Precordial Lead Patterns in Right Ventricular Hypertension

By G. P. Human, M.B., Ch.B., M.Med.

Current criteria for the electrocardiographic diagnosis of right ventricular hypertrophy\(^1\) depend primarily upon abnormal axis deviation and changes in the right precordial leads (V\(_1\), V\(_4R\)). The R/S ratio in these leads has proved to be of great value in the recognition of right ventricular hypertrophy. Various patterns (qR, rS, rsR', Rs) are encountered and many theories have been put forward to explain the exact mechanisms producing them. Attempts have also been made to correlate specific electrocardiographic patterns with specific hemodynamic changes. The concept of systolic and diastolic overload patterns introduced by Cabrera and Monroy\(^4\) correlated resistance to right ventricular outflow (systolic overloading) with a high R wave in V\(_1\), while dilatation of the ventricle (diastolic overload) was associated with the rsR' pattern. Although this is a useful physiologic concept, many authors have shown that each of these patterns is found in both hemodynamic situations and these attempts at correlation are not always successful.

In this paper our findings in cases with right ventricular hypertension are presented with an attempt to correlate the right precordial electrocardiogram with the pressure in the right ventricle. The significance of the various patterns found will be discussed.

Method

The electrocardiograms of 80 patients (48 European, 32 Bantu) presenting clinical or roentgenologic evidence of right ventricular hypertrophy and with right ventricular systolic pressures greater than 50 mm. Hg, were studied. A 12-lead electrocardiogram was obtained and the ventricular pressure was measured by catheter in each case. The cases were divided into groups according to etiology. All forms of mitral valve lesions (stenosis, stenosis with regurgitation, pure regurgitation) were included in the same group. All cases with QRS duration in any lead longer than 0.10 second or with signs of myocardial ischemia were excluded from this series. The mean frontal plane axis of each case was measured. The accepted criteria for the diagnosis of right ventricular hypertrophy were used in diagnosis with special attention to the eight cases that were under 5 years of age.\(^5\)

The ventricular complexes in V\(_1\) were studied, measured, and grouped according to pattern. All cases with rR, notched R, or pure R waves were grouped together. In cases with the rsR pattern, the duration of the initial r was carefully measured. Right ventricular pressures were compared with the height of the R or R' wave and with the R/S or R'/S ratio.

Intracavity electrocardiograms were obtained from the right ventricle with an intracardiac electrode. Simultaneous recordings of these and of right and left precordial leads were obtained in selected cases with a Hellige multichannel apparatus.

Results

The age distribution and the mean frontal plane axes of the patients are shown in table 1 and figure 1.

In 64 per cent of the cases the diagnosis of right ventricular hypertrophy could be made on Milnor's criterion\(^6\) of QRS duration less than 0.12 second and a R/S or R'/S ratio in V\(_1\) greater than 1.0 with R or R' greater than 0.5 mv. A further 9 per cent of electrocardiograms had a mean frontal plane axis between +110° and ±180° or between −91° and ±180° as the only indication of right ventricular hypertrophy. The pattern of R > S in V\(_4R\)\(^7\) was of diagnostic aid in six additional adult cases. In a total of 64 cases (80 per cent of the series) the electrocardio-
graphic diagnosis of right ventricular hypotrophy could thus be made.

The incidence of the various patterns in V₁ in the different groups of patients is shown in Table 2. The rsR' pattern was the most frequent finding, occurring in 22 of the studied cases. It was noted that the duration of the initial r was of very short duration; it never exceeded 0.02 second. A qR pattern was found in nine cases, with rR', notched R, or pure R in 21 cases, and Rs in five cases. Eleven electrocardiograms showed a RS pattern, while 12 had a rS pattern. The latter two ventricular patterns were mostly seen in patients with mitral insufficiency, patent ductus arteriosus, or ventricular septal defect, where the left ventricle might also have been enlarged.

No correlation between the height of the R or R' wave or the R/S or R'/S ratio in V₁ and the height of the ventricular pressure could be found (figs. 2 and 3).

The synchronously recorded leads revealed that in cases with a rsR' pattern in V₁ (patient W.P., fig. 4) the R' wave was absent in the intracavity leads. The R wave in left precordial lead V₆ preceded the R wave of the right precordial leads (patients H.N., fig. 5, and M.R., fig. 6). It was also shown that the q wave in V₁ (fig. 6) occurred 0.01 second later than the q wave in V₆.

