The Correlation Between the Electrocardiographic Patterns of Ventricular Hypertrophy and the Anatomic Findings

By Ralph C. Scott, M.D.

The correlation of the electrocardiographic patterns of ventricular hypertrophy with the anatomic findings offers a firm basis for an appraisal of the accuracy of electrocardiographic criteria.

The attempt to make such a correlation, if it is to have value, however, must be based upon certain careful considerations. The anatomic evidence of ventricular enlargement must be critically approached. Separating the right and left ventricles and weighing and measuring each is the ideal manner of determining individual chamber hypertrophy. This was the method employed (with slight variation) by Müller,1 Lewis,2 Herrmann and Wilson,3 Jones,4 and Stofer and Hiratzka.5 However, this is a tedious process, and accurate division and apportionment of the interventricular septum to the appropriate ventricle frequently poses a difficult problem. An alternative and easier method, advocated by Fulton and associates6 and employed by Grant,7 involves cutting the right ventricle free at its junction with the interventricular septum and considering the latter a part of the left ventricle. The weights of the normal ventricles as determined by these investigators are listed in table 1. The normal left ventricular/right ventricular weight ratios are also given.

The anatomic division of the septum into right and left ventricular components in the past has been a source of difficulty and inaccuracy.2-4,6 More recent studies8-10 have shown that the right and left septal masses can be separated. It has also been shown that in the upper two thirds of the septum the left septal mass constitutes 70 to 80 per cent of the total septal mass.10 In left ventricular hypertrophy (LVH) the left septal mass forms more than 80 per cent of the total and in right ventricular hypertrophy (RVH) the right septal mass may comprise 50 per cent or more of the septal weight.10

The majority of studies dealing with electrocardiographic and pathologic correlations, however, have not included such careful separation of the ventricular muscle mass. Instead, they have been based upon careful measurement of the thickness of the free wall of the right and left ventricles or the determination of the total heart weight. The normal total heart weight varies with the sex, body length, and weight11 and ranges roughly from 200 to 400 Gm.11,12 Most workers consider that in order to diagnose RVH in an adult the greatest thickness of the right ventricle must be 5 mm. or more, and to diagnose LVH the greatest thickness of the left ventricle must be 13 (or 14) mm. or more. Care must be taken not to include the papillary muscles in this measurement. In studies from our laboratory the additional stipulation was made that the total heart weight exceed one standard deviation above the mean of normal predicted for the body length according to Zeeck's criteria.11 It is true that some cases of anatomic RVH may not be included by insisting on this last criterion, since, as has been pointed out by White,12 Thomas and James,13 and Fulton and associates,6 the right ventricular
VENTRICULAR HYPERTROPHY

Table 1

Normal Ventricular Weights

<table>
<thead>
<tr>
<th>Author Year</th>
<th>No. of cases</th>
<th>RV Gm. Range</th>
<th>Av.</th>
<th>LV Gm. Range</th>
<th>Av.</th>
<th>Septum Gm. Range</th>
<th>Av.</th>
<th>Total ventricular weight Gm. Range</th>
<th>Av.</th>
<th>LV/RV Range</th>
<th>Av.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lewis 1914</td>
<td>11*</td>
<td>35-60</td>
<td>44</td>
<td>54-117</td>
<td>81</td>
<td>14-30</td>
<td>23</td>
<td>104-108</td>
<td>149</td>
<td>1.50-2.06</td>
<td>1.83</td>
</tr>
<tr>
<td>Herrmann and Wilson 1922</td>
<td>16</td>
<td>28-70</td>
<td>47</td>
<td>60-112</td>
<td>82</td>
<td>13-46</td>
<td>27</td>
<td>105-221</td>
<td>156</td>
<td>1.57-2.18</td>
<td>1.79</td>
</tr>
<tr>
<td>Fulton et al. 1950</td>
<td>43</td>
<td>23-68</td>
<td>46</td>
<td>48-123</td>
<td>86</td>
<td>17-61</td>
<td>39</td>
<td>88-235</td>
<td>171</td>
<td>1.46-2.14</td>
<td>1.74</td>
</tr>
<tr>
<td>Stofer and Hiratzka 1952</td>
<td>82</td>
<td>61.87</td>
<td></td>
<td>119.78</td>
<td>307.43</td>
<td>±11.81</td>
<td>±13.88</td>
<td>±21.65</td>
<td>1.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(64 males) (28 females)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jones 1953</td>
<td>85</td>
<td>33</td>
<td>66</td>
<td>66</td>
<td>165</td>
<td>1.36-2.43</td>
<td>1.92</td>
<td>2.0</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

