Pulmonary Stenosis
Electrocardiographic, Vectorcardiographic, and Catheterization Data

By LEONARD SCHERLIS, M.D., ROLF J. KOENKER, M.D.,
and YU-CHEN LEE, M.D.

When pulmonary stenosis is accompanied by a septal defect, the work imposed on each ventricle differs from that imposed when pulmonary stenosis exists as an isolated lesion. Isolated pulmonary stenosis is characterized by an increased load on the right ventricle during the ejection phase. Pulmonary stenosis associated with an interatrial communication combines this feature with an increased pulmonary blood flow, when the shunt is from left to right. When pulmonary stenosis occurs with a ventricular septal defect, increased loads may be placed on both the right and left ventricles.

Numerous electrocardiographic studies of isolated or complicated pulmonary stenosis have been reported. However, there are relatively few vectorcardiographic studies. Electrocardiographic, vectorcardiographic, and catheterization data in 100 patients with isolated or complicated pulmonary stenosis are analyzed in this report, and correlated with data of right heart catheterization, the anatomic lesion, and the morphology of the electrocardiogram and vectorcardiogram.

Material and Methods

This study includes 100 patients observed at the University Hospital of the University of Maryland School of Medicine in whom the diagnosis of pulmonary stenosis was confirmed by right heart catheterization. In many instances, this included dye-dilution or Krypton-85 studies. There were 45 patients with isolated pulmonary stenosis, 21 with pulmonary stenosis and interatrial communication, and 34 patients with pulmonary stenosis and ventricular septal defects. The diagnosis was further confirmed at operation in 55 patients.

Routine scalar electrocardiograms including V3R and vectorcardiograms with use of the cube system of electrode placement were obtained in each patient. The electrocardiograms were analyzed for rate, rhythm, P-R interval, amplitude and duration of the P wave, QRS duration, frontal plane axis, R and S wave amplitude, and R:S ratio in aV1, V3R, V1 and V6; intrinsicoid deflection in V3R, V1, and V6; configuration of P wave in V3R and V1. The electrocardiographic and vectorcardiographic diagnosis of right ventricular hypertrophy was based on the various criteria summarized in our previous report. The electrocardiographic diagnosis of left ventricular hypertrophy and combined ventricular hypertrophy was based on the various criteria summarized by Scott. The vectorcardiographic diagnosis of left ventricular hypertrophy utilizes the criteria of Grishman and Scherlis and Mazzoleni et al. The criteria of Beregovich et al., Dack et al., and Pileggi et al. were applied for the vectorcardiographic diagnosis of combined ventricular hypertrophy.

Of the 100 patients studied, 52 were male and 48 were female. The ages of the patients ranged from 1 year to 62 years (table 1).

Results

Analysis of Electrocardiograms

Rhythm. Normal sinus rhythm was present in all but two patients. One patient, 62 years of age, had atrial fibrillation; the other, a child age 8, had an electrocardiogram charac-
teristic of the Wolff-Parkinson-White syndrome. Each patient had pulmonary stenosis and an atrial septal defect.

P Waves. Thirty-three tracings revealed P-wave enlargement; ten were recorded in patients with isolated pulmonary stenosis, nine in pulmonary stenosis with interatrial communication, and 14 in pulmonary stenosis with a ventricular septal defect.

P-R interval. The P-R interval was 0.20 second or less in the 38 patients over the age of 13. In five of these patients, it was 0.20 second. In 20 of the 62 children under the age of 13 years, the P-R interval was 0.16 second or longer. It was 0.16 second in 16 tracings, 0.17 in two, 0.19 in one, 0.20 in one, and 0.24 in one. The criteria of Alimurung and Massell were used for children.

QRS complex. The QRS duration was less than 0.10 second in 93 tracings, 0.10 to 0.11 second in four, and 0.12 second in three. In the last group, there were two instances of atrial septal defects, one of whom had the Wolff-Parkinson-White syndrome, and one ventricular septal defect in addition to pulmonary stenosis.

The configuration of the QRS complex in lead V1 is shown in table 2.