**Discussion**

The poor correlation between electrocardiographic and anatomic hypertrophy of the heart is a well-known fact. From this study

### Table 1

<table>
<thead>
<tr>
<th>Pattern</th>
<th>rsR'</th>
<th>qR</th>
<th>rS</th>
<th>R</th>
<th>RS</th>
<th>Rs</th>
<th>Total</th>
</tr>
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<tbody>
<tr>
<td>Mitral valve lesions</td>
<td>8</td>
<td>3</td>
<td>4</td>
<td>2</td>
<td>2</td>
<td>-</td>
<td>19</td>
</tr>
<tr>
<td>Atrial septal defect</td>
<td>6</td>
<td>1</td>
<td>2</td>
<td>-</td>
<td>-</td>
<td>1</td>
<td>10</td>
</tr>
<tr>
<td>Ventricular septal defect</td>
<td>1</td>
<td>-</td>
<td>3</td>
<td>6</td>
<td>2</td>
<td>-</td>
<td>12</td>
</tr>
<tr>
<td>Patent ductus arteriosus</td>
<td>2</td>
<td>1</td>
<td>3</td>
<td>-</td>
<td>4</td>
<td>-</td>
<td>10</td>
</tr>
<tr>
<td>Pulmonary stenosis</td>
<td>-</td>
<td>2</td>
<td>-</td>
<td>4</td>
<td>1</td>
<td>-</td>
<td>7</td>
</tr>
<tr>
<td>Pulmonary stenosis and atrial septal defect</td>
<td>2</td>
<td>-</td>
<td>-</td>
<td>5</td>
<td>1</td>
<td>-</td>
<td>8</td>
</tr>
<tr>
<td>Pulmonary stenosis and ventricular septal defect</td>
<td>2</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>1</td>
<td>-</td>
<td>3</td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>1</td>
<td>2</td>
<td>-</td>
<td>4</td>
<td>-</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td>Cor pulmonale</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Total</td>
<td>22</td>
<td>9</td>
<td>12</td>
<td>21</td>
<td>11</td>
<td>5</td>
<td>80</td>
</tr>
<tr>
<td>Per cent of total</td>
<td>28</td>
<td>11</td>
<td>15</td>
<td>26</td>
<td>14</td>
<td>6</td>
<td>100</td>
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</table>

### Table 2

<table>
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<tr>
<th>Age, yr.</th>
<th>European</th>
<th>Bantu</th>
</tr>
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<tbody>
<tr>
<td>0-2</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2-5</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>5-10</td>
<td>10</td>
<td>4</td>
</tr>
<tr>
<td>10-12</td>
<td>9</td>
<td>5</td>
</tr>
<tr>
<td>12-20</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>20-40</td>
<td>11</td>
<td>10</td>
</tr>
<tr>
<td>40-60</td>
<td>7</td>
<td>4</td>
</tr>
</tbody>
</table>

*Figure 1*

The mean QRS vector in the frontal plane in the 80 patients.

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it is clear that this lack of correlation is also present even when the type of ventricular work is considered.

In this series of right ventricular hypertension a confident diagnosis of hypertrophy could be made in 80 per cent. Furthermore, no typical pattern of systolic overloading was found in the right precordial lead. Both R and rsR’ patterns occurred in roughly equal proportions.

The failure of the height of the R wave to correlate with the height of the systolic pressure probably represents a defect in this method. Objection to the correlation of hemodynamic and electric events can be raised on the grounds that the electrocardiogram cannot reflect pressure changes but only the anatomic and conduction alterations that may result from such changes. A more logical approach would be to compare the electrocardiogram with the product of the radius of the ventricle and the pressure within, which gives a closer approximation of myocardial tension (T = P \times R; Laplace’s law). It should also be emphasized that it might be quite possible for a correlation between electrical and mechanical events to exist at some stage of ventricular hypertrophy. It is obviously impossible to examine all patients at that particular phase of their illness.

The large R wave in leads overlying the right ventricle is probably produced by a similar mechanism under all circumstances and irrespective of whether it is part of a qR, rsR’, or pure R pattern. In this respect it is of interest to examine the electrocardio-

Figure 2

*Height of R wave in V₁ plotted against right ventricular pressure.*
grams of our patients H.N. and M.R. showing V₄R, V₁, and V₆ recorded simultaneously. The R wave in the right precordial leads is more or less synchronous with the S wave in V₆, indicating that both are produced by the same forces. The R wave in V₁ occurs later than the R in V₆.

The prominent R waves in the precordial leads of patients with right ventricular hypertension were absent in the intraventricular leads. These facts seem to indicate that the R in right precordial leads is caused by depolarization of the thickened free wall of the right ventricle and that it is preceded by activation of the left ventricle.

The rsR' pattern has now been accepted by many authors as a manifestation of hypertrophy of the right ventricular outflow tract. Myers and also Silver and co-workers distinguish two types of rsR' patterns according to the duration of the initial r.

