The Electrocardiographic Pattern of Right Ventricular Hypertrophy in Mitral Valve Disease

By Ralph C. Scott, M.D., Samuel Kaplan, M.D., Noble O. Fowler, Jr., M.D.
and William J. Stiles, M.D.

The electrocardiograms in 32 patients with mitral valve disease have been compared with the pulmonary resistances, the pulmonary artery pressures, and the size of the mitral valve orifice. In patients with pure stenosis if the electrocardiogram showed the pattern of right ventricular hypertrophy the total pulmonary resistance was usually 1000 dynes seconds cm.\(^{-5}\) or greater. If mitral insufficiency and/or aortic valve lesions were present in addition to mitral stenosis the total pulmonary resistance often exceeded 1000 dynes seconds cm.\(^{-5}\) without right ventricular hypertrophy being evident in the electrocardiogram.

The electrocardiographic pattern of right ventricular hypertrophy is frequently seen in patients with mitral stenosis. Lewis and associates\(^1\) have pointed out that if the level of total pulmonary resistance is 1000 dynes seconds cm.\(^{-5}\) or greater the electrocardiogram usually shows right ventricular hypertrophy.

Although right ventricular hypertrophy is often taken as evidence in favor of predominant mitral stenosis it is also known that this pattern can occur in patients with both mitral stenosis and insufficiency.\(^1\)

The purpose of the present study was to investigate further the correlation of the pattern of right ventricular hypertrophy in patients with mitral valve disease with the pulmonary artery pressures, the pulmonary resistances, and the mitral valve areas.

Material and Methods

Thirty-nine patients with mitral valve disease have undergone cardiac catheterization in the Cardiac Laboratory at the Cincinnati General Hospital during the past three years. The present report concerns only one phase of the study. The additional physiologic measurements obtained are described in a separate communication.\(^5\) Briefly, venous catheterization was carried out according to the method of Courmand and Ranges.\(^8\) The resting pressures were obtained in the right auricle, right ventricle, and pulmonary artery. Pulmonary "capillary" pressures were obtained by the method described by Hellem and co-workers.\(^4\) The pressures were recorded on a Hathaway recording oscillograph. The cardiac output was determined in the basal state according to the Fick principle. The mean "capillary," pulmonary artery, right ventricular and right auricular pressures were determined by planimetric integration.

Pulmonary arteriolar* resistance was calculated according to the formula:†

\[ R = \frac{P_{Am} - "PC"}{CO} \times 1332 \text{ dynes seconds cm.}^{-5} \]

Total pulmonary resistance was calculated as follows:

\[ R' = \frac{P_{Am} - 0}{CO} \times 1332 \text{ dynes seconds cm.}^{-5} \]

Mitral valve orifice areas were calculated as follows:\(^5\):

\[ MVA = \frac{MVF}{31 \sqrt{\text{"PC"}^2 - 5}} \]

Where \( P_{Am} \) = pulmonary arterial mean pressure in mm. Hg

"PC" = pulmonary "capillary" mean pressure in mm. Hg

CO = cardiac output, cc. per second

* "Arteriolar" is used to designate small vessels between the large pulmonary branches and the true pulmonary capillaries.

† This and the succeeding formulae have been criticized recently as not being correct.\(^6\),\(^7\) However, it has also been pointed out that although the results so obtained from them are crude they do serve a useful purpose in estimating the severity of augmented pulmonary or mitral valve resistances.\(^8\) Until better formulae are devised we shall employ them, being fully aware that the results they give are not absolute but only relative.
VENTRICULAR HYPERTROPHY IN MITRAL VALVE STENOSIS

1332 = conversion factor from mm. Hg to dynes per cm.²

\[ MVF = \text{m} \text{itral valve flow, cc. per diastolic second} \]

\[ \left( \frac{\text{Cardiac output, cc. per min.}}{\text{Diastolic filling period, seconds per min.}} \right) \]

5 = assumed left ventricular mean diastolic pressure, mm. Hg

31 = empirical constant.