QRS axis. In most tracings, the frontal QRS axis ranged from +60 to -150° (fig. 1). Of four patients with left axis deviation, three were complicated by a ventricular septal defect with a left-to-right shunt. The remaining patient with left axis had an electrocardiogram characteristic of the Wolff-Parkinson-White syndrome, and one ventricular septal defect in addition to pulmonary stenosis.

The configuration of the QRS complex in lead V1 is shown in table 2.

Table 2

<table>
<thead>
<tr>
<th>Configuration of QRS in V1</th>
<th>rS</th>
<th>rSr'</th>
<th>rSR'(rR')</th>
<th>R</th>
<th>BS (Rs)</th>
<th>qR (qr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PS</td>
<td>7</td>
<td>1</td>
<td>5</td>
<td>11</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>PS + IAC</td>
<td>3</td>
<td>2</td>
<td>5</td>
<td>7</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>PS + VSD</td>
<td>3</td>
<td>4</td>
<td>9</td>
<td>15</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>13</td>
<td>1</td>
<td>11</td>
<td>25</td>
<td>43</td>
<td>8</td>
</tr>
</tbody>
</table>

PS, pulmonary stenosis; IAC, interatrial communication; VSD, ventricular septal defect.

Figure 1

The distribution of the frontal QRS axis is shown in isolated pulmonary stenosis, pulmonary stenosis plus interatrial communication, and pulmonary stenosis plus ventricular septal defect. In each figure, the solid symbol indicates that surgery was performed. In the open symbol, surgery was not performed.
White syndrome and had pulmonary stenosis with an atrial septal defect.

The presence of electrocardiographic evidence of right ventricular hypertrophy was evaluated upon the criteria listed in table 3.34–37

In seven patients, none of these criteria was met. Among those patients with pulmonary stenosis and a ventricular septal defect, two had characteristic evidence of left ventricular hypertrophy, and four had combined ventricular hypertrophy. The sum of the S deflection in V1 plus the R deflection in V5 or V6 was more than 40 mm. in three patients who had isolated pulmonary stenosis.

**Catheterization Data**

The right ventricular systolic pressure ranged from 35 to 200 mm. Hg. In 14 patients, it was under 50 mm. Hg; in 56 instances, between 50 and 100 mm. Hg; and in 30 cases, more than 100 mm. Hg. The systolic gradients across the pulmonary valve ranged from 20 to 160 mm. Hg. In one patient whose right ventricular pressure was 200 mm. Hg, the pulmonary pressure was not measured. The average systolic pressure in the right ventricle was 77 mm. Hg in the group with isolated pulmonary stenosis; 98 mm. Hg among those who had pulmonary stenosis and an intratral communication; 97 mm. Hg among those who had pulmonary stenosis and a ventricular septal defect.

**Table 3**

<table>
<thead>
<tr>
<th>Right Ventricular Hypertrophy: Electrocardiographic Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Criteria</td>
</tr>
<tr>
<td>-----------</td>
</tr>
<tr>
<td>No. cases</td>
</tr>
<tr>
<td>1. qR in V₅₆ or V₇</td>
</tr>
<tr>
<td>2. R in V₅ equal to or more than 7 mm.</td>
</tr>
<tr>
<td>3. R in V₇ equal to or more than 5 mm.</td>
</tr>
<tr>
<td>4. R/S in V₅ equal to or more than 1.</td>
</tr>
<tr>
<td>5. Intrinsocoid deflection in V₁, 0.035 to 0.05 second.</td>
</tr>
<tr>
<td>6. P enlargement.</td>
</tr>
<tr>
<td>7. rsR' in V₅₆ or V₇ with intrinsicoid deflection 0.05 to 0.075 second.</td>
</tr>
<tr>
<td>8. Criterion 7 plus R' more than 10 mm.</td>
</tr>
<tr>
<td>9. Axis of +110° to —90°.</td>
</tr>
<tr>
<td>10. R in aV₅ equal to or more than 5 mm.</td>
</tr>
<tr>
<td>11. R/S in V₅₆ equal to or more than 1.</td>
</tr>
</tbody>
</table>

The average right ventricular systolic pressure was 53 mm. Hg in the rS group, 71 mm. Hg in the rsR' (including one with rSr') and 96 mm. Hg among those with qR, R, or Rs in V₁.

