Electrocardiogram and Vectorcardiogram in Right Ventricular Hypertrophy and Right Bundle-Branch Block

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Electrocardiographic and vectorcardiographic studies on normal subjects and patients with heart disease suggest that criteria for the diagnosis of right ventricular hypertrophy can be improved and simplified. This report proposes new criteria for the electrocardiographic diagnosis of right ventricular hypertrophy. These criteria decrease the incidence of "false negative" and "false positive" interpretations, and also make possible the recognition of right ventricular hypertrophy in the presence of the R' – V1 pattern, provided the QRS duration is less than 0.12 second. The findings and limitations of vectorcardiography in right ventricular hypertrophy and "right bundle-branch block" are discussed.

The earliest and most reliable signs of right ventricular hypertrophy are the characteristic changes in the electric field of the heart, which are reflected in the conventional leads of the scalar electrocardiogram. The investigation reported here was undertaken in the hope that present criteria for the electrocardiographic diagnosis of right ventricular hypertrophy1,2 could be improved so as to identify correctly a larger proportion of cases in which right ventricular hypertrophy is present, reduce the number of false positives in normal subjects, and allow interpretation of records that show an R' in lead V1.

Electrocardiographic and vectorcardiographic study of normal persons3 and patients with heart disease showed that the most important and characteristic electric change produced by right ventricular hypertrophy is the shift of the spatial QRS axis rightward and anteriorly. We therefore adopted tentatively the following criteria, which depend only on the QRS complex in lead V1 and the frontal plane QRS axis: QRS duration less than 0.12 second, and either: (1) Mean frontal plane QRS axis between +110° and ±180°, or between −91° and ±180°; or (2) R/S or R'/S ratio in V1 greater than 1.0, with R or R' greater than 0.5 mv. In calculating mean QRS axis for this purpose the method of Carter, Richter, and Green4 was used. The smallest polar angle between the values given is the one that applies.

These criteria were applied to a consecutive series of electrocardiograms recorded on routine hospital patients, to test the number of false positives that would result, and to another series of records from patients with autopsy-confirmed right ventricular hypertrophy, to estimate the incidence of false negatives. Spatial vectorcardiograms were investigated in selected patients, to see what additional information might be provided by this technic.

Methods and Patient Selection

Normal series. This group of 103 persons has been described elsewhere.5 It included 64 males and 39 females, ranging in age from 10 to 70 years. Electrocardiograms on these subjects were recorded on a 4-channel Hathaway S-14C oscillograph,6 with amplifiers constructed in our laboratory.7 Frequency response of this recording system is linear to at least 180 c.p.s., down 5 per cent at 200 c.p.s. and 50 per cent at 275 c.p.s. A time constant of 2.7 seconds determines the low frequency response. Time intervals can be measured accurately to 0.0025 second. Standard bipolar and unipolar limb leads, and chest leads V6s, V4s, and V1 to V4 were recorded. A simultaneous lead II was recorded with all other leads as a timing reference.

Heart Station Series. Electrocardiograms recorded on consecutive patients in the Johns Hopkins Hos-
hospital Heart Station during a period of 18 months were reviewed. Records were selected for further study if they satisfied the criteria previously described for the diagnosis of right ventricular hypertrophy, or if the QRS duration were 0.12 second or more with the pattern of right bundle-branch block in lead V1. All diagnostic information available on 71 patients thus selected was reviewed, including history, physical findings, laboratory tests, roentgenograms, and other pertinent special studies, excluding the electrocardiographic interpretation. Patients were then eliminated from the series if the evidence available was inadequate to make a reasonably certain diagnosis (5 patients), if there was evidence of both coronary disease and a disease that would lead to right ventricular hypertrophy (17 patients), or if they were over the age of 50 years (3 patients), even though no evidence of heart disease was found. Twenty-five patients were thus excluded in an effort to arrive at a group with relatively well-established diagnoses that could be divided without overlapping into normal subjects, patients with cardiac lesions that produce right ventricular hypertrophy, and patients with coronary artery disease. We had planned also to eliminate cases with disease that would lead to isolated left ventricular hypertrophy, but none was found in this group. Some of the rheumatic and congenital cases doubtless had combined right and left ventricular hypertrophy, but the clinical evidence indicated that the burden was predominantly right-sided in all cases. Two of the 18 cases with coronary disease also had arterial hypertension.

The 46 remaining cases are summarized in table 2. One person had no demonstrable heart disease, 18 patients had coronary artery disease (4 with a history of previous myocardial infarction), 17 patients had rheumatic heart disease with predominant mitral stenosis, 9 patients had congenital heart disease, and there was 1 case of chronic cor pulmonale.

Autopsy Series. Autopsy records of the Johns Hopkins Hospital over a period of 33 months were reviewed. Out of 192 autopsies performed during that period, 48 cases over the age of 1 year had definite right ventricular hypertrophy without complicating left ventricular hypertrophy or myocardial disease due to coronary obstruction. Requirements for the anatomic diagnosis of right ventricular hypertrophy were heart weight more than 25 per cent over normal average for body weight and sex, and right ventricular thickness greater than 4 mm. Only those cases that satisfied both requirements were included.

In 32 of these 48 cases, electrocardiograms were taken within 9 months of death that included at least 3 limb leads and 3 precordial leads. In 24 cases electrocardiograms had been recorded less than 1 week before death.

Vectorcardiographic Series. Spatial vectorcardiograms were recorded on a total of 67 patients whose scalar electrocardiograms either met the proposed criteria or showed a QRS duration of 0.12 second or more but otherwise met these criteria. On the same grounds for exclusion described above, 7 of these patients were eliminated because of insufficient evidence to establish a diagnosis, 1 case because of combined coronary disease and right ventricular hypertrophy, and 5 because of age over 50 years, leaving a total of 54 patients.

Vectorcardiograms were recorded with a cubical lead system and apparatus described previously. Scalar records of the x, y, and z components of the spatial vectorcardiogram were used to aid in the interpretation of the vectorcardiographic loops.

Definitions. 1. R/S ratio: the ratio of the amplitude of the tallest single positive deflection in a lead (numerator) to that of the deepest negative deflection in the same lead (denominator). Amplitudes are measured from the P-Q segment.

2. R' : the second of 2 positive components of a QRS complex that are separated by a downward deflection that reaches or crosses the iso-electric line.

3. Complete right bundle-branch block (RBBB): QRS duration 0.12 second or more, with a positive late deflection in lead V1 and a negative late deflection in lead I.

4. Mean QRS axis: mean manifest frontal plane QRS axis calculated from the nomogram of Carter, Richter, and Green, (not by the more accurate method of measuring the area beneath each deflection). This axis is not necessarily identical with the maximal QRS vector of the spatial vector-cardiogram.

