The Electrocardiogram and Ventricular Gradient in Isolated Congenital Pulmonary Stenosis

By Nicholas DePasquale, M.D., and G. E. Burch, M.D.

A SPECTS of the electrocardiogram in isolated congenital pulmonary stenosis have been described previously by others.1-6 This report is concerned with a study of the electrocardiograms and ventricular gradients of 41 patients from the Charity Hospital and Tulane Medical School at New Orleans, Louisiana, who had proved isolated congenital pulmonary stenosis. The diagnoses for the 41 patients were established by cardiac catheterization; 10 were confirmed at surgery and 2 at autopsy.

Methods and Materials

The 41 patients varied in age from 3 to 38 years, the mean being 14 years. Their distribution according to age, sex, and race is shown in Table 1. The patients were studied by cardiac catheterization and by conventional clinical and electrocardiographic methods. The electrocardiograms were recorded within 2 or 3 days of cardiac catheterization.

Results

The results are summarized in tables 2 to 4 and figures 1 to 8.

There was electrocardiographic evidence of right ventricular hypertrophy in all but 2 patients and right deviation of the electrical axis of the QRS complex in all patients. Typical examples of 4 types of patterns found are shown in figure 1. In general, however, in all of the electrocardiograms the ratio of the amplitude of the R wave to that of the S wave in lead V1 was directly related to the pressure recorded in the right ventricle (figs. 2 and 7). The S wave was large and the R wave small in leads recorded to the left of the transition zone, e.g., V5 and V6 (fig. 1). The duration of the QRS complex was not affected by age nor by increase in right ventricular pressure.

Table 1

<table>
<thead>
<tr>
<th>Age</th>
<th>Sex</th>
<th>Race</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-10</td>
<td>11-20</td>
<td>21-30</td>
</tr>
<tr>
<td>21</td>
<td>13</td>
<td>4</td>
</tr>
</tbody>
</table>

The P wave tended to be prominent in leads II, III, and V1 but its magnitude showed no significant relationship to the pressure recorded in the right ventricle (fig. 3), the correlation coefficient being 0.12.

\[ \hat{\alpha} \text{QRS was oriented to the right in the frontal plane (figs. 4, 5, and table 2). The degree of orientation of } \hat{\alpha} \text{QRS to the right in the frontal plane varied directly with the right ventricular systolic pressure (figs. 6 and 7).} \]

\[ \hat{\alpha} \text{T was oriented more to the left in the frontal plane than } \hat{\alpha} \text{QRS (fig. 4, table 2). The angle between } \hat{\alpha} \text{QRS and } \hat{\alpha} \text{T was greater than normal in 28 of the 41 patients (68 per cent) due to rightward deviation of } \hat{\alpha} \text{QRS without an associated change in } \hat{\alpha} \text{T, or to rotation of } \hat{\alpha} \text{T to the left (fig. 4).} \]

\[ \hat{\alpha} \text{ had a mean direction in the frontal plane of +65° and a mean magnitude of 54 } \mu \text{v.s., (fig. 4, table 2). The angle between } \hat{\alpha} \text{QRS and } \hat{\alpha} \text{ was greater than normal in 26 patients (63 per cent) (table 3). } \hat{\alpha} \text{QRS was located to the right of } \hat{\alpha} \text{ in 38 of the 41 patients (93 per cent) and to the left of } \hat{\alpha} \text{ in 3 patients (7 per cent).} \]

Surgical reduction of the pulmonary stenosis resulted in migration of \( \hat{\alpha} \text{QRS, } \hat{\alpha} \text{T, and } \hat{\alpha} \text{ toward normal locations and magnitudes in the frontal plane projection. The only exception was in a patient in whom the right ventricular pressure failed to decrease after surgery (patient 6, table 4). } \hat{\alpha} \text{QRS rotated to the left, } \hat{\alpha} \text{T to the right, and } \hat{\alpha} \text{ to a more normal direction with respect to } \hat{\alpha} \text{QRS and } \hat{\alpha} \text{T (table 4, fig. 8). The mean magnitude of } \hat{\alpha} \text{QRS de-}
Figure 1

Four types of electrocardiographic patterns in 41 cases of isolated pulmonary stenosis.
increased, that of $\hat{A}$ increased, and that of $A_T$ remained essentially the same after surgery.

