HEMODYNAMIC CORRELATES OF VECTORCARDIOGRAPHIC
QRS LOOPS IN PURE MITRAL STENOSIS

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The hemodynamic correlates of the vectorcardiographic types of right ventricular hypertrophy (RVH) according to Chou and Helm and those with normal QRS loop in the horizontal plane of Frank system were analyzed in 100 patients with pure mitral stenosis. All underwent right and left heart catheterization. Additionally, coronary arteriography was done on 16 whose ages were above 40. Type A RVH was associated with the most severe hemodynamic alterations with markedly elevated total pulmonary vascular resistance (TPVR), mean pulmonary artery pressure (MPAP), peak right ventricular pressure (RRVP) and the smallest mitral valve area (MVA). The severity of these parameters were to a lesser degree obtainable in type C but with no significant difference from type A (p > 0.05). However, types A and C were clearly separated from type B and normal QRS loop (p < 0.05). Type B RVH and normal QRS loop showed milder hemodynamic changes and were not significantly different (p > 0.05). Our results indicate that in pure mitral stenosis the development of RVH is from a normal loop into type B, C and A reflecting an increasing severity of hemodynamic changes which affect the right ventricle. This order of development is different from the traditional view.

OBSTRACTION of the inflow tract into the left ventricle as found in pure mitral stenosis produces a chain of events behind the blockade which is capable of altering the vectorial forces responsible for the insulation of the QRS loop. Therefore, it would seem logical to assume that various degrees of hemodynamic changes obtainable in this condition will have different expressions of right ventricular strain and/or hypertrophy. The vectorcardiographic patterns of the QRS loop in right ventricular hypertrophy (RVH) have been described by Chou and Helm. These patterns have been associated with a variety of cardiac and lung diseases.

The vectorcardiogram has been found superior to the electrocardiogram in detecting RVH.7 Using computer multi-variate analysis Walston et al.8 have improved the recognition of RVH in mitral stenosis at each pulmonary artery pressure level. In 1969, studying only the horizontal plane Lal et al.9 found vectorcardiographic abnormalities which indicate RVH with a mean pulmonary artery pressure of greater than 40 mmHg in 20 patients with mitral stenosis. We will report the relationship of the types of QRS loops in the horizontal plane to the hemodynamic changes in 100 patients with pure mitral stenosis.

MATERIALS AND METHODS

One hundred patients with pure mitral stenosis diagnosed hemodynamically constituted the sub-

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TABLE I DISTRIBUTION OF ONE HUNDRED PATIENTS ACCORDING TO THE QRS LOOP ON THE HORIZONTAL PLANE

<table>
<thead>
<tr>
<th>Group</th>
<th>QRS loop</th>
<th>No. of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Type A RVH</td>
<td>42</td>
</tr>
<tr>
<td>II</td>
<td>Type B RVH</td>
<td>21</td>
</tr>
<tr>
<td>III</td>
<td>Type C RVH</td>
<td>23</td>
</tr>
<tr>
<td>IV</td>
<td>Normal</td>
<td>14</td>
</tr>
</tbody>
</table>

Fig. 1. A 37-year-old male with type A right ventricular hypertrophy (RVH). In this and subsequent figures the direction of the QRS loop is indicated by the dot and dash (----) sequence. The hemodynamic parameters are:

- TPVR = 1772 dyne·sec·cm⁻⁵
- MPAP = 57.3 mmHg
- PRVP = 80 mmHg
- MVA = 0.5 cm²

Fig. 2. A 28-year-old male with type B RVH. The hemodynamic parameters are:

- TPVR = 175 dyne·sec·cm⁻⁵
- MPAP = 20 mmHg
- PRVP = 36 mmHg
- MVA = 1.8 cm²

Fig. 3. A 23-year-old male with type C RVH. The hemodynamic parameters are:

- TPVR = 1737 dyne·sec·cm⁻⁵
- MPAP = 77.3 mmHg
- PRVP = 100 mmHg
- MVA = 0.8 cm²

Projects of this study. Forty-seven were males and 53 were females. Their ages ranged from 16 to 68. There were 16 over 40 years old, 10 below 20 and 74 between 20 and 40.