5. Frontal plane QRS axis: “Normal” = 0° to +110°, “left” = 0° to -89°, “right” = +111° through 180° to -90°, inclusive.

6. QRS duration: Durations equal to or greater than 0.12 second are termed abnormal.

Results

Electrocardiograms

1. Autopsy Series. The proposed criteria correctly diagnosed 24 of the 32 autopsied cases with right ventricular hypertrophy (table 1). Six of the 24 cases correctly diagnosed by our criteria would have been missed by the criteria of Myers, Klein, and Stoefer or Sokolow and Lyon. Seven of the 8 cases missed by our criteria would also have been missed by theirs. The remaining case had an R - V1 greater than 0.7 mv. and an R/S ratio of 0.3, and would therefore have been diagnosed correctly by the criteria of Sokolow; this same finding, however, is present in many normal persons.

2. Heart Station Series.
marized in table 2. None of the 29 patients whose electrocardiograms fulfilled the proposed criteria was found to have a normal cardiovascular system: 26 had disorders that characteristically produce right ventricular hypertrophy; 3 had coronary artery disease.

TABLE 1.—Results of Proposed Criteria for Diagnosis of Right Ventricular Hypertrophy, in Thirty-two Autopsied Cases

<table>
<thead>
<tr>
<th>Etiologic diagnosis</th>
<th>Congenital</th>
<th>Chronic cor pulmonale</th>
<th>Rheumatic heart disease</th>
<th>Total</th>
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<tbody>
<tr>
<td>Correctly diagnosed as</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>right ventricular</td>
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<tr>
<td>hypertrophy:</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>met axis criteria only...</td>
<td>0</td>
<td>3</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>met V1 criterion only.....</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>met both criteria..........</td>
<td>6</td>
<td>3</td>
<td>5</td>
<td>14</td>
</tr>
<tr>
<td>Total</td>
<td>6</td>
<td>8</td>
<td>10</td>
<td>24</td>
</tr>
<tr>
<td>Not diagnosed by proposed</td>
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<td>4</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>criteria</td>
<td></td>
<td></td>
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<tr>
<td>Total</td>
<td>6</td>
<td>12</td>
<td>14</td>
<td>32</td>
</tr>
</tbody>
</table>

Of the 17 patients with complete RBBB, 1 subject had no heart disease, 15 had coronary disease (3 with previous myocardial infarction), and 1 patient had right ventricular hypertrophy with congenital heart disease (atrial septal defect).

Significance of QRS Duration. The electrocardiographic pattern of complete RBBB was much more frequently due to coronary disease than to right ventricular hypertrophy (table 2). QRS durations of 0.15 second or more were seen only in patients with coronary disease, but this was of little diagnostic help, since the range of QRS durations was wide, the average in the coronary cases being 0.129 second, and QRS duration 0.130 second in the 1 case of right ventricular hypertrophy in the Heart Station series.

Most of the cases that had QRS duration less than 0.120 second and satisfied our criteria had clinical signs of right ventricular hypertrophy (26 of 29 cases, or 90 per cent).

The incidence of “high normal” QRS durations (0.10 to 0.12 second), was about the same

TABLE 2.—Electrocardiographic Data

<table>
<thead>
<tr>
<th>Patient series and diagnosis*</th>
<th>Total number of patients</th>
<th>QRS duration &lt;0.120</th>
<th>QRS duration ≥ 0.120</th>
<th>Mean frontal QRS axis</th>
<th>V1 criteria for RVH</th>
<th>R' - V1 present</th>
<th>R/S &gt; 1</th>
<th>R/S 0.5 mv.</th>
<th>Both</th>
<th>Number of pts.</th>
<th>Mean frontal QRS axis</th>
<th>R' - V1 present</th>
<th>Left</th>
<th>Normal</th>
<th>Right+</th>
<th>Right</th>
<th>Left</th>
<th>Normal</th>
<th>Right+</th>
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<td>0</td>
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<td>0</td>
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<td>0</td>
<td>2</td>
<td>0</td>
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<td>1</td>
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<td>9</td>
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<td>0</td>
<td>6</td>
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<td>Total</td>
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</tr>
</tbody>
</table>

* In this and subsequent tables RVH = right ventricular hypertrophy; Congenital = congenital heart disease; Cor pulm. = chronic cor pulmonale; RHD = rheumatic heart disease.

† The limits used for normal, left, and right axis are given in the text under “Definitions.” “Right +” = +111° to 180°, and “Right −” = −90° to −179° inclusive.
in the cases of right ventricular hypertrophy (4 of 26 patients, or 15 per cent) as in the normal series (19 of 103, or 18 per cent).

**Significance of R or R' Amplitude.** The amplitudes of the latest positive deflection in V1 were not significantly different in the cases of coronary disease, right ventricular hypertrophy, and the normal case, regardless of QRS duration (table 4). The largest positive
deflection found in V1, however, was a 5.5 mv. R' in a case of pure pulmonic stenosis.

**Significance of Mean QRS Axis.** The frontal plane mean QRS axis varied widely, although the highest incidence of right axis deviation was found in the cases of right ventricular hypertrophy, as expected. In 10 of the 29 cases, with QRS duration less than 0.12 second, right axis deviation was present but V1 did not satisfy the proposed criteria.

When the QRS duration was greater than 0.12 second, right axis deviation appeared in 3 of the 15 cases with coronary disease, as well as in the 1 case with right ventricular hypertrophy. The axis was normal in the 1 normal subject in this group of complete RBBB, but was also normal in 10 of the 15 coronary cases.

**Significance of R'—V1.** A recognizable R' (0.1 mv. or larger) was found in 4 of the 103 normal subjects, and had an amplitude of less than 0.3 mv. in each case. In the Heart Station series, with QRS duration less than 0.12 second, an R'—V1 was found in cases of coronary disease and cases of right ventricular hypertrophy, with about the same incidence: 1 out of 3 coronary cases, 11 of 26 right ventricular hypertrophy cases. In the autopsy series, an R'—V1 was present in 6 of 31 cases with QRS duration less than 0.12 second.

An initial Q—V1 was present in 4 of the 14 coronary cases with QRS duration of 0.12 second or more, but not in the normal case or the right ventricular hypertrophy case.