The average right ventricular systolic pressure in the group with a frontal QRS axis of 0° to 90° was 55 mm. Hg; in the group with a QRS axis of + 91° to —91°, 103 mm. Hg; and with a QRS axis of 0° to —90°, 76 mm. Hg.

The relationship between right ventricular systolic pressure and the amplitude of the R wave in V₁ is shown in figure 2. Although exceptions exist, the amplitude of the R wave in V₁ tends to increase as the right ventricular systolic pressure increases.

**Analysis of Vectorcardiogram**

The vectorcardiograms were classified as follows: five normal, 79 right ventricular hypertrophy, one Wolff-Parkinson-White syndrome, one terminal conduction delay of the right bundle-branch block type, 12 combined ventricular hypertrophy, and two left ventricular hypertrophy.

According to the configuration of the QRS sE loop in the horizontal plane, the vectorcardiograms with right ventricular hypertrophy were divided into three subgroups: 12 were type 1, seven type 2, and 60 type 3.

The distribution of the vectorcardiographic findings in each lesion is shown in table 4. Types 1, 2, and 3 were described in detail in our previous communication.27 Briefly, type 1 included those with a figure-eight configuration (fig. 3) or with a terminal appendage but without slowing in the horizontal projection.

The direction of inscription of the QRS sE loop in the horizontal plane is clockwise in both types 2 (fig. 4) and 3 (figs. 5, 6, and 7). However, the centrifugal QRS sE loop extends significantly to the left in type 2, whereas in type 3 it extends very little to the left and the entire QRS sE loop is displaced markedly to the right and anteriorly. Some of the tracings classified as type 3 were characterized by a figure-eight configuration in
The relationship between right ventricular systolic pressure and the amplitude of the R wave in V; is shown in isolated pulmonary stenosis, pulmonary stenosis plus interatrial communication, and pulmonary stenosis plus ventricular septal defect.

Discussion

Isolated Pulmonary Stenosis

In the present study, all 45 patients had normal sinus rhythm. There was P-wave enlargement in 10 electrocardiograms, and in

Table 4

Vectorcardiographic Diagnosis and Anatomic Lesion

<table>
<thead>
<tr>
<th>Lesion</th>
<th>PS isolated</th>
<th>PS + IAC</th>
<th>PS + VSD</th>
</tr>
</thead>
<tbody>
<tr>
<td>VCG type</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>4</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>RVH-type 1</td>
<td>9</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>RVH-type 2</td>
<td>3</td>
<td></td>
<td>4</td>
</tr>
<tr>
<td>RVH-type 3</td>
<td>21</td>
<td>17</td>
<td>22*</td>
</tr>
<tr>
<td>TCD</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVH</td>
<td>1</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>CVH</td>
<td>6</td>
<td></td>
<td>6</td>
</tr>
<tr>
<td>W-P-W</td>
<td>1</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*One record revealed diffuse conduction delay (QRS: 0.12 second).

RVH, right ventricular hypertrophy; TCD, terminal conduction delay; LVH, left ventricular hypertrophy; CVH, combined ventricular hypertrophy; PS, pulmonary stenosis; IAC, interatrial communication; VSD, ventricular septal defect.
H. E., age 18. Isolated pulmonary stenosis. Right ventricular systolic pressure 40 mm. Hg. Pulmonary artery systolic pressure 20 mm. Hg. The vectorcardiogram reveals type 1 right ventricular hypertrophy. The slight difference in the configuration of the QRS sE loop in the horizontal plane is due to respiratory changes. The electrocardiogram reveals a vertical QRS axis in the frontal plane and RS configuration in V1.
E. S., age 8. Pulmonary stenosis and ventricular septal defect with right-to-left shunt. Right ventricular systolic pressure 65 mm. Hg. Pulmonary artery systolic pressure 30 mm. Hg. The vectorcardiogram reveals type 2 right ventricular hypertrophy. The electrocardiogram is characterized by rsR' in V3R and notched R wave in V1. There is a prominent R in V5, and a prominent S in V2.

