Correlation of Hemodynamic Data and Pulmonary Angiography in Mitral Stenosis

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SUMMARY

The abnormalities of pulmonary arterial blood supply in mitral stenosis, consisting primarily of constriction of lower lobe vessels with resultant upper lobe preferential flow, was assessed by means of pulmonary angiography in 30 patients with mitral stenosis uncomplicated by other cardiac diseases. The abnormalities in blood flow correlated best with both the pulmonary artery pressure and the pulmonary arteriolar resistance, and only reasonably well with the area of the mitral valve and the pulmonary capillary wedge pressure. It is suggested that the reversal of the normal lower lung field preferential blood flow in mitral stenosis is primarily a function of pulmonary artery hypertension due to increased pulmonary arteriolar resistance.

Similarly, the width of the right main pulmonary artery as measured on pulmonary angiograms correlated well with both the pulmonary artery pressure and pulmonary arteriolar resistance and less well with the area of the mitral valve. Pulmonary artery size in mitral stenosis is most likely a function of the severity of the pulmonary arteriolar resistance as reflected in the pulmonary artery pressure.

PREFERENTIAL BLOOD FLOW to the upper lung fields has been considered a feature of mitral stenosis.1 This abnormal reversal of pulmonary blood flow, as observed on chest roentgenograms, is not unique in mitral stenosis, for it may occur in cardiac disorders with left ventricular disease accompanied by elevation of left atrial pressure. This study is an attempt to assess the abnormalities in pulmonary blood flow in patients with pure mitral stenosis, by means of pulmonary angiography, and to correlate these with hemodynamic data obtained during cardiac catheterization.

Methods

From January 1963 to April 1966, 99 patients with clinical mitral stenosis, without prior cardiac surgery, underwent right and left heart catheterization at this hospital. Sixty-nine of this group who had mitral regurgitation, aortic valve disease, coronary disease, or elevation of left ventricular end-diastolic pressures above 11 mm Hg either at rest or during exercise were eliminated from the study. The findings in the remaining 30 patients, with mitral stenosis uncomplicated by other cardiac disease, comprise the material for this study.* The diagnosis was further confirmed in the 21 patients who subsequently had cardiac surgery. Twenty-eight subjects were males and two were females.† The average age was 44 years with a range of 30 to 65 years.

Pressures were recorded with Statham P-23D transducers on a Model DR-7 Electronics for Medicine photorecorder. Mitral valve diastolic gradients were obtained from simultaneous pulmonary capillary wedge and left ventricular pressure tracings. Left ventriculograms, performed

*Two of these patients were studied at the New England Deaconess Hospital, Boston, Massachusetts.
†The sex distribution is a consequence of the study being performed in a VA hospital.
with 60 ml of contrast substance, were obtained in the lateral projection using a rapid multiple film changer. Coronary arteriography was performed through a loop-end catheter positioned above the aortic valve. Pulmonary angiography was done with the patient in the supine position, through an angiographic catheter positioned just below the pulmonic valve in the outflow tract of

Figure 1
Sample angiograms from each of four groups with the numbers corresponding to the group numbers: Film 1 (group 1) is the angiogram of a 48-year-old normal subject with pulmonary artery mean pressure of 19 mm Hg, and a normal pattern with equal flow to upper and lower lung fields. Film 2 (group 2) is the angiogram of a 42-year-old patient with pulmonary artery mean pressure of 25 mm Hg and slight preferential flow to upper lung fields with minimal constriction of lower lung field peripheral vessels. Film 3 (group 3) is from a 45-year-old patient with pulmonary artery mean pressure of 52 mm Hg and more advanced degree of preferential upper lobe flow. Film 4 (group 4) is from a 40-year-old patient with pulmonary artery mean pressure of 68 mm Hg, extreme upper lobe preferential flow, and virtually absent flow in severely constricted lower lobe vessels.
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the right ventricle. Multiple films were made in the arterial filling phase. Exercise studies were obtained during a 3-minute period on a bicycle ergometer following a 3-minute warm-up period. Cardiac outputs were obtained by the Fick method.