"Controls.

LV: Left ventricle; RV: Right ventricle; LV/RV: Left ventricular/right ventricular weight ratio; LVH: Left ventricular hypertrophy; RVH: Right ventricular hypertrophy.

LV/RV above 2.20 indicates left ventricular preponderance. LV/RV below 1.50 indicates right ventricular preponderance.

LV+S/RV ranges from 2.3 to 3.3 normally; in isolated RVH this ratio always less than 2.

Free wall RV of 80 Gm. or more indicates RVH; right ventricular preponderance may occur in small hearts with right ventricular weights less than 80 Gm.

Free wall of LV+S normally less than 190 Gm.; when 250 Gm. or more indicates LVH.

muscle mass may be increased in some cases without the total heart weight being abnormal.

Many sets of electrocardiographic criteria have been developed over the years for the diagnosis of RVH, LVH, and combined ventricular hypertrophy (CVH). These have been largely empirical and some have proved unreliable. The ideal electrocardiographic criteria for ventricular hypertrophy would indicate all cases of hypertrophied right or left ventricles and would not so diagnose any normal cases. This is not possible with presently available criteria.

Résumé of Electrocardiographic-Pathologic Correlation Studies

Left Ventricular Hypertrophy

The electrocardiographic diagnosis of LVH is one of the easiest and one of the most important diagnoses to be made from the clinical electrocardiogram. The many criteria that have been proposed can be summarized as being based upon abnormal left axis deviation, high voltage in left precordial leads, delayed left ventricular activation time, or ST-segment and T-wave abnormalities.

Noth, Myers, and Klein analyzed the precordial electrocardiogram in 84 pathologically proved cases of LVH, and in 52 cases in which the heart was normal at autopsy. They found that the normal electrocardiogram and the LVH pattern could not be differentiated from the amplitude of the R waves in V5 and V6 alone. Of the 84 cases of LVH, 34 (41 per cent) showed a Q-R duration of 0.05 second or longer or an R duration of 0.04 second or longer. The average cardiac weight in this group was 610 Gm. Eight (10 per cent) showed borderline values (Q-R of 0.045 to 0.049 or R of 0.038 to 0.039 second) and the cardiac weight averaged 569 Gm. Forty-two
cases (50 per cent) showed normal Q-R or R
duration and had an average cardiac weight
of 536 Gm. These workers found a general
trend toward increasing duration of Q-R or
R with increasing heart weight but many indi-
vidual exceptions were encountered. Of inter-
est was their observation that 4 cases of anato-
mic LVH showed normal electrocardio-
grams.

Levine and Phillips studied 38 cases that
satisfied the electrocardiographic criteria of
Wilson for LVH.

At autopsy all cases showed anatomic LVH.
Scott and associates collected 100 cases of
autopsy-proved isolated LVH in adults. Eight
sets of currently employed electrocardio-
graphic criteria for the diagnosis of LVH
were then subjected to analysis in these 100
cases. The criteria of Wilson and of Sokol-
low were found to give the highest number of
positive diagnoses. One or more of Wilson’s
criteria were present in 81 per cent of cases.
However, it was observed that if a minimum
of 2 of these criteria were required, only 53
per cent of the cases showed positive diag-
noses. One or more of Sokolow’s criteria
were found in 85 per cent of cases. Similarly, if
2 or more of these criteria were required, the
number of positive diagnoses diminished to
by proven LVH. It did not test the specificity
of these criteria, that is, the occurrence of false
positive diagnoses.