Only two of these patients was right ventricular systolic pressure below 100 mm. Hg. Six per cent of the patients with pressures less than 100 mm. Hg had P-wave enlargement as contrasted to 70 per cent of those with pressures above 100 mm. Hg. Therefore, P-wave enlargement was more frequent in patients with the highest right ventricular systolic pressures. This is in accord with the observation of Cayler and associates,15 that P-wave enlargement was found in 25 per cent of patients whose right ventricular pressure...
T. S., age 3. Isolated pulmonary stenosis. Right ventricular pressure 140 mm. Hg. Pulmonary artery pressure 15 mm. Hg. The vectorcardiogram reveals type 3 right ventricular hypertrophy. The initial portion of the QRS sE loop in the horizontal plane is inscribed directly anteriorly. The electrocardiogram reveals a tall R in V₃R and Rs in V₁. The T wave is upright in the right precordial leads.

Figure 6
E. S., age 32. Pulmonary stenosis and ventricular septal defect with a right-to-left shunt. The right ventricular systolic pressure is 120 mm. Hg. Pulmonary artery pressure 20 mm. Hg. The vectorcardiogram reveals type 3 right ventricular hypertrophy. The initial portion of the QRS sE loop is convex to the left. The T sE loop is inscribed opposite the QRS sE loop in the horizontal plane. The electrocardiogram reveals marked right axis deviation, qRs in V₃R and V₁, and a prominent S wave in V₃ and V₆. (See next page)
Figure 6 (See legend opposite page)
F. H., age 21. Isolated pulmonary stenosis. Right ventricular systolic pressure 80 mm. Hg. Pulmonary artery systolic pressure 15 mm. Hg. The initial portion of the QRS sE loop extends considerably to the left. This is classified as type S right ventricular hypertrophy because the major portion of the QRS sE loop is displaced to the right. The electrocardiogram reveals a notched R wave in V₃R and V₁, and a prominent S wave in V₅ and V₆.

Figure 7

L. S., age 4. Pulmonary stenosis and ventricular septal defect with a left-to-right shunt. Right ventricular systolic pressure 90 mm. Hg. Pulmonary artery systolic pressure 36 mm. Hg. The vectorcardiogram reveals combined ventricular hypertrophy, type A. The three sets of vectorcardiograms were taken in sequence and showed respiratory changes in the configuration of the QRS sE loop. The electrocardiogram reveals atrial enlargement, RS in V₃R and V₁, and upright T waves in V₃R and V₁. (See next page)
Figure 8 (See legend opposite page)
Table 5

Vectorcardiographic Diagnosis and Right Ventricular Pressure

<table>
<thead>
<tr>
<th>VCG type</th>
<th>Number of patients</th>
<th>Average RV systolic pressure (mm. Hg)</th>
<th>Range of RV systolic pressure (mm. Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>5</td>
<td>41</td>
<td>35 to 50</td>
</tr>
<tr>
<td>RVH-type 1</td>
<td>12</td>
<td>52</td>
<td>40 to 80</td>
</tr>
<tr>
<td>RVH-type 2</td>
<td>7</td>
<td>78</td>
<td>40 to 130</td>
</tr>
<tr>
<td>RVH-type 3</td>
<td>60*</td>
<td>107</td>
<td>45 to 200</td>
</tr>
<tr>
<td>TCD</td>
<td>1</td>
<td>35</td>
<td></td>
</tr>
<tr>
<td>LVH</td>
<td>2</td>
<td>82</td>
<td>65 to 100</td>
</tr>
<tr>
<td>CVH</td>
<td>12</td>
<td>73</td>
<td>35 to 120</td>
</tr>
<tr>
<td>W-P-W</td>
<td>1</td>
<td>50</td>
<td></td>
</tr>
</tbody>
</table>

*One record revealed diffuse conduction delay.

was less than 100 mm. Hg and in 59 per cent of patients whose right ventricular pressure exceeded 100 mm. Hg. However, DePasquale and Burch17 found no relationship between the level of right ventricular pressure and the incidence of P-wave enlargement.

The frontal QRS axis was between +60° and −150° with two thirds lying between +90° and +150°. Although complete right bundle-branch block has been reported38 widening of the QRS interval is rare in isolated pulmonary stenosis. The QRS duration was 0.11 second in two patients, 0.10 second in one, and less than 0.10 second in 42 patients.