Thirty-two of these patients had electrocardiograms consisting of the three standard leads, aVR, aVL, aVF, V1-V6 (and V5R in some) usually taken within a few days or at most a few weeks of the time of catheterization. Seventeen were thought to have pure stenosis on the basis of absent (or grade II or less) systolic mitral murmurs, no evidence of increased amplitude of the V wave in the pulmonary "capillary" pulse tracing, or the absence of a regurgitant jet at the time of operation (13 cases). Twelve cases were thought to have both mitral stenosis and mitral insufficiency. This was based on the presence of a grade III or more mitral systolic murmur as well as a diastolic murmur, increased amplitude of the V waves of the pulmonary "capillary" tracing, or the presence of a regurgitant jet at the time of operation (nine cases).

Three patients had combined valvular lesions. C. H. came to autopsy. He had mitral stenosis and insufficiency and aortic stenosis and insufficiency. The mitral regurgitation had been detected at the time of surgery. S. K. had mitral stenosis and aortic insufficiency (of a mild degree). H. F. had mitral stenosis and insufficiency and aortic stenosis.

The criteria used for right ventricular hypertrophy were those of a qR pattern in V4R or V3, an R wave in V1 of 7 mm. or greater, an R/S ratio in V1 greater than 1, or a delay in the onset of the intrinsicsoid deflection in V1, of from 0.035 to 0.05 second. Incomplete right bundle branch block was diagnosed on the basis of an rsR' pattern in V4R or V1, with delay in the onset of the intrinsicsoid deflection of R' from between 0.05 and 0.075 second. If the height of R' exceeded 10 mm. it was taken as evidence of right ventricular hypertrophy in addition to incomplete right bundle branch block. The P waves in the standard leads were considered to be abnormal if they were 0.12 second or greater in duration or 3 mm. or more in height, or were notched. Heart position was classified according to the criteria of Wilson. The patterns encountered in these 32 patients were compared with the resting pulmonary artery pressures (systolic and diastolic, and mean), with the pulmonary arteriolar resistance, with the total pulmonary vascular resistance, and with the mitral valve area.

RESULTS

Pure Mitral Stenosis (table 1)

Seventeen patients had pure mitral stenosis. The total pulmonary vascular resistance ranged from 608 to 4411 dynes seconds cm.⁻⁵. Nine cases showed the pattern of right ventricular hypertrophy, all except one having a total pulmonary vascular resistance exceeding 1000 dynes seconds cm.⁻⁵ and averaging 1822 dynes seconds cm.⁻⁵. Two cases exhibited incomplete right bundle branch block, one with a total pulmonary vascular resistance of 1163 and one with a resistance of 608 dynes seconds cm.⁻⁵.

Six cases presented no evidence of right ventricular hypertrophy or bundle branch block. The total pulmonary vascular resistance was less than 1000 dynes seconds cm.⁻⁵ in all, averaging 754 dynes seconds cm.⁻⁵.

The mean pulmonary artery pressure ranged from 41 to 90 mm. Hg, averaging 61 mm. Hg for the nine patients with right ventricular hypertrophy, 49 mm. Hg for the two with incomplete right bundle branch block, and 50 mm. Hg for the remaining six cases.

Four patients had auricular fibrillation. Of the 13 with sinus rhythm, twelve had abnormal P waves.

Sixteen of the 17 patients in whom the valve area was either estimated at operation or was calculated were found to have orifices of 1.0 cm.² or less. Four of the nine patients who had the pattern of right ventricular hypertrophy had mitral valve areas of 0.5 cm.² or less.

Mitrail Stenosis and Insufficiency (table 2)

Twelve patients had both mitral stenosis and insufficiency. The total pulmonary vascular resistance was measured in 11 and was found to range from 471 to 2384 dynes seconds cm.⁻⁵.