All underwent right and left heart catheterization. The clinical charts were reviewed to exclude pulmonary disease, ischemic heart disease, systemic arterial hypertension, primary pulmonary hypertension, congenital heart disease and other valvular lesions. All 16 over 40 years old had coronary arteriography to rule out significant coronary artery disease.

The hemodynamic parameters studied were:

1) total pulmonary vascular resistance (TPVR),
2) mean pulmonary artery pressure (MPAP),
3) peak right ventricular pressure (PRVP) and
4) mitral valve area (MVA).

The vectorcardiograms were taken with ICR (Instruments for Cardiac Research) Instant Vectorcardiograph Model #VCG-1B using the Frank system of electrode placement. All had vectorcardiograms within 2 days prior to cardiac catheterization. The horizontal vectorcardiograms were analyzed according to the following 3 types of RVH described by Chou et al.¹,¹⁰:

Type A: The QRS loop is located anteriorly and to the right with a clockwise (CW)
TABLE II HEMODYNAMIC PARAMETERS (MEAN ± SD) OF THE FOUR GROUPS ACCORDING TO THEIR QRS LOOPS

<table>
<thead>
<tr>
<th>Group</th>
<th>QRS loop</th>
<th>TPVR (dynes·sec·cm⁻²)</th>
<th>MPAP (mmHg)</th>
<th>PRVP (mmHg)</th>
<th>MVA (cm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>A</td>
<td>1066.4 ± 713</td>
<td>53.87 ± 15.66</td>
<td>78.5 ± 22.37</td>
<td>0.56 ± 0.17</td>
</tr>
<tr>
<td>II</td>
<td>B</td>
<td>343.3 ± 258</td>
<td>38.24 ± 13.43</td>
<td>57.0 ± 18.64</td>
<td>1.52 ± 0.47</td>
</tr>
<tr>
<td>III</td>
<td>C</td>
<td>800.6 ± 541</td>
<td>50.5 ± 17.5</td>
<td>70.9 ± 24.5</td>
<td>0.64 ± 0.26</td>
</tr>
<tr>
<td>IV</td>
<td>Normal</td>
<td>234.3 ± 258</td>
<td>34.0 ± 12.7</td>
<td>51.0 ± 19.45</td>
<td>1.80 ± 0.5</td>
</tr>
</tbody>
</table>

TPVR = total pulmonary vascular resistance; MPAP = mean pulmonary artery pressure; PRVP = peak right ventricular pressure; MVA = mitral valve area

Rotation.

Type B: The QRS loop has an increased anterior area such that 70% is anterior but displaced to the left with a counterclockwise (CCW) rotation.

Type C: The QRS loop is displaced posteriorly to the right with a CCW rotation. The major portion of the loop is in the left posterior and right posterior quadrants. The terminal portion of the loop in the right posterior quadrant is 20% or greater.

In the present study the 100 patients were grouped into I (type A), II (type B), III (type C) and IV (normal QRS loop). All QRS loops in group IV met the criteria for normal by Chou et al.10 Cooksey et al.11 and Bristow12 on the horizontal plane. None of the QRS loops were more than 0.10 sec in duration. Table I shows the distribution of cases according to the QRS loop on the horizontal plane.

The mean values of the different hemodynamic parameters in each group were determined. The significance of the differences of the mean values between groups was determined by the analysis of variance (ANOVA).

RESULTS

Eighty-six patients satisfied the vectorcardiographic criteria for RVH, while 14 showed normal QRS loop. Those with RVH were distributed in groups I, II and III, and the normals in group IV. Forty-two patients met the criteria of type A RVH, 21 type B RVH and 23 type C RVH. Their ages ranged from 16 to 68, from 16 to 57 and from 16 to 58, respectively. Fourteen patients whose ages ranged from 20 to 53 showed a normal QRS loop constituting Group IV. Representative vectorcardiograms and the altered hemodynamics in types A, B and C RVH, and normal QRS loop are illustrated in Figs. 1 to 4.

