Determinants of the Left Atrial Pressure Pulse in Mitral Valve Disease


The difficulties of preoperative evaluation of patients with combinations of mitral stenosis and incompetence prompted analysis of the left atrial pressure pulse in a series of patients studied by left atrial puncture. Theoretic considerations indicate that similar pressure pulses may be produced by a variety of mitral valve lesions if the volume-pressure characteristics of the left atrium are appropriate. An explanation is offered for the differences found in the presence of mitral regurgitation.

Uncomplicated mitral stenosis and mitral incompetence are easily distinguished clinically and produce characteristic changes in the left atrial pressure pulse. Patients with a combination of stenosis and incompetence are more difficult to evaluate. The mitral diastolic pressure gradient and the cardiac output can be obtained by current techniques, but for complete assessment in the presence of mitral incompetence an estimate of regurgitant flow is necessary. This has been attempted by indicator-dilution methods, rapid biplane angiocardiography, and ultrasonic echo techniques. None of these approaches is free from practical or theoretic objection.

Several attempts have been made to evaluate mitral valve disease from the left atrial pressure pulse, or from the similar pressure pulse obtained when a catheter is wedged in a peripheral pulmonary artery. Methods based on the height of the v wave have been found less useful than indices derived from the y descent. Several workers have concluded that the left atrial pressure pulse is of little value in establishing the presence of mitral incompetence. To clarify the factors responsible for variations in the left atrial pressure pulse, a small series of patients with mitral valve disease has been reviewed.

From the Tufts Medical Services and Circulation Laboratory, Boston City Hospital and the Department of Medicine, Tufts University School of Medicine.

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Methods

Patients with mitral stenosis were studied when clinical evaluation was difficult because of associated mitral incompetence, aortic valve disease (5 cases), previous valvotomy (1 case), or doubt as to the severity of the lesion. Estimates of the size of the mitral valve orifice and the regurgitant flow were obtained at operation in all subjects. In 4 cases autopsy details were also available. Adequate data were available in 15 patients (table 1).

Left atrial puncture was made by the posterior route as described previously. Left ventricular pressures were recorded in all but 4 cases. Right heart catheterization was carried out to determine the cardiac output. Pressures were recorded with Statham P23Db strain-gage manometers, with use of Sanborn direct-writing equipment at a paper speed of 50 mm. per second. The base line for all pressure measurements was the middle of the chest at the level of the second costal cartilage with the patient recumbent. Measurements were made in records selected for adequate frequency response. In atrial fibrillation 10 cardiac cycles were measured and in sinus rhythm 5 were measured, and the mean value was taken. The Ry/v ratio was calculated with pressures adjusted to a base line at the level of the sternal angle as used by Owen and Wood.

Phasic gradients were obtained by replottting pressures at 0.05 second intervals on the same scale, the electrocardiogram being used as a control when simultaneous records were not available. The mitral valve area equivalent was calculated with use of the standard orifice formula. In patients with no left ventricular pressure record a mean diastolic pressure of 5 mm. Hg was assumed. Left atrial enlargement was estimated from standard radiographs and fluoroscopy, and was graded from 1 to 4.
TABLE 1.—Hemodynamic Data and Valve Areas in Fifteen Patients with Mitral Stenosis and Incompetence