**Table 3.—Relation between QRS Duration and Presence of R'—V1, for QRS Duration Less than 0.12 Second (Heart Station Series)**

<table>
<thead>
<tr>
<th>QRS duration (second)</th>
<th>R'—V1</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Absent</td>
<td>Present</td>
</tr>
<tr>
<td>&lt;0.100</td>
<td>16</td>
<td>8</td>
</tr>
<tr>
<td>≥0.100</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Total</td>
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<td>12</td>
</tr>
</tbody>
</table>

**Table 4.—Relation between Amplitude of Largest Positive Deflection in V1 (R or R') and Etiologic Diagnosis (Heart Station Series)**

<table>
<thead>
<tr>
<th>Etiologic diagnosis</th>
<th>Total number of cases</th>
<th>QRS duration &lt; 0.12</th>
<th>QRS duration ≥ 0.12</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of cases</td>
<td>Number (mv.)</td>
<td>Number of cases</td>
</tr>
<tr>
<td>Normal</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Coronary</td>
<td>18</td>
<td>3</td>
<td>15</td>
</tr>
<tr>
<td>RVH</td>
<td>27</td>
<td>26</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>46</td>
<td>29</td>
<td>17</td>
</tr>
</tbody>
</table>

**Table 5.—Data from Vectorcardiographic Series of Fifty-seven Patients with a Tall or Late R in Electrocardiographic Lead V1**

<table>
<thead>
<tr>
<th>QRS duration (sec)</th>
<th>R'—V1</th>
<th>Etiologic diagnosis</th>
<th>Total number</th>
<th>QRS rotation in transverse projection</th>
<th>Abnormal 10-msec QRS vector</th>
<th>Terminal slowning</th>
</tr>
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<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Counter-clockwise</td>
<td>Figure-eight</td>
<td>Clockwise</td>
</tr>
<tr>
<td>&lt;0.12</td>
<td>Absent</td>
<td>Coronary</td>
<td>2</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>RVH</td>
<td>11</td>
<td>1</td>
<td>4</td>
<td>6</td>
</tr>
<tr>
<td>&lt;0.12</td>
<td>Present</td>
<td>Normal</td>
<td>4</td>
<td>4</td>
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<td>0</td>
</tr>
<tr>
<td></td>
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<td>Coronary</td>
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<td>1</td>
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</tr>
<tr>
<td></td>
<td></td>
<td>RVH</td>
<td>15</td>
<td>5</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>≥0.12</td>
<td>Present</td>
<td>Normal</td>
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<td>1</td>
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<tr>
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<td>Coronary</td>
<td>20</td>
<td>6</td>
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<td>3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>RVH</td>
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<td>0</td>
</tr>
<tr>
<td>Total</td>
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<td>57</td>
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</table>
RIGHT VENTRICULAR HYPERTROPHY AND RIGHT BUNDLE-BRANCH BLOCK

Fig. 1. "Complete right bundle-branch block" in a 28-year-old man with an atrial septal defect (ostium secundum), later confirmed at operation. Pulmonary artery pressure was 90/38 mm. Hg.

In the vectorcardiogram horizontal bars = 0.5 mv. The bar given in the upper right transverse view applies to the lower right frontal view, and the lower center sagittal view. Separate standardization bars are shown for the more highly amplified portions in the center panel. Timing is provided by interrupting the cathode-ray beam every 2.5 milliseconds.

The QRS duration is 0.12 second. The R' in V1 is of large amplitude, but no larger than that seen in some cases of coronary disease alone (see table 4). The early portion of the QRS complex is normal, but the latter portion is bent anteriorly and to the right, with terminal slowing.

As this case illustrates, marked right ventricular hypertrophy is not always accompanied by clockwise QRS rotation in the transverse projection.

Vectorcardiograms

QRS sE Loop Rotation in Transverse View.
Results are summarized in table 5. Of 11 patients with clinical and electrocardiographic signs of right ventricular hypertrophy, but no R'—V1, 10 showed an abnormal QRS rotation in the transverse projection (6 clockwise, 4 figure eight), while 1 had a very narrow loop with counterclockwise rotation. Two coronary cases with the same electrocardiographic characteristics had normal counterclockwise QRS rotation in the transverse plane.

Vectorcardiograms were available on 20 patients with an R' — V1 and QRS duration less than 0.12 second. In 4 normal subjects QRS rotation was counterclockwise. In 1 coronary case it was figure eight. In 15 right ventricular hypertrophy cases the 3 types of rotation were about equally represented.

In 24 cases with the electrocardiographic
MILNOR

FIG. 2. Electrocardiograms and vectorcardiograms from a 26-year-old man with congenital isolated pulmonic stenosis. Right ventricular pressure was 246/17 mm. Hg on cardiac catheterization. Diagnosis was confirmed at operation.

QRS duration is 0.125 second. The QRS sE-loop is displaced anteriorly and to the right. In all projections there is a prominent slowing in the midportion of the loop, rather than the terminal portion. The initial QRS vectors are within normal limits.

pattern of complete RBBB, the 1 normal subject showed normal counterclockwise transverse rotation, 2 of the cases with clinical right ventricular hypertrophy showed figure eight rotation, and 1 counterclockwise (fig. 1), and the coronary cases varied.

Cases with an rsR'S' in V1 were not significantly different vectorcardiographically from those with an rsR'. All 4 cases in the vectorcardiographic series were cases of right ventricular hypertrophy, however, and none of the cases with prolonged QRS duration showed an S'.

Abnormal Slowing in QRS sE-Loop. All but 1 of the cases with QRS duration of 0.12 second or more showed a marked slowing of the terminal portion of the loop. The 1 exception, shown in figure 2, had a marked slowing in the midportion only. Two coronary cases showed a generalized slowing throughout the whole QRS complex, and 1 had abnormal slowing in the early portion of the loop.

The terminal slow portion of the loop was directed anteriorly and rightward in all cases, and was usually near the transverse plane.

When the QRS duration was less than 0.12 second, accentuated terminal slowing was seen in a few instances, but in most cases there was no more than the normal degree of slowing in the final 20 milliseconds, regardless of the diagnosis, or the presence or absence of an R'—V1.

Initial QRS Vectors. The spatial axis of the 10-millisecond QRS vector was normal in about half of the 57 cases in table 5. In the frontal projection the normal range for this vector is quite wide (0° to −170°, and 0° to +175°), and all 57 cases were within this normal range. In the sagittal projection, 10 coronary and 12 cases of right ventricular hypertrophy had abnormal 10-millisecond vectors.
QRS Plane. In the cases with complete RBBB, the QRS plane was displaced with little or no bending in 2 cases, bent longitudinally along its major axis in 9 cases, and more complexly distorted in 13 cases. No clear correlation between clinical diagnosis and the abnormality of the QRS plane was apparent.

Discussion
Criteria for the Electrocardiographic Diagnosis of Right Ventricular Hypertrophy

The most widely used criteria for the electrocardiographic diagnosis of right ventricular hypertrophy at present are those presented by Myers, Klein, and Stofer, and by Sokolow and Lyon. These investigators clearly demonstrated the potential value of electrocardiography in the diagnosis of right ventricular hypertrophy, but the criteria they suggest are not entirely satisfactory for several reasons: (1) they lead to misdiagnosis in a significant proportion of normal persons; (2) emphasis on lead V_R rather than mean QRS axis results in failure to diagnose some cases with right ventricular hypertrophy; (3) a number of superfluous criteria are included, for example the timing of the “intrinsicoid deflection;” (4) they do not provide for the diagnosis of right ventricular hypertrophy when an R' appears in V_1.