The pulmonary angiograms were grouped as to the degree of abnormal pulmonary arterial blood flow on a scale of 1 to 4 (fig. 1). The angiographic standards were selected from this series with the exception of film 1 (group 1) which represents an angiogram performed in a normal 46-year-old man without cardiac disease as determined clinically and by cardiac catheterization. The pulmonary angiograms were interpreted and classified by three observers according to these four standards. Without prior knowledge of the patients' identity, each observer evaluated the entire series twice on separate days. In the event an observer disagreed with his first reading, the angiogram in question was reread on a third day. There was agreement among the three observers in all but two cases and in these two, the results were averaged.

The marked decrease in lower lobe vascularity in the angiograms of group 4 raises the question of pulmonary embolic disease. Pulmonary angiograms usually demonstrate asymmetric flow disparity in patients with embolic disease. Consequently, the symmetrical lower lobe vascular constriction noted in the angiograms of groups 3 and 4 is more likely the result of mitral stenosis uncomplicated by emboli although the latter always remain a possibility.

The width of the right main pulmonary artery was measured on the pulmonary angiogram at the narrowest point in that vessel (fig. 2). The main trunk and the left pulmonary artery could not be adequately measured because of the projection of the roentgenograms. All measurements were corrected on the basis of the width of the catheter in the right ventricular outflow tract in order to compensate for differences in magnification due to variation in chest configuration (the target-to-film distance being 46 inches in all cases). The magnification ranged from 10 to 20% with the average distortion being 14%.

Results

Evaluation of Pulmonary Angiograms

Four patients (group 1), had normal pulmonary blood flow patterns, nine patients (group 4), had extreme preferential flow to the upper lung fields associated with severe constriction of lower lung field vessels and seven patients (group 2) and 10 patients

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Figure 2

Arrow shows point of measurement of width of the right main pulmonary artery. Subject is 51-year-old man, classified in angiographic group 1, pulmonary artery mean pressure of 23 mm Hg.

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Figure 3

Pulmonary artery mean pressure plotted against the four angiographic groups.
(group 3) had intermediate vascular patterns. Groups 1 and 2 (11 patients) form a single hemodynamically homogeneous group. Similarly, groups 3 and 4 (19 patients) form a second separate hemodynamic group.

**Pulmonary Artery Pressure**

Groups 1 and 2 had pulmonary artery mean pressures of 36 mm Hg or less (range 16 to 36 mm Hg) and pulmonary systolic pressures of 55 mm Hg or less (range 22 to 55 mm Hg) without exception (fig. 3). All patients in groups 3 and 4 had pressures above these levels with a range of 38 to 80 mm Hg in mean pressure and 60 to 130 mm Hg in systolic pressure. The difference between groups 1 and 2 as compared to groups 3 and 4 is highly significant \((P < 0.001)\).

**Pulmonary Arteriolar Resistance**

Ten patients in groups 1 and 2 had a calculated resistance of less than 250 dynes sec cm\(^{-5}\) which is the upper limit of normal in our laboratory (fig. 4). The exception was one patient in group 2 with a resistance of 360. Conversely, all patients in groups 3 and 4 had resistances of more than 250 dynes sec cm\(^{-5}\) with the exception of one patient in group 3 with a resistance of 170. Fourteen of the 19 patients in groups 3 and 4 had striking elevations in the arteriolar resistance with values ranging from 500 to 1,960 dynes sec cm\(^{-5}\). The difference between groups 1 and 2 as compared to groups 3 and 4 is highly significant \((P < 0.001)\).