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age</th>
<th>Rhythm</th>
<th>Cardiac index ((L./M^2/min.))</th>
<th>Mean mitral diastolic gradient ((mm. Hg))</th>
<th>Calculated mitral valve area ((cm.2))</th>
<th>Operation findings</th>
<th>Valve Regurgitation ((0 to 4))</th>
<th>Left atrial size ((1 to 4))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitral stenosis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P.C.</td>
<td>M</td>
<td>43</td>
<td>AF</td>
<td>2.2</td>
<td>19</td>
<td>0.8</td>
<td>1.0</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>T.E.</td>
<td>M</td>
<td>42</td>
<td>AF</td>
<td>2.4</td>
<td>21</td>
<td>0.8</td>
<td>0.8</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>D.H.</td>
<td>F</td>
<td>28</td>
<td>NS</td>
<td>2.8</td>
<td>5</td>
<td>1.0</td>
<td>0.8</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>L.H.</td>
<td>F</td>
<td>39</td>
<td>AF</td>
<td>2.5</td>
<td>13</td>
<td>1.0</td>
<td>0.8</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>S.H.</td>
<td>F</td>
<td>39</td>
<td>NS</td>
<td>3.1</td>
<td>21</td>
<td>1.2</td>
<td>1.2</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>W.H.</td>
<td>F</td>
<td>54</td>
<td>AF</td>
<td>1.6</td>
<td>5</td>
<td>1.0</td>
<td>0.8</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>E.O.</td>
<td>F</td>
<td>17</td>
<td>NS</td>
<td>3.2</td>
<td>27</td>
<td>0.8</td>
<td>1.0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>J.Q.</td>
<td>M</td>
<td>23</td>
<td>NS</td>
<td>3.0</td>
<td>16</td>
<td>0.8</td>
<td>1.0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>H.R.</td>
<td>F</td>
<td>51</td>
<td>AF</td>
<td>1.5</td>
<td>7</td>
<td>0.9</td>
<td>0.8</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>M.S.</td>
<td>F</td>
<td>61</td>
<td>AF</td>
<td>1.2</td>
<td>16</td>
<td>0.4</td>
<td>0.4</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Mean</td>
<td></td>
<td></td>
<td></td>
<td>40</td>
<td>2.4</td>
<td>14</td>
<td>0.8</td>
<td>0.9</td>
<td>0.7</td>
</tr>
</tbody>
</table>

| Mitral incompetence | | | | | | | | | |
| E.D. | F | 53 | AF | 2.7 | 7 | 1.7 | 2.0 | 3 | 3 |
| M.G. | F | 33 | AF | 2.6 | 4 | 1.8 | 2.5 | 3 | 4 |
| D.L. | M | 36 | NS | 1.6 | 13 | 0.6 | 1.2 | 3 | 3 |
| E.M. | F | 29 | NS | 3.0 | 15 | 1.3 | 3.0 | 3 | 3 |
| J.P. | F | 48 | AF | 1.4 | 12 | 0.6 | 3.0 | 3 | 3 |
| Mean | | | | 40 | 2.5 | 10 | 1.2 | 2.4 | 2.8 | 3.2 |

Hypothetical diagrams of phasic flow changes were constructed from a series of characteristic pressure curves with a cycle length of 0.6 second, a diastolic filling time of 0.33 second, and a mean mitral diastolic gradient of 16 mm. Hg. A stroke volume index of 30 ml. was assumed, giving a mitral valve area index of 0.7 cm.²

RESULTS

The 15 patients were divided into 2 groups. In 10 patients mitral incompetence was either absent or insignificant. The remaining 5 patients had considerable narrowing of the mitral valve, but gross regurgitant jets were felt by the surgeon. The incidence of atrial fibrillation was similar in each group, but the left atrium tended to be larger in patients with mitral incompetence. The observed and calculated mitral valve areas were similar in the stenotic group (table 1).

The \( y \) descent of the left atrial pressure pulse was analyzed by 3 methods. When the rate of the \( y \) descent was related to the height of the \( v \) wave \((Ry/v)\),¹¹ there was no clear-cut separation of the stenotic and incompetent groups, though a trend to higher values was evident in the presence of regurgitation. Similar results were obtained when the mean left atrial pressure was taken as a reference level \((Ry/LAP)\).¹² Use of the first 0.1 second of the \( y \) descent \((Ry^{0.1}/LAP)\) showed some advantage in that 2 patients (E.D. and M.G.) in the incompetent group were clearly distinguished (fig. 1). These were the only patients in which the left atrial and ventricu-
The Mechanism Producing Pressure Changes in the Left Atrium. Peterson studied the parameters determining the pressure response to pulsatile blood flow in large arteries and veins. He concluded that 3 factors were involved: acceleration, viscous friction, and distensibility. The introduction of a test volume into an isolated segment of the vascular system produces a brief pressure transient due to the acceleration factor and the development of viscous friction. Sustained flow is associated with a constant pressure due to viscous friction. If distention occurs, there is an additional pressure rise related to the physical characteristics of the system. These factors are illustrated in figure 2.