1. First, as Braunwald, Donoso, Sapin, and Grishman have pointed out, the standards of Sokolow and Lyon are so broad that they would incorrectly attribute right ventricular hypertrophy to a number of normal persons. This is to some extent inevitable whenever rigid diagnostic criteria are set up, necessitating a clear understanding of the difference between a normal heart and a heart with a normal electrocardiogram. The specific criteria of Sokolow and Lyon, however, would have led one to suspect right ventricular hypertrophy in at least 14 per cent of our normal subjects, since 7 of them had an R - V_1 taller than 0.7 mv., and 7 others had an R/S ratio in V_1 greater than 1.0.

This difficulty can be resolved at least partially by considering both absolute amplitude of the R wave and R/S ratio in the right precordial leads, specifically V_1. An RV_1 larger than 0.5 mv. is not in itself abnormal, and was found in 8 of our 103 normal subjects, but always with a large S wave, so that the R/S ratio was less than 1.0. An R/S ratio greater than 1.0 is similarly not abnormal per se, and was found in 7 of our 103 normal subjects, but always with a small total QRS amplitude such that the R was less than 0.5 mv. The combination of these 2 characteristics was not found in any normal subject in the cases here reported. As with all criteria, it is to be expected that exceptions will appear, but they should be less frequent than with criteria based on amplitude or R/S ratio alone.

The limit of 0.5 mv. for R or RV_1 combined with an R/S ratio of 1.0, which we propose, applies to adult patients, and the specific limits in younger age groups remain to be established. We have not studied patients younger than 10 years of age, but the measurements reported by Ziegler indicate that our criteria probably apply after the age of 5 years. Unfortunately, most electrocardiographic studies of normal subjects report average R wave amplitudes and R/S ratios, with range and standard deviations, but do not evaluate the relation between these 2 measurements in each individual.

2. The second disadvantage of the criteria given by Myers and by Sokolow, in our opinion, lies in their dependence on QRS contour in the unipolar extremity leads, particularly V_R, and their exclusion of mean QRS axis as a diagnostic sign.

Reluctance to use axis deviation as an indication of ventricular hypertrophy can be traced to several sources. First, the arbitrary normal limit of +90°, which was used in early electrocardiography, soon proved to be invalid, since axes between +90° and +110° occur in a significant proportion of normal subjects. Axes in this range do not lead to a dominant R in V_R, and this, together with the concept of “electric position of the heart,” led to the conclusion that V_R was a more trustworthy guide than the mean electric axis.

The work of Ashman on the ventricular gradient gave support to this idea by implying that the normal ÂQRS had an extremely wide range. He reported the normal average Â
QRS as $+41.7^\circ$ with a standard deviation of $31.6^\circ$, so that the mean $\pm 3$ S.D. gave a range of $-53^\circ$ to $+136^\circ$. In this instance, the use of the standard deviation of a series of observations to define the normal range is misleading, since the distribution around the mean is skewed rather than symmetric. Moreover, in Ashman and Byer's original paper on the gradient, they pointed out that their normal subjects were not a random sample, since some were selected because of their extreme axis measurements, and that at least 1 of the marked right axes plotted in their charts was present only with the patient standing.

In our study and in many reports in the literature it is apparent that only a very small proportion of normal subjects have mean frontal plane QRS axes more positive than $+110^\circ$.

In our normal series no subject had a mean QRS axis further rightward than $+95^\circ$. In a series of 200 normal medical students, nurses, and hospital personnel reported by Shipley and Hallaran from Western Reserve University, the most extreme rightward axis observed was $+96^\circ$, and only 3 cases had an axis of $+90^\circ$ or more. In Ziegler's data, only 1 child in 356 between the ages of 1 and 16 years had a positive axis greater than $+105^\circ$. Packard, Graettinger, and Graybiel, in their follow-up studies on a large group of normal young aviators, found that 2 of 631 had a QRS axis from $+105^\circ$ to $+120^\circ$ on their original examination in 1940, while none of the 631 (including the original 2) had an axis beyond $+90^\circ$ when re-examined 10 years later. Vastesaeger and Rochet measured the maximal QRS vector of the frontal plane QRS sE-loop, which usually approximates, but is not identical with, the mean frontal plane QRS axis in 100 normal subjects aged 20 to 45 years, using the Wilson tetrahedron, and found a range of $-30^\circ$ to $+110^\circ$. Winsor gave the normal range of mean QRS axis as $-30^\circ$ to $+102^\circ$ in 79 normal subjects.

It seems reasonable to conclude from these data that less than 0.15 per cent of the normal adult population will have a mean frontal plane QRS axis more positive than $+110^\circ$.

Myers and co-workers stated that right axis deviation due to vertical position of the heart can be distinguished from that due to right ventricular hypertrophy by the unipolar extremity leads. We know of no evidence to support this statement. The example given in their figure 1 shows a QRS axis very close to $+90^\circ$, and could be diagnosed as within normal limits without reference to any of the extremity leads.

Unipolar extremity leads ($V_R$, $V_L$, $V_F$) are related to mean QRS axis in the same way as the standard extremity leads. A QRS axis from $+120^\circ$ to $+180^\circ$, or $-60^\circ$ to $-179^\circ$ will of necessity produce a prominent R in $V_R$. With the usual QRS sE-loop, the ratio of R/Q or R/S in $V_R$ will usually not reach the value of 4.0 or greater suggested by Myers unless the mean QRS axis is between $+135^\circ$ and $180^\circ$, or $-75^\circ$ to $-179^\circ$. Some cases with right axis deviation well beyond normal limits will therefore not show the diagnostic changes in $V_R$ described by Myers. Figure 3 illustrates such a case, and 4 similar cases were found in our autopsy series.

It has been suggested that additional right chest leads may show signs of right ventricular hypertrophy when they are absent in lead $V_R$ or $V_L$. When the QRS axis is displaced toward the right, with relatively little displacement anteriorly, there may be an increase in the R wave in $V_{3R}$ to $V_{6R}$ without diagnostic changes in $V_L$. In such cases the right axis deviation in the standard leads is almost always diagnostic without reference to $V_{3R}$, as in Myers, figure 7A and B.

Leads $V_{3R}$ and $V_{6R}$ are rarely of any additional help, since they largely reflect the frontal plane QRS axis, as evidenced by the fact that they closely resemble either lead III or an inverted lead $V_L$.