Four of the five cases who showed the pattern

* One of these nine cases (D. M.) showed a voltage of 23 mm. in aVR; this was interpreted as probably representing left ventricular hypertrophy although the cause for this was not apparent (no mitral insufficiency, aortic valve lesions, or systemic hypertension).
<table>
<thead>
<tr>
<th>Patient</th>
<th>Rhythm</th>
<th>Pressures (mm Hg)</th>
<th>Resistances (dyne sec cm(^{-2}))</th>
<th>Area Mitral Valve (cm(^2))</th>
<th>Electrocardiogram</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RA</td>
<td>&quot;PC&quot; PA</td>
<td>Pulmonary Arteriolar</td>
<td>Total Pulmonary</td>
<td>Abnormal P Waves</td>
</tr>
<tr>
<td></td>
<td></td>
<td>S/D Mean</td>
<td></td>
<td></td>
<td>Broad</td>
</tr>
<tr>
<td>B. P.</td>
<td>A. F.</td>
<td>22 -- 134--158</td>
<td>90 1758* 2813</td>
<td>&lt;0.5†</td>
<td>-</td>
</tr>
<tr>
<td>J. F.</td>
<td>Sinus</td>
<td>15 25 132/50</td>
<td>85 3114 4411</td>
<td>&lt;0.5†</td>
<td>+</td>
</tr>
<tr>
<td>M. E.</td>
<td>A. F.</td>
<td>15 -- 74--100</td>
<td>59 600* 1497</td>
<td>0.9†</td>
<td>-</td>
</tr>
<tr>
<td>M. H.</td>
<td>Sinus</td>
<td>9 -- 80/30</td>
<td>42 443* 1033</td>
<td>0.4†</td>
<td>+</td>
</tr>
<tr>
<td>H. S.</td>
<td>Sinus</td>
<td>8 22 68/46</td>
<td>51 368 648</td>
<td>1.4</td>
<td>+</td>
</tr>
<tr>
<td>J. Pi.</td>
<td>Sinus</td>
<td>2 26 84/48</td>
<td>60 897 1583</td>
<td>0.8</td>
<td>0</td>
</tr>
<tr>
<td>I. E.</td>
<td>Sinus</td>
<td>5 24 80/30</td>
<td>42 443 1033</td>
<td>0.6†</td>
<td>+</td>
</tr>
<tr>
<td>D. M.</td>
<td>Sinus</td>
<td>2 -- 85/25</td>
<td>52 425* 1299</td>
<td>0.8†</td>
<td>0</td>
</tr>
<tr>
<td>W. F.</td>
<td>Sinus</td>
<td>24 23 102/44</td>
<td>65 1795 2278</td>
<td>0.4†</td>
<td>+</td>
</tr>
<tr>
<td>M. S.</td>
<td>A. F.</td>
<td>4 23 70/35</td>
<td>54 668 1163</td>
<td>0.7†</td>
<td>-</td>
</tr>
<tr>
<td>I. K.</td>
<td>Sinus</td>
<td>7 -- 70/35</td>
<td>43 110* 608</td>
<td>1.0</td>
<td>+</td>
</tr>
<tr>
<td>S. K.</td>
<td>Sinus</td>
<td>6 -- 60/30</td>
<td>41 99* 674</td>
<td>0.7†</td>
<td>+</td>
</tr>
<tr>
<td>J. P.</td>
<td>Sinus</td>
<td>3 -- 74/36</td>
<td>51 204* 650</td>
<td>0.5†</td>
<td>0</td>
</tr>
<tr>
<td>G. W.</td>
<td>Sinus</td>
<td>9 27 62/33</td>
<td>42 311 889</td>
<td>&lt;0.5†</td>
<td>0</td>
</tr>
<tr>
<td>S. M.</td>
<td>Sinus</td>
<td>-- 98/52</td>
<td>78 -- 775</td>
<td>0.7†</td>
<td>+</td>
</tr>
<tr>
<td>H. B.</td>
<td>A. F.</td>
<td>5 27 54/26</td>
<td>43 293 786</td>
<td>0.8</td>
<td>-</td>
</tr>
<tr>
<td>A. T.</td>
<td>Sinus</td>
<td>17 54/32</td>
<td>42 447 751</td>
<td>0.3†</td>
<td>+</td>
</tr>
</tbody>
</table>