All hemodynamic parameters showed elevated values. Types A and C showed the most severe abnormalities, while type B and normal QRS loop displayed milder derangements. These changes are summarized in Table II.

The coronary arteriograms of 16 patients over 40 years old were all normal.

Total Pulmonary Vascular Resistance

Group I (type A RVH) was associated with the highest mean TPVR (1066.4 ± 7.3 dynes·sec·cm⁻²). This value is significantly higher (p < 0.05) than that of Group IV (normal QRS loop) (234.3 ± 258 dynes·sec·cm⁻²) or that of Group II (type B RVH) (343.3 ± 258). Group III (type C RVH), had the second highest mean TPVR (800.6 ± 541 dynes·sec·cm⁻²). Although the mean TPVR of Group III was less than that of Group I, its difference was statistically not significant (p > 0.05). This value however is sig-
significantly higher than that of Group II (type B RVH) and Group IV (p < 0.05). The mean TPVR of Group II was not significantly higher than that of Group IV (p < 0.05).

Mean Pulmonary Artery Pressure

Group I showed the highest mean value of 53.87 ± 15.66 mmHg. This value is significantly higher than that of Group II (38.24 ± 13.43 mmHg) and Group IV (34 ± 12.7) (p < 0.05). The MPAP of Group III (50.5 ± 17.5 mmHg) was significantly higher than either Group II or IV (p < 0.05). The difference between the mean values of Groups I and III were statistically not significant (p > 0.05). Although the MPAP of Group II was higher than that of Group IV, the difference was statistically not significant (p > 0.05).

Peak Right Ventricular Pressure

The PRVP of Group I was the highest, followed closely by that of Group III, that of Group II and that of Group IV in descending order. The PRVP of Group I (78.5 ± 22.37 mmHg) and Group III (70.9 ± 24.5) were significantly higher (p < 0.05) that of Group II (57 ± 18.64) and Group IV (51.0 ± 19.45). The difference between Groups I and III was not significant (p > 0.05). The same was true concerning the difference between Groups II and IV (p > 0.05).

Mitral Valve Area

Group I was associated with the smallest mitral valve orifice with a mean MVA of 0.56 ± 0.17 cm², which was followed by that of Group III with a mean MVA of 0.64 ± 0.26. Group II had a mean MVA of 1.52 ± 0.47 cm², while Group IV had a value of 1.8 ± 0.5. Again, Groups I and III were clearly separated from Groups II and IV (p < 0.05).

DISCUSSION

The qualitative description of the different types of RVH by Chou and his associates¹,¹⁰ and Cooksey et al.¹¹ has made the clinical recognition of right ventricular hypertrophy quite easy and expeditious. This is not to preclude the usefulness of a more meticulous study of the instantaneous vectors which are exceedingly important in recognizing RVH, as suggested by Cueto et al.¹³ It is well known that the QRS loop in RVH varies in configuration and orientation. Such changes in configuration have been correlated with certain hemodynamic parameters⁵,¹³,¹⁴ Both PRVP and TPVR have a close relation with the degrees of RVH. It has been suggested that the higher the PRVP and TPVR, the more severe the RVH.

The classic patterns of RVH were described by Chou and Helm¹ into types A, B and C. Type A RVH was associated with severe degree of RVH commonly due to congenital heart disease, but it was also found in advanced cases of cor pulmonale and mitral stenosis. The dominant electrical forces from the hypertrophied free wall.
of the right ventricle make the anterior loop predominant causing a CW rotation on the transverse plane. The predominantly anterior type B loop with a normal rotation on the transverse plane is considered to be the pattern of moderate degree of RVH. The milder form of hypertrophy is associated with the posteriorly oriented type C loop. Selective hypertrophy of the outflow tract of the right ventricle is believed to be responsible for the posterior and the rightward orientation of the terminal portion of type C RVH.