A significant proportion of cases with right ventricular hypertrophy (6 of 32 cases in our autopsy series) show right axis deviation beyond $+110^\circ$, without diagnostic changes in $V_L$. We have therefore included right axis deviation of right ventricular hypertrophy, whether or not $V_L$ is abnormal. Obviously this criterion will not apply in the presence of dextrocardia.

Ventricular hypertrophy can, of course, be present without either right axis deviation or
abnormalities in V1, but we can arrive at no further criteria at present that will identify these cases without at the same time increasing the number of “false positives.”

3. A third objection to the previously available criteria for electrocardiographic diagnosis of right ventricular hypertrophy arises from their multiplicity and complexity.

There would be no disadvantage in using a large number of criteria if diagnostic accuracy were improved thereby, but it appears that many criteria, particularly those involving T waves and intrinsicoid deflections, add to the electrocardiographer's burdens without increasing his diagnostic ability.

Inversion of T—V1 is often cited as an indication of right ventricular hypertrophy, or at least as supporting evidence, but it is a frequent normal finding. Of our normal subjects 29 per cent (30 of 103) had an inverted T—V1. Four of these subjects had an R—V1 of 0.5 mv. or more, and therefore met one of Sokolow’s criteria for right ventricular hypertrophy. Ziegler’s tables report an inverted TV1 in 86 per cent of a group of 87 normal children between the ages of 5 and 12 years.

The incidence of inverted TV1 does decrease with age in normal subjects: it was present in 37 per cent of our normal subjects under 30 years of age (27/73), and in only 10 per cent (3/30) of those 30 years and older. In the older age groups, however, an inverted TV1 is as likely to be due to coronary disease as to right ventricular hypertrophy. In the absence of other abnormalities, therefore, an inverted TV1 cannot be regarded as evidence of right ventricular hypertrophy at any age.

Ventricular hypertrophy is, of course, often associated with abnormal T waves and S—T segments, which led early observers to introduce the term “strain” to describe these changes. The abnormalities consist of deviation of the S—T segment and T wave in a direction away from the hypertrophied ventricle, until
in extreme cases they point in a direction diametrically opposite the spatial QRS axis. These changes are not specific diagnostically, since coronary disease and its effects on the myocardium, as well as many other abnormalities, can produce this same ST–T alteration.

The “Intrinsicoid Deflection.” Measurements of the “intrinsicoid deflection” are not included in this study, or in our clinical interpretation of electrocardiograms, because we believe they are misleading in theory and of no value in practice. Since this opinion is at variance with some textbooks of electrocardiography, the reasons for our views on this subject will be given in some detail.

The normal adult range (mean ± 2 S.D.) for the “intrinsicoid deflection” is given as 0.0044 to 0.030 second for V1, and 0.0244 to 0.0496 second for V6 (Kossman10). In our normal subjects the range was 0.000 to 0.030 (mean = 0.017) second for V1, and 0.020 to 0.050 (mean = 0.038) for V6.

These are said to represent the time of arrival of the excitation wave in the myocardium “beneath” the precordial electrode. Careful measurements on the exposed mammalian heart, from Lewis and Rothschild16 to Scher and Young,17 however, show that activation of the various parts of the right ventricle extends through the last 75 per cent of the total ventricular activation period, or QRS duration. It follows that a large proportion of the normal right ventricle is activated considerably later than the “intrinsicoid deflection” in lead V1, and that the time of R–V1, if it can be given any localized significance, is at best a reflection of arrival of excitation over an unidentified midportion of the right ventricle.

It seems more likely that no precise meaning referable to a localized area of the myocardium can be assigned to the “intrinsicoid deflection,” especially in the diseased heart. As Wilson pointed out, the essential difference between the “intrinsic deflection” of direct contiguous bipolar electrodes, and the “intrinsicoid” deflection of unipolar chest leads, is that all parts of the myocardium contribute, though in unequal degree, to a unipolar chest lead. Vectorcardiographic work by Duchosal and Sulzer18 and in our laboratory5 supports the conclusion that the R peak in V1 simply represents the time at which the net sum of the contributions from both ventricles to the electric potential at V1 reaches a positive maximum, and begins to decrease. The proximity of V1 to the right ventricle gives right ventricular potentials a slightly greater influence than those from the left ventricle, but the magnitude of this difference is so small that it scarcely affects the QRS contour in V1.5

It is true that many cases with right ventricular hypertrophy have an R–V1 later than normal, but only if the R is taller than normal, or if an R' is present. In both situations, if the QRS duration is normal, the evidence is consistent with the concept that the tall R–V1 of right ventricular hypertrophy represents right ventricular activity that is entirely normal in time, but increased in magnitude. The right ventricular R wave then rises out of the depths of the S wave, and may incorporate the initial small r en route, or maintain a separate existence to produce an rsR'.

When the QRS duration is prolonged, or “high normal,” on the other hand, conduction delay must be involved, as discussed below and elsewhere.19

No case in this report showed a delayed R–V1 in the absence of other QRS abnormalities. If it could be shown empirically that criteria for delayed “intrinsicoid deflection” in V1 would make possible the diagnosis of right ventricular hypertrophy when other electrocardiographic signs were lacking, then these criteria would clearly have a place in clinical interpretation. Until such evidence is forthcoming, measurement of the “intrinsicoid deflection” seems an unnecessary labor, and might profitably be abandoned.

One example may be given of the contradictions involved in the use of the intrinsicoid deflection. In following some cases as they develop right ventricular hypertrophy, R–V1 is found to increase gradually in size and become later in time, while the R/S ratio in V6 simultaneously decreases and R–V6 becomes earlier in time, the QRS duration remaining unchanged. If the late R–V1 is to be attributed to delayed activation of the right
ventricle, then one must also assume that the left ventricle is being activated earlier than usual, which seems improbable.

Reversed Precordial Transition. The normal precordial "transition," with the R/S ratio increasing from V1 to V6, is often reversed in right ventricular hypertrophy, but we have not included this in our criteria because it may be obscured when both ventricles are hypertrophied. Our present criteria for left ventricular hypertrophy are an RV6 greater than 2.5 mv., SV1 greater than 3.0 mv., or R greater than 2.2 mv. in either lead I or lead II; any one is regarded as evidence of left ventricular hypertrophy. An electrocardiogram that satisfies the criteria for both left and right ventricular hypertrophy is regarded as evidence of combined hypertrophy.

4. A fourth objection to current criteria is that an R' in V1 is regarded as evidence of a conduction delay, which makes it impossible to identify right ventricular hypertrophy. This subject is further discussed below and elsewhere.19 Our criteria apply to the greatest positive deflection in V1, whether an R or R'.

