, according to Lukas, but also to failure of the right ventricular myocardium to keep pace with increased resistances (cases 24 and 25, table 1). We
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never saw a patient with severe pulmonary artery hypertension who had a hilar pulmonary artery of normal size.

The assessment of the degree of arterial "tapering off" still remains the most useful correlation with arterial pressure. For this purpose, tomography is considerably superior to plain chest x-ray. It was brought out that the vascular resistance, as expressed by the sudden narrowing of the arteries is effected in the medium-size arteries and not in the smaller ones. This observation would be in favor of an active muscular mechanism, since the muscular coat is better developed in the bigger than in the smaller arteries. The study of the most peripheral branches proved to be of no value in mitral stenosis.

The progressive slowing of the pulmonary circulation with the advance of the disease can only be seen by angiography. Nonetheless, there is a parallel tomographic sign, especially in the right lateral position. In this view, an idea of this retardation of flow can be obtained by the progressive increase in size of both the right main pulmonary artery and the confluence of the upper lobe veins into the left atrium (figs. 11 and 14).

Summary

The hemodynamic findings in 31 patients with mitral valvular disease were correlated with the signs obtained by chest tomography. The normal and pathologic findings are described. The advantages of the tomographic technic for the study of the pulmonary vasculature are pointed out.

The progression of mitral valve narrowing was considered to be best related to the integrated picture of "impaired venous drainage." On this basis, three stages were defined.

The over-all response of the pulmonary circulation to the severity of the disease can be judged by the pattern of blood vessels filling throughout the lung.

It is concluded that for the evaluation of mitral valvular disease routine tomographic technic can indicate many of the essential hemodynamic findings with less hazard than either angiography or cardiac catheterization.

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