The incidence of rsR' in V1 was 11 per cent in the present series. In contrast a rsR' pattern in V1 was reported in 60 to 90 per cent of patients with atrial septal defects. Increased QRS duration also occurs more frequently in patients with atrial septal defect. Approximately 40 per cent of our 70 patients with atrial septal defect had a QRS duration of 0.10 second or more.27

Cabrera and Monroy89 explained the variation in the frequency of right bundle-branch block on the basis of systolic overload in pulmonary stenosis and the diastolic overload in atrial septal defect.

The precise mechanism responsible for these differences remains obscure. There is a significant number of patients with pulmonary stenosis whose electrocardiograms are characterized by rsR' in V1, whereas patients with atrial septal defect may have R or qR in V1 in the presence of high right ventricular systolic pressure. It is well known that following successful surgical repair, the QRS configuration in V1 may change from R to rsR' in pulmonary stenosis.12 Nevertheless, rsR' is more frequently seen in V1 in instances of atrial septal defect.

Reports differ as to the relationship between the level of right ventricular pressure and the configuration of the QRS complex, the height of R or R', and the R/S ratio in V1 in isolated pulmonary stenosis.

Although exceptions exist, our data show that there is fairly good correlation between the height of R in V1 and the level of the right ventricular systolic pressure (fig. 2A). The average right ventricular pressure was lowest among those patients whose electrocardiograms revealed an rS configuration in V1. The average right ventricular systolic pressure in patients with electrocardiograms characterized by qR or Rs was significantly higher than those with rsR' in V1.

The average right ventricular systolic pressure in those patients whose frontal QRS axis was +90° to −91° was significantly higher than those with a QRS axis of 0° to +90°.

Vectorcardiograms are useful for the detection of right ventricular hypertrophy. In four patients with normal vectorcardiograms, the right ventricular pressure was only slightly elevated. Those showing evidence of right ventricular hypertrophy were arbitrarily classified into three types. Type 3, right ven-

Figure 9

M. H., age 10. Isolated pulmonary stenosis. Right ventricular systolic pressure 40 mm. Hg. Pulmonary artery pressure 20 mm. Hg. The electrocardiogram reveals terminal conduction delay of the right bundle-branch block type. The electrocardiogram is characterized by a broad S wave in lead I, aVL, V5, and V6. There is rsR' in V6 and V1. QRS duration: 0.11 second. (See next page)
Figure 9 (See legend opposite page)
ventricular hypertrophy was accompanied by the highest right ventricular systolic pressure. Twenty-one patients manifested this type of vectorcardiogram. The average right ventricular systolic pressure of patients with type 2 right ventricular hypertrophy was slightly higher than those showing type 1 right ventricular hypertrophy. Essentially similar findings were obtained by Bilger in his analysis of vectorcardiograms recorded in a variety of clinical entities. However, no significant difference in the right ventricular systolic pressure was seen between type 1 and type 2 right ventricular hypertrophy in the series of patients with atrial septal defect which we had previously reported.

The vectorcardiograms of six patients were classified as combined ventricular hypertrophy type B. Three of these patients also had prominent S waves in the right precordial leads or prominent R waves in the left precordial leads. The right ventricular systolic pressures of these six patients ranged from 40 to 90 mm. Hg and averaged 62 mm. Hg. This type of vectorcardiogram may represent both combined ventricular hypertrophy or mild right ventricular hypertrophy. There was one 29-year-old patient with posterior displacement of the QRS sE loop classified as left ventricular hypertrophy. Cardiac catheterization revealed pressures of 70 mm. Hg in the right ventricle and 15 mm. Hg in the main pulmonary artery. At operation, the lesion was isolated valvular pulmonary stenosis. He also had extensive pulmonary emphysema. A similar type of vectorcardiographic change was reported by Caelho and associates in nine of 38 patients with cor pulmonale. They regard this particular type of orientation of the QRS sE loop in cor pulmonale as indicative of right ventricular hypertrophy. Our findings in cor pulmonale are similar although it is difficult to explain why right ventricular hypertrophy shows such a peculiar QRS sE loop. Only one patient had distinct terminal conduction delay of the right bundle-branch block type (fig. 9). The QRS duration in this patient was 0.11 second. No evidence of con-

duction delay was found in two patients with a QRS duration of 0.11 and 0.10 second. Since slight degrees of right bundle-branch block are known to cause alteration of the QRS sE loop without terminal conduction delay it is impossible to rule out minimal degrees of right bundle-branch block.