Vectorcardiograms

Right Ventricular Hypertrophy. Our vectorcardiographic investigations in this field were undertaken in the hope that the spatial vectorcardiogram might improve our ability to recognize right ventricular hypertrophy and might clarify the relation between hypertrophy and bundle-branch block. Neither of these aims has been realized to date. The vectorcardiogram has proved to be a great conceptual aid in under-

![Fig. 4. Electrocardiogram and vectorcardiogram of an 11-year-old girl with tetralogy of Fallot, later confirmed at operation.](image-url)

In the electrocardiographic precordial leads arrows indicate time of R peak in lead I. QRS duration 0.08 second. The QRS sE-loop is displaced toward the right shoulder and anteriorly.

These records illustrate extreme effects of right ventricular hypertrophy. On comparison with the records in figure 7, which show a similar QRS axis in a patient with coronary disease, the only significant differences are the total QRS duration, and the slight terminal slowing of the QRS sE-loop in figure 7.
standing the widely varying patterns seen in different scalar leads, but in our hands it has not been of much additional help in the differential diagnosis of right ventricular hypertrophy.

The characteristic alterations of the QRS sE-loop with right ventricular hypertrophy can be summarized as follows: the early QRS vectors take an abnormal direction, often simulating the changes of old myocardial infarction; the midportion of the loop, usually including the maximal QRS vector, is displaced rightward and anteriorly, the degree of this displacement being roughly proportional to the degree of right ventricular hypertrophy; the terminal portion of the loop may be normal in direction, or may be directed more anteriorly than normal. Evidence cited below suggests that the anteriorly directed terminal segments may indicate conduction delay even when no slowing is apparent.

Rightward and anterior displacement of the midportion of the loop can produce clockwise rotation of the QRS sE-loop in the transverse projection, as described by Grishman and his collaborators20, 21 and others (fig. 4). With varying degrees of displacement of the QRS sE-loop, however, the transverse projection may show a figure-eight, or a normal counterclockwise QRS rotation, as indicated by the summary in table 5, and figures 5 and 6. Moreover, clockwise or figure-eight QRS rotation in this projection is not pathognomonic of right ventricular hypertrophy, since it also occurs frequently with myocardial infarction or arteriosclerotic heart disease, particularly when the QRS duration is 0.12 second or more (fig. 7). With these reservations our data justify at least 2 conclusions: (1) in normal persons the

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**Fig. 5.** Electrocardiogram and vectorcardiogram from a 35-year-old man with severe rheumatic mitral stenosis, and right ventricular hypertrophy, confirmed at autopsy. The electrocardiogram shows normal axis, with very tall R - V1, and very large R/S - V1 ratio. QRS duration 0.09 second. QRS sE-loop is almost linear in the transverse projection.
QRS sE-loop rotation in the transverse view is always counterclockwise, even when an R'–V1 is present; (2) clockwise or figure-eight rotation in the transverse projection usually indicates right ventricular hypertrophy, provided the QRS duration is less than 0.12 second. In pediatric cardiology, where the question of coronary disease rarely arises, the latter conclusion can be extended to QRS complexes longer than 0.12 second.

Whether the spatial vectorcardiogram will identify some cases of anatomic right ventricular hypertrophy that do not have diagnostic signs in the routine scalar electrocardiogram is a question we cannot answer from the small number of patients in this category we have studied, but in 2 such cases (not included in table 5) the vectorcardiogram was within normal limits.

The Problem of “Bundle-Branch Block.” One of the earliest results of the renewal of interest in spatial vectorcardiography in this country in 1949 and 1950 was the discovery that an RSR'–V1 might exist with either of 2 different types of spatial vectorcardiogram. As Lasser, Borun, and Grishman described,21 an early and a late inflection of the QRS sE-loop is directed toward the right chest in both types, but in one the intervening portion of the loop progresses clockwise in the transverse projection, and in the other, counterclockwise. Lasser and co-workers21 reported that the clockwise type indicated right ventricular hypertrophy, while the counterclockwise type occurred with normal hearts or myocardial disease (presumably infarction), a hypothesis not borne out by our observations (table 5 and figs. 1, 6, 7).

They also concluded that “QRS vector loops of those cases not associated with right ventricular hypertrophy are all characterized by a slow irregular terminal loop directed to the
right and anteriorly..." while we have encountered this same finding in cases of right ventricular hypertrophy (fig. 7). It is probable that their conclusions were influenced by the fact that their 5 cases of right ventricular hypertrophy all had normal QRS duration, while in the 3 nonhypertrophied cases the QRS duration was prolonged.

Grishman's criterion for distinguishing between right ventricular hypertrophy and right bundle-branch block is an abnormal slowing of the terminal portion of the QRS sE-loop in the latter.21 Our observations do not support this conclusion, although we agree that the "benign" right bundle-branch block occasionally seen in normal subjects usually has counterclockwise QRS rotation in the transverse projection.

In electrocardiograms that might be interpreted as "incomplete right bundle-branch block," i. e., with QRS duration less than 0.12 second and an rsR'—V1, terminal slowing was rare, and not related to clinical diagnosis (figs. 4–6). In patients with QRS duration of 0.12 second or more, and R'—V1, terminal slowing in the coronary cases was indistinguishable from that in right ventricular hypertrophy (figs. 1, 7, 8).

It should be emphasized that while terminal slowing of the QRS sE-loop is the spatial analog of the slurred S—1 and R'—V1 of the scalar electrocardiogram, it is not direct evidence of slowed intraventricular conduction. The speed with which the cathode ray spot moves during the QRS complex is a function of the rate of change of direction and magnitude of the net vector sum of electric forces produced by ventricular depolarization, and is therefore not necessarily related to the speed with which the depolarization wave travels through any
particular area of the myocardium. It would be possible, for example, for the spot to remain entirely motionless during a large part of the QRS complex, even though activation were spreading through the myocardium, if the vector sum of the advancing activation fronts in all parts of the heart at each instant maintained the same magnitude and direction. In practice, this rarely happens, probably because of the complex structure of the mammalian heart, but it is theoretically possible, and implies that slowing of the spot is theoretically compatible with normally rapid spread of ventricular activation. Conversely, a very slowly advancing activation wave could produce normal or rapid movement of the spot, by changing its direction rapidly. Prolongation of the total QRS duration, on the other hand, is definite evidence of conduction delay, though not of its site.

With our present knowledge, one can only say that when the electrocardiogram shows the pattern designated as “complete right bundle-branch block,” the QRS sE-loop will almost always show abnormal slowing of some part of the loop. To assert that this slowing is therefore pathognomonic of conduction delay in the bundle branches is to assume facts not in evidence.