Chou and co-workers attempted to evalu-
ate the specificity of these same electrocardio-
graphic criteria in the diagnosis of LVH. The
autopsy findings of 100 cases diagnosed as
LVH by the electrocardiogram were analyzed
with regard to anatomic ventricular hyper-
trophy. The electrocardiographic diagnoses
were made with the combined criteria previ-
ously reported. In all cases the tracings were
taken within 3 months before death. No cases
of myocardial infarction were included. There
were 44 cases with isolated LVH, 45 with
CVH, 1 with isolated RVH, 1 with no ventric-
ular hypertrophy, and 9 with questionable
hypertrophy. The 9 cases with questionable
hypertrophy included 3 with increased heart
weight but normal ventricular measurements
and 6 cases with normal heart weight but in-
creased ventricular measurements. A review
of the electrocardiographic findings in the
positive, false positive, and questionable groups
failed to reveal any distinguishing features
among the various criteria.

Selzer and associates have also recently
approached the problem of the reliability of
the electrocardiographic diagnosis of LVH.
They studied 108 tracings that demonstrated
the pattern of LVH by the same electrocardio-
graphic criteria employed in our earlier
study in which autopsy findings were avail-
able. These workers relied upon total heart
weight in the assessment of anatomic LVH,
considering abnormally heavy those hearts
weighing more than 25 Gm. above the upper
limit of normal according to Zeek’s criteria. Some of the hearts in this study, however, also
showed anatomic evidence of RVH, based
upon an LV/RV wall thickness ratio of 3 to 1
or less. They encountered 75 cases in which
the electrocardiographic diagnosis of LVH
was confirmed pathologically, 16 cases in
which the hearts were of borderline hyper-
trophy, and 17 in which there was no anatomic
LVH. In none of the 17 cases with normal
heart weights was there demonstrable cause
for cardiac hypertrophy. The majority of pa-
tients in this group died of malignant disease and showed considerable emaciation. They evaluated the frequency of the 3 main classes of electrocardiographic criteria: high voltage, prolonged ventricular activation time, and ST-T alterations. In their 75 cases of autopsy-proved LVH, these criteria were encountered in 71, 40, and 65 instances respectively. Of particular interest were the observations in the 17 cases without LVH in which these criteria were found 16, 6, and 7 times respectively. Thus, any of these electrocardiographic criteria may occur in the absence of anatomic LVH. Nineteen of the 75 proved cases of LVH exhibited a horizontal heart position. There was only a slight preponderance of this position in the heavier hearts than in the lighter and false-positive cases. Eleven of their 75 cases of proved LVH also had anatomic RVH, yet displayed LVH in the electrocardiogram. These authors suggest that CVH may tend to rotate the mean electrical axis to a more vertical position. While high voltage in the precordial leads was the most sensitive criterion, being present in most cases in the series, it was also most frequently responsible for a false diagnosis of LVH. A somewhat surprising finding was their observation that the prolonged ventricular activation time was the least reliable sign. Also of interest was the fact that 6 of the 40 heaviest hearts in the series showed normal ST-T portions. These workers found that electrocardiograms displaying more than one criterion were more reliable in the diagnosis of LVH. While these authors considered ST-segment depression and T-wave inversion (in leads exhibiting high upright QRS complexes) not specific enough to constitute the sole basis for a diagnosis of LVH, yet the addition of this criterion to the criteria of high voltage and delayed ventricular activation time constituted the most specific combination for the diagnosis of LVH.