They attribute rsR' patterns with initial r of less than 0.025 second to hypertrophy of the crista supraventricularis, whereas those with an initial r greater than 0.03 second represent true "incomplete bundle-branch block."

It is interesting to note that the initial r in our series was less than 0.025 second in every case and we believe that this pattern is a reliable indication of hypertrophy of the outflow tract of the right ventricle.

Various explanations have been advanced for the qR morphology in right precordial leads.

Sodi-Pallares stated that the qR complex is a manifestation of right atrial enlargement with the result that the high basal portions of the interventricular septum face the exploring electrode through the dilated atrium. This is in accordance with Silver's view that the qR pattern represents hypertrophy.

Figure 3

R/S ratio in V₁ plotted against right ventricular pressure
of the crista, since this structure belongs to the higher part of the septum.

Goldberger and others proposed that extreme clockwise rotation of the heart about the longitudinal axis will cause the back of the heart and the epicardial surface of the left ventricle to face the right precordial electrode. The q wave would then represent a left ventricular cavity potential and the spread of the stimulus outward through the left ventricular wall would cause the R wave. It has been shown, however, that the q in V₁ is not a septal q and is thus manifestly different from the q in V₆. In the synchronous records published by Morris and Whitaker, the q of qR in V₁ has the same time relationship to the R in V₆ as the S of the rsr’ in V₁ has. They concluded that there is no support for the view that a qR complex in V₁ is derived from potentials developed in the cavity of the left ventricle. Grant’s work also denies the importance of rotation of the heart on the morphology of the precordial complexes. Our patient, M.R., had right ventricular hypertension following mitral valve disease and showed a qV₄R and a qV₆. It is apparent from figure 6 that the right precordial q occurs 0.01 second later than the q in V₆. These observations lend support to the theory (Myers, Fowler) that the initial r of the rsR’ pattern may be lost in the preceding isoelectric line during transmission to the precordium. The rsR’ patterns in V₁ would then be essentially the same as qR patterns, because through loss of

**Figure 5**

Unipolar leads V₁R, V₁, V₆, and standard lead III recorded simultaneously. The R in right precordial leads occurs 0.02 second later than the R in V₆.

Simultaneous recording of unipolar precordial and intraventricular leads showing absence of R’ wave in the latter. Paper speed 50 mm. per second.
hypertrophy is often obscured by left ventricular forces. It is generally accepted that in lesions associated with left ventricular hypertrophy the electrocardiographic signs of right ventricular hypertrophy are masked to such an extent that a normal R/S pattern may occur in right precordial leads. Hypertrophy of the free wall of the right ventricle would have to be of considerable magnitude to outweigh increased left ventricular potentials.

**Summary and Conclusions**

The right precordial lead patterns in 80 patients with right ventricular hypertension were studied. The importance of these leads and of abnormal right axis deviation in the diagnosis of right ventricular hypertrophy is again confirmed. These criteria failed however in 20 per cent of cases.

No correlation was shown between R-voltage and ventricular pressure. Time relationships between different deflections were determined by synchronously recorded intracavity and right or left precordial electrocardiograms.

The incidence of the various QRS patterns revealed that no typical pattern can be attributed to systolic overloading of the right ventricle. The mechanisms causing the different patterns are discussed and the relevant literature is reviewed.

It is concluded that rsR' and qR patterns in right precordial leads are essentially similar and represent hypertrophy of the outflow tract of the right ventricle; while the large R wave is produced by activation of the hypertrophied free wall of the right ventricle.

**Acknowledgment**

I wish to thank Prof. H. W. Snyman for his helpful advice and criticism in the preparation of this paper.

**References**


3. Scott, R. C.: The correlation between the elec-

William Harvey: The Pulmonary Circulation

On one occasion the body of a hanged highwayman was brought to the institute of anatomy in London from the place of execution.

Harvey proceeded as he had planned. Only one blood vessel was allowed free connection with the outside—this was the inferior vena cava—into which he inserted a tube which in turn was attached to a cow’s bladder filled with water. He could press as hard as he liked. The right ventricle and auricle expanded to the point of rupture, but no water trickled through into the left ventricle. Then he tied the ligature from the pulmonary artery: The water flowed freely across the lungs into the pulmonary veins, from there across the left auricle into the left ventricle and out, through the hole he had cut into it.

Harvey never made this public among the “general community of scholars,” but described it in a private letter to a London colleague.—Tibor Dobry, M.D. Discoverers of Blood Circulation. From Aristotle to the Times of Da Vinci and Harvey. New York, Abelard-Schuman, 1963, p. 219.
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