Braunwald and associates have taken a direct approach to the question of delayed ventricular activation by measuring the interval between the beginning of electric and of mechanical ventricular activity, by means of cardiac catheterization. Their results complicate the question still further by showing that neither the total QRS duration nor the presence of an R’-V₁ is a reliable indication of delayed mechanical activation. They found terminal slowing of the QRS sE-loop only in cases with...
delayed right ventricular contraction, but their observations did not include studies on normal subjects with QRS duration less than 0.10 second and R'—V1.

Initial QRS Vectors. The normal 10-millisecond QRS vector has a wide range, and may be directed toward right or left, superiorly or inferiorly. It is always anterior, however, and in the right sagittal projection the normal range with our technic is from −70° to 0° and 0° to +88°,2 thus excluding the extreme superior and inferior segments.

The direction of the initial QRS vectors was normal in about half the cases in table 5.

In general, the 10-millisecond QRS vector was more inferiorly directed in patients with clinical right ventricular hypertrophy than in normal subjects but was no further to the left than normal, contrary to the observations of Fowler and Helm.24 The most important finding is that cases of right ventricular hypertrophy may have abnormally directed initial vectors simulating myocardial infarction.

Richman and Wolff25 have reported that initial QRS vectors pointing toward the left, inferiorly, and anteriorly, indicate left BBB. They suggest that this criterion can be used to identify left BBB in cases where the standard leads suggest right BBB, but precordial leads suggest left BBB. Three of the cases in table 5 with QRS duration of 0.12 second or more had 10-millisecond QRS vectors with this orientation, but in each case the standard limb and precordial leads were characteristic of right BBB.

The Wolff-Parkinson-White anomaly sometimes produces a tall R wave in V1, simulating right BBB, and 1 case of this kind (not included in table 5) has been studied in this laboratory. The transverse projection of the QRS loop was displaced so that it lay entirely anteriorly, but was counterclockwise in rotation, a unique finding in our vectorcardiographic collection.

Origin of Q, R, or R' in V1

It is worth emphasizing that spatial vectorcardiography cannot explain the origin of the waves in the scalar electrocardiogram; it simply records them in a different way. Records from electrodes on the body surface can provide grounds for speculation about the specific events in the myocardium which give rise to different parts of the electrocardiogram or the vectorcardiogram, but confirmation requires correlation with direct epicardial or intramyocardial leads. Unfortunately the few published studies using direct leads in man give apparently conflicting results.26, 27

The 12 cases in our vectorcardiographic series that showed a Q in V1 had 10-millisecond QRS vectors directed leftward or posteriorly, most of them outside the normal spatial range. Since an initial R may be absent in V1 in normal persons, the Q—V1 in our cases is not particularly surprising, but the fact that the initial vectors are so often outside the normal spatial range requires explanation. In the coronary cases (4 out of the 12) this is presumably analogous to the usual Q wave of myocardial infarction, in which loss of some of the normal electric forces alters the resultant vector. In the cases of right ventricular hypertrophy (8 of 12), the resultant vector must be altered by addition of new forces or an increase in electric forces normally present, but why the initial vectors should be displaced in this direction is not clear.

We are unwilling to assume anatomic rotation of the heart around its long axis as an explanation, particularly in view of Grant's28 evidence that relatively little such rotation occurs. The concept that a unipolar lead that gives a QRS complex with a tall R wave must "face" the left ventricle29 seems to us untenable, particularly in an abnormal heart.

When the QRS duration is less than 0.12 second it seems probable that the tall R or R' in V1 is the result of vectors of increased magnitude from the hypertrophied right ventricle, which is in accord with the results of Carousou, Chevalier, Latsche, and Lenegre,26 but difficult to reconcile with those of McGregor.27 The interpretation of unipolar records from the ventricular surface is rendered difficult, however, by the fact that a unipolar electrocardiogram, even from the epicardium, is significantly influenced by the entire heart, and a negative deflection from one point on the right ventricular surface does not exclude the possibility of large positive deflections at other
points, e.g., the pulmonary conus. Why hypertrophied ventricular fibers should produce abnormally large voltages is not known, and one wonders how much of this increased voltage arises from the decreased distance between the enlarged heart and the chest wall. In the cases with coronary disease myocardial scarring may produce the same net result by eliminating part of the normal left ventricular contribution to the QRS sE-loop.

As noted above, conduction delay may or may not also be present when the QRS duration is less than 0.12 second. When the QRS duration is 0.12 second or more, it may be assumed that the late R or R′ − V1 actually represents delayed right ventricular activation, as Wilson’s group originally predicated. In the exceptional cases where a prolonged QRS occurs with right ventricular hypertrophy, the site of the conduction delay may well be in the hypertrophied myocardium itself.

Chronic Cor Pulmonale

All electrocardiographic criteria for the diagnosis of right ventricular hypertrophy, including ours, fail most often in older patients with chronic cor pulmonale, as many of the cases reported by Walker, Helm, and Scott and by Salazar and Sodi-Pallares demonstrate. Such patients often have a mean frontal QRS axis from +80° to +100°, and an R/S ratio in V6 of 1.0 or less, although V1 is normal. In patients over the age of 40, these characteristics may be regarded as presumptive evidence of chronic cor pulmonale, although they may occasionally occur with arteriosclerotic heart disease.

Differential Diagnosis of Coronary Disease and Right Ventricular Hypertrophy

Myocardial scarring as a result of circumscribed myocardial infarction or the more diffuse fibrosis produced by chronic coronary insufficiency can lead to electrocardiographic changes in axis and in V1 identical with those of right ventricular hypertrophy. When definitely abnormal Q waves are present, the diagnosis of old myocardial infarction is suggested, but it is impossible to be sure that right ventricular hypertrophy does not co-exist (fig. 8). In the absence of signs of old myocardial infarction, differential diagnosis from the electrocardiogram or vectorcardiogram is difficult.

Use of Diagnostic Criteria

Any proposal to establish specific criteria for clinical diagnosis should bear a caveat. Criteria for electrocardiographic or vectorcardiographic diagnosis, attempting to relate electric phenomena to changes in structure and function of the heart, are unavoidably empiric. Ideal criteria would enable a correct diagnosis in every case and would exclude all normal subjects, but neither of these aims is likely to be completely realized in the study of biologic material.

With the vectorcardiogram, as with the conventional electrocardiogram, a record “outside of normal limits” does not necessarily mean heart disease. Criteria for electrocardiographic or vectorcardiographic diagnosis of right ventricular hypertrophy provide diagnostic clues that should be appropriately weighed with all other available information, and when no other evidence to support the diagnosis is forthcoming, isolated cardiographic findings should in most cases be ignored by the clinician and filed for future reference by the investigator.