**Area of the Mitral Valve**

All patients in groups 1 and 2 had calculated mitral valve areas greater than 0.8 cm\(^2\) (0.9 to 2.0 cm\(^2\)) (fig. 5). Sixteen pa-
tients in groups 3 and 4 had smaller orifices (0.3 to 0.8 cm²). However, three patients in group 3 had valve areas of 1.0, 1.1, and 1.3 cm² respectively. The difference between the two major groups is moderately significant ($P < 0.01$).

Pulmonary Capillary Wedge Pressure

All 19 patients in groups 3 and 4 had mean wedge pressures above 19 mm Hg with a range of 20 to 37 mm Hg (fig. 6). Six patients in groups 1 and 2 had pressures of less than 19 mm Hg while five patients had higher values. The differences between the two major groups is significant only at the $P < 0.05$ level. It is apparent that although the patients with the most abnormal pulmonary flow patterns (group 4) had the higher wedge pressures, there was considerable overlap in the wedge pressures among patients in groups 2 and 3.

Cardiac Index

The cardiac index was less than 2.3 L/min/m² in 18 patients in groups 3 and 4; the exception was one subject in group 3 with an index of 3.1 (fig. 7). Six patients in groups 1 and 2 had indices above 2.3 L/min/m² and five had indices less than this value. Patients with the lesser degree of angiographic abnormality (groups 1 and 2) tend to have higher indices, but the difference is not striking ($P < 0.05$).

Evaluation of Pulmonary Artery Width

The width of the right main pulmonary artery was plotted against pulmonary artery mean pressure (fig. 8). The higher the pressure, the more dilated was the vessel although moderate scattering was present ($r = 0.7; P < 0.001$).
The width of the right main pulmonary artery, plotted against the pulmonary arteriolar resistance (fig. 9), also demonstrated a highly positive correlation ($r = 0.68; P < 0.001$).

The width of the right main pulmonary artery, plotted against mitral valve area (fig. 10), indicated a lesser correlation ($r = 0.45; P < 0.05$).

**Evaluation of Age Distribution**

The age distribution of patients with the more normal flow patterns (groups 1 and 2) and the more abnormal patterns (groups 3 and 4) did not differ significantly ($P > 0.20$). Similarly, there was no significant correlation between patient age and width of the right main pulmonary artery ($P > 0.20$).

**Discussion**

The pulmonary arterial blood flow in upright normal human subjects is known to be less in the upper lung fields than in the lower.$^4,5$ Studies utilizing inspired radioactive $^{15}O$ showed the perfusion ratio of the uppermost parts of the lungs to be approximately one fifth that of the lowermost areas in upright normal subjects, and that these differences are likely to be a consequence of gravitational effects.$^6$ Two other studies quantitated similar but somewhat less striking disparity in normal upright subjects with upper lobe flow being one half to one third of the lower lobe flow.$^5,7$ However, in the absence of gravitational effects, as in normal supine subjects, the flow to upper and lower lung fields is equal.$^6$ This is also seen in the pulmonary angiogram performed in the supine position in our patient without heart disease (fig. 1).

The normal pulmonary blood flow distribution is frequently altered as a consequence of mitral stenosis. Dollery and Hugh-Jones,$^8$ using inhaled radioactive oxygen and carbon dioxide, noted reversal of the preferential flow pattern recording as much as a fivefold higher flow through the upper lung zones in patients with mitral stenosis. Similar results have been obtained in other studies.$^9,10$

It has been suggested that the degree of abnormal preferential upper lobe flow correlates with the degree of pulmonary artery...
hypertension in patients with mitral stenosis. Several angiographic studies in which the pulmonary vessels were assessed in both the arterial and venous filling phases showed this correlation. However, the majority of the patients in one of these studies had associated aortic disease or mitral regurgitation. It became of interest, therefore, to correlate the pulmonary blood flow abnormalities with other hemodynamic measurements in addition to the pulmonary artery pressure in patients with pure mitral stenosis.