**Pulmonary Stenosis with Interatrial Communication**

In the presence of pulmonary stenosis, a right-to-left or left-to-right shunt may occur at the atrial level. Most instances of a right-to-left shunt have been attributed to a patent foramen ovale. However, the shunt may be a true atrial septal defect.

During the procedure of cardiac catheterization, a number of cases of left-to-right interatrial shunts associated with pulmonary stenosis have been encountered. The left-to-right shunt is generally considered to be through true atrial septal defects.

When an atrial septal defect is associated with pulmonary stenosis, it is usually of the secundum type. However, instances of endocardial cushion defect with pulmonary stenosis and a right-to-left shunt have been reported by several authors. Valvular pulmonary stenosis was present in each instance.

The electrocardiogram in pulmonary stenosis associated with atrial septal defects with a left-to-right shunt has been reported to be similar to the uncomplicated atrial septal defect. When the right ventricular pressure is high, the electrocardiogram shows the characteristics of both.

Burch and DePasquale stated that in one patient with pulmonary stenosis and an atrial septal defect with right ventricular pressure of 105 mm. Hg, the QRS configuration was qR in V1. In addition, there was a wide S wave in lead I, which they regard as a fairly frequent finding in atrial septal defect, but not in pulmonary stenosis.

In many reported instances, the diagnosis of pulmonary stenosis and left-to-right interatrial shunt was made by cardiac catheterization, although the actual presence of pulmonary stenosis was often not confirmed. Especially when the pressure gradient be-
tween the right ventricle and pulmonary artery is small, the pressure gradient may not be due to anatomic stenosis. Rudolph and associates\textsuperscript{18} described a patient with an atrioventricular canal and a pressure gradient across the pulmonary valve of 33 mm. Hg (62 mm. Hg in the right ventricle and 29 mm. Hg in the main pulmonary artery). Neither valvular nor infundibular pulmonary stenosis was found but the pulmonary artery was markedly dilated. Even though there was a significant pressure gradient, the systolic pressure in the main pulmonary artery was well within normal limits.

In the present series we have arbitrarily excluded those patients with pressure gradients across the pulmonary valve of less than 20 mm. Hg. Campbell and associates\textsuperscript{17} have suggested that a pressure gradient greater than 20 mm. Hg between the right ventricle and the pulmonary artery indicates significant pulmonary stenosis in the presence of an atrial septal defect. Similar criteria have been used by Kjellberg and associates.\textsuperscript{18} There were 21 patients with pulmonary stenosis and interatrial communications. Six had left-to-right, 10 had right-to-left, and four had bidirectional shunts. In one patient the catheter was apparently introduced into the left atrium through a small patent foramen ovale, since there was no evidence of a significant interatrial shunt.

The degree of right ventricular hypertrophy by electrocardiographic and vectocardiographic criteria and the level of right ventricular pressure were less among those patients with left-to-right shunts as compared with those who had either right-to-left or bidirectional shunts.

Three patients with rS patterns in V\textsubscript{1} and the only one patient with a normal vectocardiogram in this group, had left-to-right shunts. The right ventricular systolic pressure ranged from 45 to 65 mm. Hg among those who had left-to-right shunts. It ranged from 50 to 180 mm. Hg with the majority above 100 mm. Hg among those who had right-to-left or bidirectional shunts. The electrocardiogram and the vectocardiogram of these patients generally revealed evidence of marked right ventricular hypertrophy. However, the relationship of the height of R in V\textsubscript{1} and the level of the right ventricular pressure was not significant (fig. 2B). In one patient, age 62, the right ventricular pressure was 165 mm. Hg. The configuration of the QRS complex was qr in V\textsubscript{1} through V\textsubscript{3}. The height of r in V\textsubscript{1} was 2 mm. There was right axis deviation, and the R/S ratio in V\textsubscript{6} was less than 1. There was additional evidence of anterosetal myocardial infarction, which may explain the low R in the right precordial leads.