Summary and Conclusions

Electrocardiographic and vectorcardiographic studies have been carried out on normal subjects and patients with heart disease with 2 aims, to establish improved criteria for the electrocardiographic diagnosis of right ventricular hypertrophy, and to examine the relation between right ventricular hypertrophy and right bundle-branch block. Data from 238 cases have been analyzed, comprising electrocardiograms from 32 autopsied cases of right ventricular hypertrophy and 46 hospital patients with electrocardiographic signs of right ventricular hypertrophy or “complete right bundle-branch block”; electrocardiograms and vectorcardiograms from 103 normal subjects and 57 patients with a tall or late R − V1.

The following criteria for the electrocardiographic diagnosis of right ventricular hypertro-
phy are proposed: QRS duration less than 0.12 second, plus either a mean frontal plane axis from $+110^\circ$ to $\pm 180^\circ$, or $-91^\circ$ to $\pm 180^\circ$, or R/S or R'/S ratio in V1 greater than 1.0, with R or R' greater than 0.5 millivolt. The advantages over previously published criteria include fewer misdiagnoses in normal persons, correct diagnosis in a higher proportion of cases of right ventricular hypertrophy, elimination of unnecessary criteria, and applicability to records with R'–V1.

A small proportion of records that meet these criteria will be from patients with coronary artery disease and no right ventricular hypertrophy. This is equally true of other published criteria.

Prolongation of the QRS duration to 0.12 second or more, with the electrocardiographic pattern of the right bundle-branch block, is much more commonly due to coronary disease than to right ventricular hypertrophy. It occasionally occurs in persons without heart disease.

An R'–V1 with normal QRS duration occurs frequently with right ventricular hypertrophy, and occasionally in normal subjects and patients with coronary disease. The designation of this pattern as “incomplete right bundle-branch block” is misleading and should be abandoned until more information is available.

The spatial vectorcardiogram is sometimes helpful in establishing the presence of heart disease in a patient with the electrocardiographic pattern of complete right bundle-branch block, since figure eight or clockwise rotation of the QRS sE-loop in the transverse projection has not been observed in normal persons, even with prolonged QRS duration.

In right ventricular hypertrophy, transverse projection QRS rotation is clockwise or figure eight in the majority of cases, but this also occurs with coronary disease and is therefore not pathognomonic. Initial QRS vectors or other characteristics of the QRS sE-loop do not help to distinguish the cases of coronary disease.

Terminal slowing of the QRS sE-loop occurs in most cases with the electrocardiographic pattern of “complete right bundle-branch block,” but at present provides no diagnostic information beyond that available from the QRS contour and duration in the scalar electrocardiogram.

One case of Wolff-Parkinson-White anomaly is described, in which the electrocardiogram imitated right ventricular hypertrophy, but the spatial vectorcardiogram was not characteristic of right ventricular hypertrophy.

**Summario in Interlingua**

Studios electrocardiographic e vectocardiographic ha essite execute in subjectos normal e in patientes con morbo cardiac con le 2 objectivos (1) de estable meliorate criterios pro le diagnose electrocardiographic de hypertrophia dextero-ventricular e (2) de examinar le relation inter hypertrophia dextero-ventricular e bloco de branca dextere. Esseva analysate datos ab 238 casos, incluse electrocardiogrammas ab 32 necropsiase casos de hypertrophia dextero-ventricular e ab 46 patientes hospitalisate con signos electrocardiographic de hypertrophia dextero-ventricular o de “complete bloco de branca dextere” e electrocardiogrammas e vectocardiogrammas ab 103 casos normal e 57 patientes con alte o retardate R – V1.

Le sequente criterios es proponite pro le diagnose electrocardiographic de hypertrophia dextero-ventricular: Duration de QRS de minus que 0,12 secundas e in plus (a) axe medie de plano frontal inter $+110^\circ$ e $\pm 180^\circ$ o inter $-91^\circ$ e $\pm 180^\circ$ e (b) un proportion R/S o R'/S in V1 de plus que 1.0, con R o R' de plus que 0,5 millivolt. Le avantages in comparation con previemente descritbe criterios include minus diagnoses erronee in subjectos normal, correcte diagnoses in un alte procentage de casos de hypertrophia dextero-ventricular, elimination de criterios non requirite, e applicabilitate a registrationes con R' – V1.

Un parve numero de registrationes que satisface iste criterios va esser trovate inter le registrationes ab patientes con morbo de arteria coronari e sin hypertrophia dextero-ventricular. Isto es equalmente ver pro altere criterios publicate.

Le prolongation del duration de QRS a 0,12 secundas o plus in casos con le configuration
electrocardiographie de bloco de branca dextere es molto plus communmente debite a morbo coronari que a hypertrophia dextero-ventricular. In rar casos illo occurre in subjectos sin morbo cardiac.

Un $R' - V_1$ con duration normal de QRS occurre frequentemente in casos de hypertrophia dextero-ventricular. Illo occurre sporadicamente in subjectos normal e in patientes con morbo coronari. Le uso del termino “incomplete bloco de branca dextere” pro iste configuration es infeliz e debereba esser aban-donate usque informaciones additional deveni disponibile.

Le vectocardiogramma spatial es a vices utile in establir le presentia de morbo cardiac in un paciente qui exhibi le configuration electrocardiographie de complete bloco de branca dextere, proque rotation in forma de ‘8’ o rotation dextrorse del spira sE de QRS in le projection transverse ha non essite observate in subjectos normal, mesmo in casos de QRS prolongate.

In hypertrophia dextero-ventricular, le rotation de QRS in le projection transverse es dextrorse o del forma de ‘8’ in le majoritate del casos, sed le mesme observation vale pro morbo coronari, e per consequente illo non es pathognomonie. Vectores de QRS initial o altere caracteristicas del spira sE de QRS es sin valor in differeniar le casos de morbo coronari.

Relentation terminal del spira sE de QRS occurre in le majoritate del casos exhibente le configuration electrocardiographie de “complete bloco de branca dextere,” sed al tempore presente iste phenomeno provide nulle information diagnostic in ultra de lo que es providite per le contorno de QRS e per le duration de illo in le electrocardiogramma scalar.

Es describete un caso del anomalia de Wolff-Parkinson-White in que le electrocardiogramma reflecteva hypertrophia dextero-ventricular durante que le vectocardio-gramma spatial non esseva caracteristic de iste condition.

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But weightier still are the contentment which comes from work well done, the sense of the value of science for its own sake, insatiable curiosity, and, above all, the pleasure of masterly performance and of the chase. These are the effective forces which move the scientist. The first condition for the progress of science is to bring them into play.—L. J. Henderson, 1878–1942.
Electrocardiogram and Vectorcardiogram in Right Ventricular Hypertrophy and Right Bundle-Branch Block
W. R. MILNOR, Charles A. Bertrand and Frederick R. Mugler

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