Selzer and co-workers\(^{30}\) have also investigated the problem of the false-negative electrocardiographic diagnosis of LVH. They reviewed the electrocardiograms from nearly 300 patients whose hearts at autopsy weighed over 400 Gm. These cases were divided pathologically into pure LVH, pure RVH, and CVH. The actual number of cases in each category was not stated. Cases with myocardial infarction, fibrosis, or bundle-branch block were not excluded. They stated that by means of "any acceptable" electrocardiographic criterion the diagnosis of LVH was made in 45 per cent of patients displaying LVH or CVH at autopsy. In 32 per cent the diagnosis of LVH was obscured by the pattern of myocardial infarction or nonspecific ST-T abnormalities and in 8 per cent the diagnosis of LVH was obscured by bundle-branch block. In 15 per cent of the group the electrocardiograms were normal or near normal, despite appreciable LVH at autopsy. These constituted true false-negative diagnoses.

Grant\(^{31}\) assessed the role of LVH in the production of left axis deviation (LAD) in an electrocardiographic-pathologic correlation study that included 672 cases. He studied 73 cases of anatomic LVH, employing the criteria of hearts weighing over 500 Gm., together with an increase in thickness of the left ventricular wall. These, of course, are rather extreme criteria and would exclude cases of less marked LVH. He found 35 of these 73 cases to exhibit LAD, 35 with a normal axis, and 3 with a vertical axis. He further observed that the development of LAD is not dependent upon the severity of the LVH; among the 9 cases with hearts over 900 Gm. due to LVH, 6 had a normal axis, and only 3 had LAD. In addition, he observed in his study 131 cases of LAD. When those with myocardial infarction were excluded, there were 77 remaining. He stated that only 35 of these 77 (less than half) had heart weights of 500 Gm. or more; 19 had heart weights of 400 to 500 Gm., and 23 had hearts weighing less than 400 Gm. He concluded that LAD is by no means diagnostic of LVH. It should be pointed out, however, that by most pathologic criteria, the 19 hearts weighing between 400 and 500 Gm. (and perhaps even some of those weighing less than 400 Gm. if the patients were small) would be considered in the hypertrophy range. If these were included, the incidence of LAD in Grant's series of LVH

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would increase to at least 54 of the 77 cases (70 per cent). In this study and in a previous one, Grant demonstrated that neither variation in body build nor variation in the anatomic position of the heart can alone be responsible for LAD. In fact, he has shown that in LVH the electrical axis may be markedly rotated to the left while the anatomic position of the left ventricle is essentially the same as in the normal heart. He emphasized that the incidence of LAD is higher among patients with anatomic LVH than it is among patients with normal hearts. He believed that it is not the hypertrophy itself that is responsible for the LAD but that it is the myocardial fibrosis of the free wall of the left ventricle that may frequently accompany marked LVH. This patchy fibrosis is thought to result in an alteration in the peripheral parts of the left ventricular conduction network resulting in what is termed "parietal block." When the peripheral fibers of the anterior division of the left bundle are involved, the spread of excitation is upward from the diaphragmatic region of the left ventricle. The QRS forces then point superiorly and leftward, producing LAD in the clinical electrocardiogram. While there are other causes of abnormal LAD, such as peri-infarction block, chronic coronary artery disease, etc., we are concerned at this time only with those that are related to LVH.

In Grant’s series, the voltage criteria identified over 90 per cent of cases of marked LVH when the body build was normal. However, in subjects markedly overweight or underweight for their height, the criteria were totally unreliable. He cited the examples of 3 subjects with hearts weighing over 700 Gm. due to LVH with QRS complexes not more than 20 mm. in amplitude in the precordial leads; all 3 were obese. There were 7 cases with heart weights less than 450 Gm. and QRS complexes with amplitudes of more than 45 mm. in one or more precordial leads. All 7 subjects were thin and their average height was 66 inches. It should be pointed out here again, however, that some of these hearts, even though weighing less than 450 Gm., may actually have left ventricular hypertrophy. In fact, Zeek’s normal heart weights for males of 168 cm. (66 in.) in length is 317 ± 40 Gm. and for females of this height is 277 ± 30 Gm. Furthermore, Grant did not specify which precordial leads exhibited high voltage. This is not meant to detract from the observation that voltage criteria may be unreliable in thin individuals but that the examples cited are not absolutely conclusive.