RA: Right auricular, PC: Pulmonary capillary; RVH: Right ventricular hypertrophy; IRBBB: Incomplete right bundle branch block; LVH: Left ventricular hypertrophy; V: Vertical; SV: Semivertical; I: Intermediate; Ind: Indeterminate.

* = Minimal pulmonary arteriolar resistance, calculated on an assumed pulmonary capillary pressure of 35 mm Hg.
† = Size of mitral valve orifice estimated at time of surgery.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Rhythm</th>
<th>Pressures (mm Hg)</th>
<th>Resistances (dyne sec cm(^{-2}))</th>
<th>Area Mitral Valve (cm(^2))</th>
<th>Electrocardiogram</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RA</td>
<td>&quot;PC&quot; PA</td>
<td>Pulmonary Arteriolar</td>
<td>Total Pulmonary</td>
<td>Abnormal P Waves</td>
</tr>
<tr>
<td></td>
<td></td>
<td>S/D Mean</td>
<td></td>
<td></td>
<td>Broad</td>
</tr>
<tr>
<td>F. J.</td>
<td>A. F.</td>
<td>18 23 78/43</td>
<td>50 773 1285</td>
<td>1.2</td>
<td>-</td>
</tr>
<tr>
<td>R. U.</td>
<td>A. F.</td>
<td>11 36 68/34</td>
<td>43 128 779</td>
<td>2.0</td>
<td>-</td>
</tr>
<tr>
<td>F. R.†</td>
<td>Sinus</td>
<td>13 -- 122/64</td>
<td>88 1436* 2384</td>
<td>0.8</td>
<td>0</td>
</tr>
<tr>
<td>W. S.†</td>
<td>Sinus</td>
<td>44 142/76</td>
<td>97 999 1828</td>
<td>0.8</td>
<td>0</td>
</tr>
<tr>
<td>P. L.†</td>
<td>Sinus</td>
<td>3 26 92/44</td>
<td>65 861 1435</td>
<td>0.9</td>
<td>+</td>
</tr>
<tr>
<td>S. C.</td>
<td>A. F.</td>
<td>-- 72/46</td>
<td>64 378 935</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>J. K.</td>
<td>Sinus</td>
<td>4 28 56/35</td>
<td>43 132 482</td>
<td>1.1</td>
<td>+</td>
</tr>
<tr>
<td>G. B.</td>
<td>Sinus</td>
<td>9 27 44/32</td>
<td>62 861 1435</td>
<td>0.9</td>
<td>+</td>
</tr>
<tr>
<td>E. McC.</td>
<td>A. F.</td>
<td>7 41 98/52</td>
<td>78 668 1407</td>
<td>0.6</td>
<td>-</td>
</tr>
<tr>
<td>V. H.</td>
<td>A. F.</td>
<td>14 -- 80/11</td>
<td>50 641* 1466</td>
<td>0.9</td>
<td>-</td>
</tr>
<tr>
<td>D. F.</td>
<td>Sinus</td>
<td>13 26 62/35</td>
<td>49 221 471</td>
<td>-</td>
<td>0</td>
</tr>
<tr>
<td>B. B.</td>
<td>Sinus</td>
<td>18 102/68</td>
<td>81 1058* 1861</td>
<td>0.4</td>
<td>0</td>
</tr>
</tbody>
</table>

* = Same as table 1 with following additions: Forward area of mitral valve: estimation of size of orifice of mitral valve at time of surgery.
† = Regurgitant jet very small at time of surgery.
of right ventricular hypertrophy had a total pulmonary vascular resistance of greater than 1000 dynes seconds cm.\(^{-5}\), averaging 1542 dynes seconds cm.\(^{-5}\). One case (R. U.) had both right ventricular hypertrophy and incomplete right bundle branch block, with a total pulmonary vascular resistance of 779 dynes seconds cm.\(^{-5}\).