The similarity of the patterns derived from Peterson's study and the left atrial pressure pulse in patients with mitral valve disease and atrial fibrillation (fig. 3) suggested the application of these considerations to the analysis of the left atrial pressure pulse.

In mitral valve disease distention of the left atrium is a continuing factor, and the distensibility of the left atrial wall is a major
Fig. 3 Top. Left atrial pressure pulse in 11 patients with mitral valve disease. Comparison of the theoretical diagram, after Peterson, and the left atrial pressure pulse in 11 patients with mitral valve disease and atrial fibrillation.

Fig. 4 Bottom. Atrial pressure pulse expressed as travel along atriovenous volume pressure curve. Left atrial volume-pressure curve in 2 patients, one with mitral stenosis and a small atrium, the other with mitral regurgitation and a giant atrium. Cyclic volume gain and loss is greater in patient with regurgitation, but range of pressure change is similar to the stenotic case as the volume-pressure curve rises less steeply.

Fig. 5. Pulmonary vascular bed may be regarded as a screen flowmeter, flow varying linearly with pressure gradient. The mitral valve acts as an orifice flowmeter, flow varying with square root of gradient. Phasic flow changes may be estimated from variations in gradient.

Fig. 6. Phasic pressure gradient and flow rate across pulmonary vascular bed in hypothetical example of mitral valve disease with atrial fibrillation. Assuming a stroke volume index of 30 ml., the mean flow rate is 50 ml/sec. Flow is linearly related to pressure gradient and is maximal in early systole. Determinant of the pressure pulse. The relation of volume to pressure is not constant, but pressure changes are greater for a given volume change as the pressure rises. This is expressed as the volume-pressure curve (fig. 4), and variations in pressure with cyclic changes in volume may be regarded as movement along this curve.

Additional pulses are superimposed on this basic pattern. These include acceleration transients and viscous friction effects as described by Peterson, and changes due to atrial systole and diastole. The subsequent discussion is confined to the passive fibrillating atrium.

The Left Atrial Pressure Pulse as Movement Along a Volume-Pressure Curve. The mitral valve and the pulmonary vascular bed may be regarded as physiologic flowmeters of the orifice and the screen types (fig. 5). Phasic

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THE CONGESTED LUNG
THE MITRAL VALVE

As a Physiological Flow Meter
As a Physiological Flow Meter

Screen Flow Meter
Orifice Flow Meter

Pulmonary Flow Meter
Mitral Flow Meter

At constant resistance, flow varies linearly with pressure gradient.
Turbulent flow varies linearly with the square root of pressure gradient.
DETERMINANTS OF THE LEFT ATRIAL PRESSURE PULSE

**FIG. 7** Left. Phasic pressure gradient and flow across incompetent mitral valve in systole in hypothetical example. Assuming a regurgitant flow index of 25 ml. per beat, the mean flow rate is 82 ml./sec. Flow is proportional to square root of gradient and is maximal in early systole.

**FIG. 8** Right. Phasic pressure gradient and flow rate across stenotic mitral valve in diastole in hypothetical example. Assuming a stroke volume index of 30 ml., the mean flow rate is 90 ml./sec. Flow is proportional to square root of gradient and is maximal immediately after valve opens.

Changes in the pressure gradients are related to the variations in flow. At the mitral orifice, flow is turbulent and varies with the square root of the gradient. There is good evidence that the congested pulmonary vasculature behaves as a constant resistance, and flow across the lungs is therefore related directly to the pressure gradient. The significance of the phase difference between flow and pressure is difficult to evaluate and will not be considered here.