Although the pulmonary angiograms could be easily separated into four groups in our study (fig. 1), it is apparent that the patients with normal flow patterns (group 1) and with minimal abnormalities (group 2) had similar hemodynamic data. Patients with the more abnormal angiograms (groups 3 and 4) likewise formed a common group in reference to their hemodynamic data. The more abnormal pulmonary blood flow patterns occurred without exception in the patients with the higher pulmonary artery pressures and the correlation with pulmonary arteriolar resistance was nearly as high. The correlation of flow abnormality with mitral valve area was reasonable although less striking, whereas the correlation with wedge pressure and cardiac index is respectively less significant.

It is evident from this study that the abnormality in pulmonary blood flow relates best to both the pulmonary artery pressure and the pulmonary arteriolar resistance. The moderate but less striking correlation of flow patterns with mitral valve area as well as with wedge pressure is consistent with previously noted observations that secondary pulmonary arteriolar changes tend to develop in patients with smaller valve areas. That the preferential upper zone pulmonary blood flow correlated better with pulmonary arteriolar resistance than with pulmonary capillary wedge pressure suggests that the flow abnormality is due primarily to the varying degrees of obstruction at the arteriolar level.

The anatomic changes affecting pulmonary vasculature in mitral stenosis have been well recorded in autopsy material. Macroscopically, two types of narrowing of the smaller pulmonary arteries, focal and diffuse, occur in the lower zones. The diffuse narrowing is due to uniform thickening of the artery, while the focal narrowing is due to intimal plaques of atherosclerosis bulging into the lumen. Microscopically, two processes are seen in the arteries, hypertrophy and atherosclerosis, with both being distinctly more severe in the lower lung fields. The hypertrophy affects the media of the vessels while intimal thickening occurs as a result of localized atherosclerosis. The atherosclerotic thickening was never severe enough in their cases to occlude the vessels totally.

The etiology of the vascular changes in the lower lung fields in mitral stenosis is obscure. Increases in left atrial pressure up to approximately 25 mm Hg results in passive linear elevation in the pulmonary artery pressure but increases in left atrial pressure beyond this level are accompanied by much greater increases in pulmonary artery pressure due to the onset of reactive pulmonary arteriolar changes. It has been suggested that these reactive pulmonary arteriolar changes result from reflex constriction of the lower lobe muscular arteries possibly in response to increased pulmonary venous pressure or anoxia or from obliteration of the pulmonary capillary bed by alveolar fibrosis. West and associates, using isolated dog lung preparations as models, reproduced the mitral pulmonary flow pattern by raising pulmonary venous pressure; as a result cuffs of edema surrounded the lower lobe vessels. They proposed that this vascular compression resulted in preferential flow to the upper lobe. Jordan in attempting to unify these hypotheses suggested that the elevation of pulmonary venous pressure in mitral stenosis causes thickening of the lower lobe alveolar membranes initially by edema transudate and eventually by fibrosis. This process, in turn, results in uneven ventilation and alveolar hypoxia producing reflex constriction of the small pulmonary arteries. The net effect is severe pulmonary artery hypertension out of proportion to the elevation of left atrial
pressure. In view of this postulate, it is of interest that the patients in our study with more marked upper lobe pulmonary flow also had higher pulmonary artery pressures and higher pulmonary arteriolar resistances.

Lewis and co-workers12 graded the width of the pulmonary artery from the regular chest roentgenograms of 30 patients with mitral stenosis. Their data indicated minimal correlation with the pulmonary artery pressure and less correlation with pulmonary arteriolar resistance and mitral valve area. In our study, utilizing pulmonary angiograms where the right main pulmonary artery could be accurately measured, there was a reasonable correlation between the width of the artery and both the pulmonary artery pressure and the pulmonary arteriolar resistance, and less correlation with the area of the mitral valve. The implication is that the width of the pulmonary artery in mitral stenosis is a function primarily of the severity of the pulmonary arteriolar resistance as reflected in the pulmonary artery pressure and less a function of the area of the mitral valve.

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References


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