Two patients (S. C. and V. H.) who had incomplete right bundle branch block had total pulmonary vascular resistances of 935 and 1466 dynes seconds cm.\(^{-5}\) respectively.

Two patients (E. McC. and B. B.) with resistances of 1407 and 1861 dynes seconds cm.\(^{-5}\) showed neither right ventricular hypertrophy nor right bundle branch block. One case (D. F.) showed increased voltage and delay in the onset of the intrinsicoid deflection in leads over the left ventricle; these changes were thought to be consistent with left ventricular hypertrophy.\(^{15}\)

The mean pulmonary artery pressure ranged from 43 to 97 mm. Hg with an average of 70 for the five patients with right ventricular hypertrophy, 67 for the two with incomplete right bundle branch block, and 63 for the remaining five patients.

Five patients showed auricular fibrillation. Of the seven who showed sinus rhythm, four had abnormal P waves.

The mitral valve area was measured at surgery in nine patients. It was found to range from 0.4 to 2.0 cm.\(^2\).

**Mitral and Aortic Valve Disease (table 3)**

Three patients had mitral and aortic valve disease. The total pulmonary vascular resistance ranged from 594 to 5033 dynes seconds cm.\(^{-5}\) with an average of 2514. None of these cases showed either right ventricular hypertrophy or right bundle branch block.

The mean pulmonary artery pressure ranged from 29 to 100 mm. Hg with an average of 68.

Two cases had auricular fibrillation and the one with sinus rhythm had abnormal P waves.

The mitral valve area was measured at surgery in two patients, being 1 cm.\(^2\) in one case and 1.1 cm.\(^2\) in the second.

**Discussion**

Our findings in cases of pure mitral stenosis are essentially in agreement with those of Lewis and his group.\(^{1}\) If the total pulmonary resistance (which is a function of both pulmonary arteriolar resistance and mitral valve area) is 1000 dynes seconds cm.\(^{-5}\) or greater then the pattern of right ventricular hypertrophy or incomplete right bundle branch block is usually (but not invariably) present.

On the other hand we found that if in addition to mitral stenosis, mitral insufficiency and/or aortic valve lesions exist, total pulmonary resistance may well exceed 1000 dynes seconds cm.\(^{-6}\) without the pattern of right ventricular hypertrophy or incomplete right
bundle branch block being present. In other words, in lesions which will cause strain on the left ventricle, even though the total pulmonary resistance is greater than 1000 dynes seconds cm.−5, the pattern of right ventricular hypertrophy may not be present.

Johnson and associates9 found in their patients with cor pulmonale secondary to chronic lung disease that if the pattern of right ventricular hypertrophy occurred the pulmonary artery mean pressure almost always exceeded 30 mm. Hg. Our findings in cases of mitral stenosis are in agreement with this. On the other hand, like Johnson and associates,9 we also had a number of patients (12 in our study) in whom the mean pulmonary artery pressure exceeded 30 mm. Hg who did not show either right ventricular hypertrophy or incomplete right bundle branch block.

Only four of the 32 patients studied failed to show either auricular fibrillation or abnormal P waves. Most of our cases had rather severe mitral stenosis. This high incidence of auricular abnormalities may not be found in patients with lesser degrees of stenosis.

The pulmonary “capillary” pressure (presumably a reflection of left auricular pressure) was found to be elevated, as would be expected, in all patients in whom it was measured.