Hypothetical diagrams have been constructed to show the relationship between pressure gradient and flow across the pulmonary vascular bed (fig. 6) and through the mitral orifice in systole (fig. 7) and diastole (fig. 8) in mitral valve disease with atrial fibrillation. Flow into the left atrium from the lungs and through an incompetent mitral valve have similar time relations, reaching a peak in early systole and being curtailed in late systole by the rise in left atrial pressure. As rapid filling occurs on the lower, flatter part of the volume-pressure curve, a steady rise in left atrial pressure throughout systole results. When mitral incompetence complicates mitral stenosis, the magnitude but not the timing of the volume changes is affected (fig. 9).

The viscoelastic properties of the left atrium are important determinants of the pressure pulse. The absence of any correlation between left atrial volume and
pressure is evidence that the physical properties of the chamber vary from ease to ease. As the phasic volume change does not differ in timing in mitral stenosis and in combined stenosis and incompetence, a more distensible left atrium in the presence of regurgitation would result in a similar pressure pulse in the 2 situations. The tendency of the left atrium to be larger in mitral incompetence has a similar effect.

When the left atrial pressure pulse is considered as movement along a volume-pressure curve, it is apparent that a similar appearance is to be expected in pure mitral stenosis and in mitral stenosis with incompetence.

Acceleration Transients and Viscous Friction Effects. Acceleration transients are evident when there is a sudden change in the direction of blood flow. At the onset of ventricular systole the e wave appears to interrupt the steady rise in left atrial pressure. It probably represents an acceleration transient due to the abrupt check or reversal of flow through the mitral valve. At the peak of the v wave, the rapid outflow from the atrium when the mitral valve opens is associated with a corresponding negative effect that accentuates the initial phase of the y descent, and may produce a notch between this phase and the later, more gradual fall in left atrial pressure. In the presence of mitral incompetence the volume changes are greater, and these effects are accentuated (fig. 2).

Viscous friction produces a rise in pressure that is related to the velocity of flow. The velocity of systolic flow into the left atrium is greater in mitral incompetence than in isolated mitral stenosis. The viscous friction effect is therefore larger and will tend to make the ascending limb of the v wave more prominent (fig. 2).

The factors described will cause the left atrial pressure pulse to deviate from the pattern predicted on the basis of the volume-pressure curve. The increased prominence of these effects in the presence of mitral regurgitation is probably responsible for the partial success of the Ry/v ratio and similar indices in distinguishing these patients.

Summary

The left atrial pressure pulse in atrial fibrillation is an expression of travel along a volume-pressure curve during cyclic volume gain and loss.

Pressure changes due to acceleration transients and viscous friction effects are superimposed on this basic pattern.
An infinite number of combinations of stenosis and insufficiency could account for any pressure pulse, assuming larger, more distensible atria with increasing regurgitation.

No estimate of relative stenosis and insufficiency can be expected from the form of the pressure pulse in any individual case without knowledge of the atrial volume-pressure curve.

Larger acceleration transients and viscous friction effects may account for partial success of the Ry/v ratio and other methods in distinguishing mitral incompetence in the presence of stenosis.

REFERENCES


Seven persons without evident heart disease who were having a cholecystectomy were studied during various electroencephalographic levels of cyclopropane anesthesia. Cardiac index (by the Evans blue dilution method) and pulse rate decreased as the anesthesia deepened. Concomitantly, peripheral arterial resistance, radial arterial pressure, and superior vena cava pressure increased. Stroke volume decreased only during deep anesthesia and the central blood volume did not change significantly. These hemodynamic changes were considered possibly to be due to myocardial depression by cyclopropane or to an associated increase in circulating epinephrine-like substances; and, as the anesthesia was lightened, the circulatorv alterations returned rapidly toward control levels.

Rogers
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NEIL A. J. HAMER, SUJOY B. ROY and JAMES W. DOW

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