In a recent report Abdin studied the relationship between the electrocardiographic changes and postmortem findings in 19 cases of aortic stenosis and in 15 cases of hypertension (without evidence of "ischemic" heart disease at necropsy). All the cases of aortic stenosis had LVH, the thickness of the left ventricle ranging from 16 to 30 mm., with an average of 21. All 15 cases of hypertension had LVH, the left ventricular thickness ranging from 18 to 25 mm., with an average of 22. Abdin found Sokolow’s voltage criteria for LVH to be present in all but 1 case of aortic stenosis and 1 case of hypertension. The striking feature of this study was that for comparable degrees of anatomic LVH, T-wave inversion in left ventricular leads was much more marked and frequent in aortic stenosis than in hypertension. T-wave inversion of 5 to 10 mm. occurred in 10 cases of aortic stenosis but was found in only 1 case of hypertension. He offered evidence to support the concept that the T-wave inversion in aortic stenosis is ischemic in origin and due to poor coronary flow and not to coronary artery disease.

Abdin also studied the LV/RV thickness ratio and the relation to vertical heart position and "clockwise" rotation. While a vertical heart position was common in aortic stenosis, it was less frequent in advanced cases and not necessarily related to associated RVH, since half with a vertical heart position had no anatomic RVH. In contrast, extreme "clockwise" rotation (RCS in V3) in this series was found to have anatomic RVH in each instance.

Selzer and associates have quite recently
studied the effect of cardiac dilatation upon the electrocardiographic pattern of LVH. They selected 28 autopsied cases with heart weights exceeding 500 Gm., no RVH, no significant coronary artery disease, and no myocardial fibrosis or infarction. Twelve cases had pure concentric LVH and the electrocardiograph displayed LVH in 10, right bundle-branch block (RBBB) in 1, and a normal record in 1. Sixteen cases had LVH with dilatation and their electrocardiograms showed LVH in 9, bundle-branch block in 6, and no diagnostic abnormality in 1. The only trend observed was the more common occurrence of conduction defects, widening of the QRS complexes, and of LAD in the group with both hypertrophy and dilatation. Because of the wide overlapping of findings in the 2 groups, these workers concluded that the occurrence of left ventricular dilatation in addition to the pure increase in left ventricular muscle mass does not produce sufficiently characteristic electrocardiographic alterations to suggest a distinctive pattern.

Blondeau, Heller, and Lenegre have quite recently reported a significant autopsy study of 136 cases of left-sided heart disease. They correlated the voltages of the QRS complexes with the weight of the left ventricle, the LV/RV weight ratio, and the degree of left ventricular thickness and dilatation. The left ventricular weight ranged from 119 to 640 Gm. and the LV/RV ratio from 1.1 to 3.8. The Lewis index, \( \left( R_1 + S_4 \right) - \left( R_3 + S_1 \right) \), was above +17 in 30 per cent of cases and was found to be dependent upon the weight of the left ventricle. The "index" of Sokolow and Lyon was greater than 35 in 54 per cent of the cases and the sum of RV7 + SV2 was over 35 in 65 per cent. These workers observed that this appeared to be the most sensitive electrocardiographic sign of LVH. The amplitudes of the R waves in V6 and V7 were found to be influenced chiefly by the weight of the left ventricle while the depth of the S waves in V1 and V2 was dependent on the LV/RV weight ratio and also the degree of thickening and dilatation of the left ventricle.