There was no significant difference in the QRS duration or in the incidence of P-wave enlargement between the group with left-to-right shunts and the group with right-to-left or bidirectional shunts. However, the groups are too small to permit any definite conclusion. One patient with the Wolff-Parkinson-White syndrome had a right-to-left shunt, and another patient, age 62, with a left-to-right shunt had atrial fibrillation. These were the only two patients who did not have normal sinus rhythm among the entire series of 100 patients.

In the present series, the average right ventricular systolic pressure recorded in pulmonary stenosis combined with interatrial communication was significantly higher than in those who had isolated pulmonary stenosis. The majority of patients in the former group had severe pulmonary stenosis with right-to-left or bidirectional shunt.

In two of 21 patients in this group, the QRS duration was 0.12 second. In every other instance, it was below 0.10 second. One patient with a QRS duration of 0.12 second had a tracing characteristic of the Wolff-Parkinson-White syndrome. The other vectocardiogram revealed no definite slowing of the QRS inscription.

Electrocardiograms and vectocardiograms were helpful in detecting the presence of right ventricular hypertrophy. However, it was not possible to distinguish between patients with
isolated pulmonary stenosis and those complicated with a foramen ovale or ostium secondum type atrial septal defect. No patient in this series had an endocardial cushion defect. In those patients with endocardial cushion defect and pulmonary stenosis the electrocardiograms were characterized by left axis deviation and a counterclockwise inscription of the frontal plane QRS vector loop. Preordial leads indicated right ventricular hypertrophy.

Pulmonary Stenosis Associated with Ventricular Septal Defect

This group includes both cyanotic and acyanotic patients. Brotmacher and Campbell discussed the difficulty of knowing whether or not the acyanotic type of tetralogy of Fallot has overriding of the aorta. In one of their patients, acyanotic at the time of death, the necropsy finding could not be distinguished from the classical tetralogy of Fallot. They regard it better to label this group as showing ventricular septal defect with pulmonary stenosis rather than acyanotic tetralogy of Fallot. During the natural course in patients with ventricular septal defects and a large left-to-right shunt, infundibular pulmonary stenosis may develop. The direction of the interventricular shunt may remain left-to-right or may reverse. The exact incidence of this transformation is not known. One of our patients with a large ventricular septal defect developed infundibular pulmonary stenosis during the course of observation, as confirmed by serial cardiac catheterizations and subsequent open-heart surgery.

Among 34 patients in this group, the direction of the shunting through the ventricular septal defect was left-to-right in 12 patients, right-to-left in 12, and bidirectional in 10 patients. The systolic pressure in the right ventricle was generally lower in the group with left-to-right shunt as compared to the groups with right-to-left or bidirectional shunt. The systolic pressure in the pulmonary artery was generally higher in the left-to-right group as compared with the other two groups. In eight of 12 patients who had a left-to-right shunt, the systolic pressure in the pulmonary artery was more than 25 mm. Hg.

Enlargement of the P wave was noted in 14 patients, and its incidence was not related to the direction of shunting. Most patients with a right-to-left or bidirectional shunt had right axis deviation, while in seven of 12 patients with a left-to-right shunt, the QRS axis ranged from +85 to −50°. The height of the R wave in V5 and V6 was more than 25 mm. in four patients with left-to-right shunts, whereas this was not observed in any patients with a right-to-left shunt. Of the 34 patients in this group, the QRS duration was 0.10 second in one patient and 0.12 in another; there was no evidence of slowing in the vectorcardiogram in the former, and there was diffuse slowing in addition to type 3 right ventricular hypertrophy in the latter.

Luna and Crow studied the initial component of the QRS complex in lead V1 and its relationship to the right ventricular/left ventricular systolic pressure ratio in instances of systolic overloading of the right ventricle. Seventy-eight per cent of the tracings showed qR complexes in V1 when the right ventricular pressure was greater than the left ventricular pressure. In the group with balanced pressures in both ventricles 77 per cent had an initial slurring of the up-stroke of the R wave. In the group in which the left ventricular pressure was greater than the right ventricular pressure 75 per cent of the tracings had a clean R wave. They regard these electrocardiographic findings as helpful in differentiating conditions with an open or closed ventricular septum. Although qR in V1 occurs generally in patients who have a severe degree of right ventricular hypertrophy, it does not necessarily indicate that right ventricular pressure is greater than the systemic pressure. On the other hand, the configuration of the QRS may be R or Rs when the pressure in the right ventricle exceeds that of the left ventricle. There was no good relationship between the right ventricular pressure and the amplitude of R in V1 (fig. 2C).