In general, the electrocardiographic patterns found in this congenital anomaly tended to be of 4 types (fig. 1):

**Type A**

This type of electrocardiogram had a small to absent R wave and a deep S wave in lead I, and a high R wave and a small to absent S wave in lead V1, and was obtained from patients in whom the right ventricular systolic pressure was greater than 100 mm. of mercury.

**Type B**

This electrocardiographic pattern was obtained when the right ventricular systolic pressure was between 75 and 100 mm. of mercury. The R wave in lead I was more prominent in type B than in type A but was of less magnitude than the S wave in lead I, whereas the R wave in V1 was greater in magnitude than the S wave although the S wave in V1 was moderately deep.

**Type C**

When the right ventricular systolic pressure was less than 75 mm. of mercury, an electrocardiogram of type C was obtained in which the R wave in lead I was equal to or greater in magnitude than the S wave in lead I and the magnitude of the S wave in V1 was equal in magnitude to the R wave in V1.

**Type D**

This type of tracing was of normal configuration. Two of the 41 patients had a normal electrocardiogram even though the right ventricular systolic pressure was 75 and 105 mm. of mercury, respectively.
### Table 4

Electrocardiographic Data before and after Surgical Reduction of Pulmonary Stenosis in Ten Patients

<table>
<thead>
<tr>
<th>Patient number</th>
<th>Months after surgery</th>
<th>Height of R in V1 (mm.)</th>
<th>Height of P in, or PII (mm.)</th>
<th>Location Magnitude µ v.s.</th>
<th>Location Magnitude µ v.s.</th>
<th>Location Magnitude µ v.s.</th>
<th>RV systolic pressure mm. Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>15</td>
<td>15</td>
<td>1</td>
<td>1.0</td>
<td>2.0</td>
<td>117</td>
<td>84</td>
</tr>
<tr>
<td>2</td>
<td>25</td>
<td>10</td>
<td>2</td>
<td>2.0</td>
<td>2.0</td>
<td>155</td>
<td>130</td>
</tr>
<tr>
<td>3</td>
<td>9</td>
<td>5.5</td>
<td>3</td>
<td>5.5</td>
<td>3.0</td>
<td>193</td>
<td>-75</td>
</tr>
<tr>
<td>4</td>
<td>16</td>
<td>16</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
<td>140</td>
<td>101</td>
</tr>
<tr>
<td>5</td>
<td>16</td>
<td>16</td>
<td>2.3</td>
<td>2.3</td>
<td>2.0</td>
<td>125</td>
<td>92</td>
</tr>
<tr>
<td>6</td>
<td>22</td>
<td>7</td>
<td>2.0</td>
<td>1.8</td>
<td>2.0</td>
<td>103</td>
<td>25</td>
</tr>
<tr>
<td>7</td>
<td>22</td>
<td>25</td>
<td>1.0</td>
<td>1.8</td>
<td>1.0</td>
<td>168</td>
<td>69</td>
</tr>
<tr>
<td>8</td>
<td>15</td>
<td>4.1</td>
<td>1.1</td>
<td>2.0</td>
<td>1.1</td>
<td>111</td>
<td>82</td>
</tr>
<tr>
<td>9</td>
<td>7</td>
<td>7</td>
<td>0.7</td>
<td>1.0</td>
<td>0.7</td>
<td>129</td>
<td>72</td>
</tr>
<tr>
<td>10</td>
<td>16</td>
<td>15</td>
<td>2.2</td>
<td>4.0</td>
<td>2.2</td>
<td>180</td>
<td>85</td>
</tr>
<tr>
<td>Mean</td>
<td>16</td>
<td>11</td>
<td>1.9</td>
<td>2.0</td>
<td>1.9</td>
<td>+142</td>
<td>134</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Location Magnitude µ v.s.</th>
<th>Location Magnitude µ v.s.</th>
<th>Location Magnitude µ v.s.</th>
<th>RV systolic pressure mm. Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>110</td>
<td>14</td>
<td>70</td>
<td>46</td>
</tr>
<tr>
<td>120</td>
<td>24</td>
<td>0</td>
<td>28</td>
</tr>
<tr>
<td>102</td>
<td>8</td>
<td>-102</td>
<td>8</td>
</tr>
<tr>
<td>63</td>
<td>14</td>
<td>75</td>
<td>60</td>
</tr>
<tr>
<td>67</td>
<td>42</td>
<td>65</td>
<td>40</td>
</tr>
<tr>
<td>160</td>
<td>9</td>
<td>5</td>
<td>25</td>
</tr>
<tr>
<td>63</td>
<td>11</td>
<td>40</td>
<td>40</td>
</tr>
<tr>
<td>60</td>
<td>28</td>
<td>60</td>
<td>60</td>
</tr>
<tr>
<td>5</td>
<td>32</td>
<td>5</td>
<td>25</td>
</tr>
<tr>
<td>41</td>
<td>23</td>
<td>70</td>
<td>59</td>
</tr>
<tr>
<td>39</td>
<td>21</td>
<td>+35</td>
<td>38</td>
</tr>
<tr>
<td>29</td>
<td>21</td>
<td>+70</td>
<td>51</td>
</tr>
</tbody>
</table>