Left Ventricular Hypertrophy in Infancy and Childhood

The electrocardiographic criteria for LVH in children are essentially the same as in adults. However, because of the higher voltage normally generated in infancy and childhood and because of the thinner chest walls, the voltage requirements must exceed the maximum normal for their age. Nadas has proposed that LVH is probably present in infants and children if R in V5 or V6 exceeds 35 mm., the sum of R in V5 or V6 plus the S in V1 or V2 exceeds 45 mm., the R wave in aVL or aVF exceeds 20 mm., the R in I plus S in III exceeds 30 or the sum of R in II and III exceeds 45 mm., or the onset of the intrinsicoid deflection in V5 or V6 exceeds 0.04 second. Inversion of T waves in left precordial leads with or without increase in voltage has been considered to indicate LVH in infants under 1 year of age as well as in older children.

To our knowledge, however, there have been no extensive autopsy-controlled studies that establish the accuracy and reliability of these criteria in infancy and childhood. We are currently engaged in such a project.

Right Ventricular Hypertrophy

The correlation of the electrocardiographic diagnosis of RVH with the pathologic findings has been studied by a number of investigators. In some series, although the correlation between the electrocardiogram and the clinical evidence of RVH has included a large number of cases, the number with autopsy control is small. Accordingly, such studies have not been included in detail in this presentation.

The current electrocardiographic diagnosis of RVH has been based largely upon the criteria established by Wilson, Myers, and Sokolow. These criteria depend primarily upon changes in the right precordial leads: namely, increase in height of the R wave, decrease in depth of the S wave with an increase in the R/S ratio, delay in the onset of the intrinsicoid deflection, and ST-segment and T-wave changes. A qR pattern, as well as an rSR' pattern has been described in right pre-
cordial leads. In the left precordial leads the R is often small with a prominent S. The total duration of the QRS is less than 0.12 second and often normal. A tall R wave in aVR and right axis deviation (RAD) may occur.

Milnor\textsuperscript{44} has proposed new criteria for the electrocardiographic diagnosis of RVH purported to decrease the incidence of "false positive" and "false negative" interpretations and to make possible the recognition of RVH in the presence of R' in V\textsubscript{1}. These criteria consist of a QRS duration less than 0.12 second and either (1) a mean frontal plane axis between +110° and ±180° or between –90° and ±180° or (2) R/S or R'/S ratio in V\textsubscript{1} greater than 1.0 with R or R' greater than 0.5 mV. (5 mm.).

A summary of the studies with what we consider reasonably adequate autopsy control is presented in table 2. It can be seen, however, that even in these studies there was wide variation in the age groups included, in the etiology of RVH, in the electrocardiographic criteria employed, and whether isolated or only predominant RVH was present anatomi- cally.

Myers and associates\textsuperscript{42} found 7 of their 40 autopsied cases in which the electrocardiogram was not diagnostic of either RVH or a conduction defect in the right ventricle. In the cases in which the diagnosis was missed, there were 6 of cor pulmonale and 1 of mitral stenosis. These authors could find no direct correlation between cardiac weight, ventricular ratio, or thickness of the right ventricular wall and the electrocardiographic pattern.

Sokolow and Edgar\textsuperscript{45} have correlated the electrocardiographic and pathologic findings in 38 patients with congenital heart disease. Of these, 28 cases in which the electrocardiogram showed RVH there was anatomic RVH (some of these, however, also had accompanying anatomic LVH). One case had RVH at autopsy but was not diagnosed as RVH electrocardiographically by these workers. It should be pointed out, however, that this case did display marked RAD.

Carousu and co-workers\textsuperscript{46} found in their 58 cases abnormal alterations in the R/S ratio in V\textsubscript{1} and V\textsubscript{5-6} suggestive of RVH in 44, and delay in the onset of the intrinsicoid deflection in V\textsubscript{1} in 37. These workers calculated the LV/RV weight ratio and related their findings to the disturbance in the ratio of R/S in V\textsubscript{1} and V\textsubscript{5-6}. They found RVH in the electrocardiogram only when the LV/RV weight ratio was less than 1.2. When the R/S ratio was abnormal in both V\textsubscript{1} and V\textsubscript{5-6}, the anatomic RVH was extreme (LV/RV of 0.78 to 0.92).