Eight of the nine patients with pure stenosis who had the pattern of right ventricular hypertrophy had mitral valve areas less than 1.0 cm.2. The total pulmonary resistance which is a measure of the resistance of the narrowed pulmonary arterioles (pulmonary arteriolar resistance) and the stenosed mitral valve1 was greater than 1000 dynes seconds cm.−5 in eight of the nine cases. The single exception was one patient (H. S.) who had a valve area of 1.4 cm.2 and a total pulmonary vascular resistance of 648 dynes seconds cm.−5.

On the contrary two patients (G. W., A. T.) with valve areas of less than 0.5 cm.2 and total pulmonary vascular resistances of 889 and 751 dynes seconds cm.−4, respectively, failed to show right ventricular hypertrophy. This is in accord with the concept of Lewis and co-workers7 that the electrocardiographic changes diagnostic of right ventricular hypertrophy are a function of both mitral valve area and pulmonary arteriolar resistance. Thus, although the mitral valve was tight in both patients, the pulmonary arteriolar resistances (311 and 447 dynes seconds cm.−4, respectively) were not markedly increased so that the total pulmonary resistance did not reach the critical level of 1000 dynes seconds cm.−5 above which the pattern of right ventricular hypertrophy usually develops in pure mitral stenosis.

**Summary**

The electrocardiographic patterns encountered in 32 patients with mitral valve disease were compared with the resting pulmonary artery pressures, with the pulmonary resistances and with the mitral valve areas.

Seventeen patients had pure mitral stenosis. Nine of these showed the pattern of right ventricular hypertrophy and two showed incomplete right bundle branch block. Eight of the nine with hypertrophy and one with incomplete right bundle branch block had total pulmonary resistances in excess of 1000 dynes seconds cm.−5.

In pure mitral stenosis if the total pulmonary resistance is 1000 dynes seconds cm.−5 or greater the electrocardiogram will usually (although not invariably) show the pattern of either right ventricular hypertrophy or incomplete right bundle branch block.

If mitral insufficiency and/or aortic valve lesions co-exist with mitral stenosis, the total pulmonary resistance may exceed the level of 1000 dynes seconds cm.−5 many times without the development of the pattern of right ventricular hypertrophy.

When the electrocardiographic pattern of right ventricular hypertrophy is encountered in mitral valve disease the following inferences may be drawn:

a. The patient does not necessarily have pure mitral stenosis although it is probably the predominant lesion.

b. The total pulmonary resistance is probably 1000 dynes seconds cm.−5 or greater.

**Summario in Interlingua**

In 32 patienzen de morbo del valvula mitral, comparationes esseva executate inter le configurationes electrocardiographic e le valores
obtenite in mesuraciones del residentias pulmonar, del pressiones del arteria pulmonar, e del dimensions del orificio del valvula mitral. In pacientes con pur stenosis, le presentia de configurationes de hypertrophia dexteroventricular in le electrocardiogramma correspondeva generalmente a residentias pulmonar total de 1000 dyna-sec-cm$^3$ o plus. Si ultra stenosis mitral le paciente habeva insufficientia mitral e/o lesiones del valvula aortic, le residentias pulmonar total frequentemente excedeva 1000 dyna-sec-cm$^3$ sin evidencia de hypertrophia dexteroventricular in le electrocardiogramma.

**ACKNOWLEDGMENT**


The authors wish to acknowledge the valuable help of Dr. Richard N. Westcott, Dr. Emily Hess, Dr. Robert Heln, and Dr. Isom Walker in the study of some of the patients in this investigation.

The authors wish to thank Dr. M. A. Blankenhorn and Dr. Johnson McGuire for their helpful criticisms and suggestions.

**REFERENCES**


The Electrocardiographic Pattern of Right Ventricular Hypertrophy in Mitral Valve Disease
RALPH C. SCOTT, SAMUEL KAPLAN, NOBLE O. FOWLER, JR. and WILLIAM J. STILES

Circulation. 1955;11:761-766
doi: 10.1161/01.CIR.11.5.761
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1955 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/11/5/761

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/