The vectorcardiograms of the patients with
pulmonary stenosis and ventricular septal defects associated with a left-to-right shunt was more variable than the other two groups. One patient had left ventricular hypertrophy, four patients had combined ventricular hypertrophy, three patients had type 3 right ventricular hypertrophy, three patients had type 2 right ventricular hypertrophy, and one patient had type 1 right ventricular hypertrophy. In contrast, all of the 22 patients who had either a right-to-left or a bidirectional shunt had type 3 right ventricular hypertrophy, with three exceptions. Two of the three patients had combined ventricular hypertrophy and the remaining patient had type 2 right ventricular hypertrophy.

Thus, electrocardiographic or vectorcardiographic evidence of left ventricular hypertrophy or combined ventricular hypertrophy will favor the presence of left-to-right shunts in patients with pulmonary stenosis associated with ventricular septal defect.

Summary

The electrocardiographic, vectocardiographic, and right heart catheterization data of 100 patients with isolated or complicated pulmonary stenosis were analyzed.

All but two patients had normal sinus rhythm. One patient, age 62, had atrial fibrillation; the other, age 8, had the Wolff-Parkinson-White syndrome.

In isolated pulmonary stenosis, P-wave enlargement was far more frequently seen in those patients with right ventricular systolic pressures above 100 mm. Hg as compared to those with pressures less than 100 mm. Hg. This relationship was not observed in patients with complicated pulmonary stenosis.

The incidence of rsR' in V1, QRS widening, and the vectorcardiographic evidence of conduction delay was significantly lower in pulmonary stenosis, isolated or complicated, when compared to atrial septal defects alone. There was one instance of terminal conduction delay of the right bundle-branch block type and one diffuse slowing of the QRS sF loop in the present series.

The correlation between the amplitude of the R wave in V1 and right ventricular systolic pressure was better in the group of isolated pulmonary stenosis as compared to the groups complicated by interatrial communications or ventricular septal defects.

The average right ventricular systolic pressures in patients with type 3 right ventricular hypertrophy was significantly higher than those with either type 1 or type 2 right ventricular hypertrophy on the basis of vectorcardiographic criteria discussed. In the presence of pulmonary stenosis, the electrocardiographic and vectorcardiographic evidence of left ventricular hypertrophy or combined ventricular hypertrophy suggests coexisting lesions such as ventricular septal defect with left-to-right shunts. However, it was not possible to differentiate between isolated pulmonary stenosis and pulmonary stenosis complicated by interatrial communication by either electrocardiograms or vectorcardiograms.

The vectorcardiographic features of combined ventricular hypertrophy are discussed.

Acknowledgment

We wish to express our appreciation to Dr. Robert T. Singleton, for his assistance in the cardiac catheterizations, and to Dr. R. Adams Cowley, who performed the cardiac surgery.

References

34. Sokolow, M., AND Lyon, T. P.: The ventricular complex in right ventricular hypertrophy as

SCHERLIS, KOENKER, LEE

Circulation, Volume XXVIII, August, 1962
PULMONARY STENOSIS


The Effects of Animal Electricity on Muscular Motion

The effects of stormy atmospheric electricity having been tested, my heart burned with desire to test also the power of peaceful, everyday electricity.

What more fitting, what more certain, than that it should be demonstrated that animal electricity is diffused to contiguous bodies by the nerves, and, not otherwise than common and ordinary electricity, is accustomed to be arrested by insulating and dispersed by conducting substances? These are the things which we have ascertained by experiment.—Luigi Galvani. Commentary on the Effect of Electricity on Muscular Motion. Translated by Robert Moncaville Green, M.D. Cambridge, Massachusetts, Elizabeth Licht, Publisher, 1953, p. 40.
Pulmonary Stenosis: Electrocardiographic, Vectorcardiographic, and Catheterization Data

LEONARD SCHERLIS, ROLF J. KOENKER and YU-CHEN LEE

Circulation. 1963;28:288-305
doi: 10.1161/01.CIR.28.2.288
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1963 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/28/2/288

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/