**Discussion**

Of the 21 patients whose electrocardiograms were of type A, only 1 had a right ventricular systolic pressure of 133 mm. of mercury and an electrocardiographic pattern of type C, while another patient had a right ventricular systolic pressure of 69 mm. of mercury and an electrocardiogram of type A.

One patient had a right ventricular systolic pressure of 135 mm. of mercury and an electrocardiographic pattern of type D. Only 1 had a right ventricular systolic pressure of less than 100 mm. of mercury and an electrocardiogram of type A.

Relationship of right ventricular systolic pressure to the R/S ratio in lead V6. When a diphasic R wave was present the highest wave was selected.
68 mm. of mercury and complete right bundle-branch block. This was the only instance of complete bundle-branch block in this series.

The ventricular gradient of Wilson and associates, an expression of the variations in the duration of the excited state, has been studied by us in patients with congenital and acquired heart disease as well as in normal children. Ashman analyzed the electrocardiograms of 164 normal adults and found the mean $\Delta_{\text{QRS}}$ to have a direction of $+41.7^\circ$ (range, $-21.5^\circ$ to $+104.9^\circ$) and a mean magnitude of 21.8 $\mu$V.s. (40.6 to 3.1 $\mu$V.s.) in the frontal plane projection, whereas $\delta$ had a mean direction of $+39.2^\circ$ (+72.2$^\circ$ to +2.2$^\circ$) and a mean magnitude of 46.2 $\mu$V.s. (78.9 to 13.5 $\mu$V.s.). In 77 children (2 to 14 years of age) he found $\Delta_{\text{QRS}}$ to have a mean direction of $+61.1^\circ$ ($+102^\circ$ to $+20.1^\circ$) and a mean magnitude of 16.6 $\mu$V.s. in the frontal plane projection, whereas in 78 children the $\delta$ had a mean direction of $48.0^\circ$ ($+73.1^\circ$ to $+22.9^\circ$) and a mean magnitude of 46.6 $\mu$V.s. (72.2 to 16.6 $\mu$V.s.). In our study of the ventricular gradients of 172 normal infants and children from birth to 16 years of age, we found that, except for a decrease in the mean magnitude of $\Delta_{\text{QRS}}$ and to a lesser extent of $\delta$, during the first 3 years of life and a shift of $\Delta_{\text{QRS}}$ to the right during the first year of life, the values for children were the same as Ashman’s values for adults.

There were abnormally wide angles ($>30^\circ$) between $\Delta_{\text{QRS}}$ and $\delta$ in the frontal plane projection in 26 patients (63 per cent) (table 3). In addition, the $\Delta_{\text{QRS}}$ in 29 patients (71 per cent) was deviated more to the right than the normal (fig. 4), and $\delta$ was abnormal in direction in 22 patients (54 per cent) (fig. 4). The magnitude of $\Delta_{\text{QRS}}$ was abnormal in 9 patients (22 per cent) and the magnitude of $\delta$ was abnormal in 4 patients (10 per cent). The mean $\delta$: $\Delta_{\text{QRS}}$ ratio was 2.7; however, in 9 patients the ratio was less than 1.0. In 4 other patients the ratio was greater than 3.0 because of a $\delta$ of unusually great magnitude. Normally, the magnitude of $\delta$ is essentially twice the magnitude of $\Delta_{\text{QRS}}$.

The abnormal ventricular gradient indicates functional electrical changes in the myocardium other than those due to right ventricular hypertrophy. The ventricular gradient was abnormal in 26 patients (63 per cent), in 22 because of its direction and in 4 because of its magnitude, whereas 39 patients (95 per cent) had electrocardiograms that were not considered to indicate myocardial disease other than ventricular hypertrophy. These
changes apparently are reversible in large part as is indicated by the reversion of the gradient toward the normal following surgery (table 4, fig. 8).