The natural history of mitral stenosis is manifested by the progression of the right ventricular strain. Pure mitral stenosis should therefore serve as a model to evaluate the validity of the RVH pattern of the QRS loop in relation to the increasing hemodynamic abnormalities. Clinically patients with significant left ventricular disease were ruled out in our study by a thorough review of the clinical chart, right and left heart catheterization in all patients and coronary arteriography in those over 40 years old. It is important to exclude patients with left ventricular abnormalities, because abnormal left ventricular forces affect the QRS loop. Cabrera and Gaxiola have pointed out that the patterns of ventricular overloading are determined by the hemodynamic conditions of both ventricles. The electrical forces of the opposite ventricle contribute significantly to the final morphology of the QRS loop of the ipsilateral ventricle. Previous studies included patients with mixed valvular lesions and various clinical entities. In another report on 37 patients with "pure" mitral stenosis, the mean age was greater than 40 but no coronary arteriography was done to exclude coronary artery disease.

All hemodynamic parameters measured in this report show severe alterations in type A RVH as compared with other types. The incidence of type A (42%) in our series is higher than that in other studies. In the series of Mershon and his associates consisting of 44 patients with predominant MS, 6 (13%) had type A. Lal et al. have reported 4 type A RVH loops out of 6 who had an MPAP of greater than 40 mmHg. Taymore and his coworkers have found in 29 with pure MS only 5 with dominant anterior forces on the horizontal plane showing a CW or CCW rotation. They reported the predominance of posteriorly oriented loop with a normal rotation (type C) in 19 and suggested that this pattern represents early RVH.

Our results showed that type C RVH is associated with significantly greater hemodynamic derangement of all parameters than type B, and that the hemodynamic changes of type C were not significantly less than those of type A. The incidence of types B and C in our series was 21% and 23%, respectively.

The type C or the emphysematous loop has been observed frequently in patients with chronic lung disease. In 1964, Graf et al. drew attention to this type of QRS loop in mitral stenosis and explained it on the basis of similar pathologic changes of the alveolar wall in mitral stenosis and chronic pulmonary emphysema. In the absence of significant pulmonary disease, several mechanisms have been proposed. Taymore et al. considered the rotation of the heart to a more posterior and vertical position due to selective hypertrophy of the right ventricular outflow tract to be an important mechanism. That the higher electrical conductivity of the blood in the dilated left atrium accentuates the posterior electrical position of the heart was suggested by Hamer in his vectorcardiographic study on mixed mitral valve lesions. The increase in the boundary subtended by the solid angle due to the large mass of blood in the dilated left atrium may produce more posteriorly oriented QRS vectors. No study has been reported in the literature which correlates the type of RVH loop with the size of the left atrium. However, we noticed that patients with type C RVH had a significantly more dilated left atrium than those with type A or type B RVH as determined echocardiographically. We are presently conducting an echocardiographic correlation of the left atrial size with the type of RVH loop in pure mitral stenosis in our laboratory.

The type B RVH has been suggested to represent moderate hypertrophy. All hemodynamic parameters in this type, including PRVP in our study, were not significantly higher than those in patients with a normal QRS loop. This is different from the previous observation which suggested that type B RVH is a halfway position between types A and C, using PRVP as an important parameter. However, when the other parameters were considered Mershon et al. concluded that type B indicates mild RVH.

Patients with a normal QRS loop were associated with the mildest hemodynamic alterations. It appears that the normal QRS loop develops almost imperceptibly into type B RVH as the hemodynamic changes become more

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pronounced (Fig. 5). Indeed the traditional concept of the degree of RVH according to the type of QRS loop does not appear to apply to patients with pure mitral stenosis. Our data suggest that in pure mitral stenosis the development of RVH is from the normal QRS loop into types B, C and A in increasing order of severity of hemodynamic changes operating on the diseased right ventricle.

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