The degree of right ventricular hypertrophy was dependent upon the magnitude of the right ventricular pressure, a finding already indicated by others.6 The age of the patient was not found to be so important a factor in the production of an abnormal electrocardiogram in congenital pulmonary stenosis as it was in congenital atrial septal defect.13 Nevertheless, the duration of hypertension in the right ventricle must be a factor of importance in determining the degree of right ventricular hypertrophy. The reason for the absence of a significant relationship of right ventricular hypertrophy to age of the patient is not known.

An interesting aspect of the electrocardiographic pattern in pure pulmonary stenosis was the failure of the QRS complex to become abnormally prolonged with age. Atrial septal defect, on the other hand, is characteristically associated with progressive widening of the QRS complex.13 The mechanisms responsible for this difference are not clear. In both types of cardiac defects the work of the right ventricle is increased but in atrial septal defect the increase in work is due to an increase in volume output of the heart, whereas in pure pulmonary stenosis the increase in work is due to increase in right ventricular pressure. Anatomically, there is predominantly hypertrophy of the crista supraventricularis in atrial septal defect, whereas in pure pulmonary stenosis there is generalized hypertrophy of the right ventricle. Cabrera and Monroy14 have already discussed this problem. The precise mechanisms for these differences in anatomic changes remain to be explained. Regardless of the mechanism, these observations demonstrate again the significant relationship of the electrocardiogram to the normal and abnormal functional and anatomic state of the heart.

With the accumulation of more detailed data in future years the role of the electrocardiogram in the clinical diagnosis of congenital cardiac defects should be improved.
CONGENITAL PULMONARY STENOSIS

Summary

The electrocardiogram and ventricular gradient were studied in 41 patients with isolated congenital pulmonary stenosis. The ventricular gradient was abnormal in 26 of the 41 patients (63 per cent). The $A_{QR}$ migrated to the right in the frontal plane and $A_T$ migrated to the left. Following surgical reduction of the stenosis these vectors rotated rapidly toward the normal position.

The electrocardiographic pattern in isolated congenital pulmonary stenosis tended to be of 4 types that were generally related to the right ventricular systolic pressure. The electrocardiographic changes associated with this defect, in which the work of the right ventricle was increased because of right ventricular hypertrophy, were different from those associated with atrial septal defect in which right ventricular work was increased because of high volume output. The duration of the QRS complex was not prolonged in the electrocardiograms of patients with pulmonary stenosis, whereas it was characteristically prolonged in those with atrial septal defect.

Summario in Interlingua

Le electrocardiogramma e le gradiente ventricular esesva studiata in 41 patientes con isolate congenita stenosis pulmonar. Le gradiente ventricular eseva anormal in 26 del 41 (63 pro cento). $A_{QR}$ migrava verso le dextera in le plano frontal, e $A_T$ migrava verso le sinistra. Post reduction chirurgic del stenosis, iste vectores rotava rapidamente verso lor posizioni normal.

Le patrono electrocardiographic in isolate congenita stenosis pulmonar tendeva a gruppar se in un de 4 typos que eseva generalmente relateda al tension systolic dextero-ventricular. Le alterationes electrocardiographic associate con iste defecto, in que le labor del ventriculo dextere eseva augmentata a causa del presistantia de hypertension dextero-ventricular diferera ab le alterationes electrocardiographic associate con defecto atrio-septal in que le labor del ventriculo dextere eseva augmentata a causa de un alte volumine del rendimento. Le duration del complexo QRS non eseva prolongate in le electrocardiogrammas de patientes con stenosis pulmonar, durante que illo eseva characteristicamente prolongate in patientes con defecto atrio-septal.

References

1. Currens, J. H., Kinney, T. D., and White, P. D.: Pulmonary stenosis with intact interven-

9. —, and —: The electrocardiogram and ventricular gradient in ventricular septal defect. To be published.
10. —, and —: A study of the ventricular gradient in normal infants and children. To be published.
The Electrocardiogram and Ventricular Gradient in Isolated Congenital Pulmonary Stenosis

NICHOLAS DEPASQUALE and G. E. BURCH

doi: 10.1161/01.CIR.21.2.181

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1960 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/21/2/181

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/