Woods\textsuperscript{47} analyzed the electrocardiograms in 52 cases of tetralogy of Fallot and found electrocardiographic evidence of RVH in all. In 7 autopsied cases, the relative thickness of the ventricle was measured and in all there was evidence of marked RVH. However, there was no correlation between the relative thickness of the 2 ventricles and the degree of RVH in the electrocardiogram as measured by either the height of the R wave or the R/S ratio in V\textsubscript{1}. A similar conclusion was reached by Donzelot and associates in a study of 10 cases of tetralogy of Fallot.\textsuperscript{48}

On the basis of their study, Camerini and co-workers\textsuperscript{49} concluded that in an adult the pattern of R>S in V\textsubscript{4R} was almost invariably diagnostic and was encountered most frequently in subjects with considerable rather than slight RVH. Milnor\textsuperscript{44} correctly diagnosed 24 of his 32 autopsied cases. Six of these would have been missed by the criteria of Myers\textsuperscript{42} or Sokolow.\textsuperscript{43}

Walker, Helm, and Scott\textsuperscript{50} in their analysis of 22 cases of autopsy-proved, isolated RVH found that age, heart weight, or the LV/RV thickness ratio did not appear to influence the frequency of positive findings. Electrocardiographic evidence of RVH, however, occurred more frequently as the thickness of the right ventricle increased. In this study only 1 case with an R wave in aVR of 5 mm., or greater, was encountered and no electrocardiogram showed an R in V\textsubscript{1} of 7 mm. or greater. Only 1 case had a frontal plane QRS axis greater than +110° while, of considerable interest, 4 cases had LAD of –30°.

In a related study, Walker, Scott, and Helm\textsuperscript{51} determined the reliability of the elec-

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# Right Ventricular Hypertrophy

**Table 2**

<table>
<thead>
<tr>
<th>Author Year</th>
<th>Age Range</th>
<th>Heart wt. (range)</th>
<th>Increased RV thickness or decreased LV/RV ratio</th>
<th>Prep. or isolated RVH</th>
<th>Etiology</th>
<th>ECG criteria employed</th>
<th>rSR pattern in right precordial leads</th>
<th>No. of autopsied cases</th>
<th>Positive ECG diagnosis</th>
<th>Accuracy of ECG diagnosis of RVH (per cent)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myers et al. 1948</td>
<td>15-52 yrs.</td>
<td>284-700</td>
<td>LV/RV Ratio</td>
<td>Prep.</td>
<td>13</td>
<td>24</td>
<td>3</td>
<td>Other</td>
<td>Myers*</td>
<td>Included</td>
</tr>
<tr>
<td>Caruso et al. 1951</td>
<td>—</td>
<td>230-630</td>
<td>↑Thickness</td>
<td>LV/RV Ratio</td>
<td>Prep.</td>
<td>22</td>
<td>31</td>
<td>2</td>
<td>3</td>
<td>Lewis* Wilson* Myers* Sokolow*</td>
</tr>
<tr>
<td>Levine and Phillips 1952</td>
<td>—</td>
<td>90-270</td>
<td>↑Thickness</td>
<td>Isolated</td>
<td>—</td>
<td>—</td>
<td>7</td>
<td>—</td>
<td>Wilson Myers*</td>
<td>—</td>
</tr>
<tr>
<td>Woods 1956</td>
<td>Adults</td>
<td>—</td>
<td>↑Thickness</td>
<td>Isolated</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Myers* +V4R</td>
</tr>
<tr>
<td>Walker et al. 1955</td>
<td>15-57 yrs.</td>
<td>315-675</td>
<td>↑Thickness</td>
<td>Isolated</td>
<td>5</td>
<td>—</td>
<td>1</td>
<td>16</td>
<td>Myers* Sokolow*</td>
<td>Included</td>
</tr>
<tr>
<td>Milnor 1957</td>
<td>&gt;1 yr.</td>
<td>&gt;25% of normal</td>
<td>↑Thickness</td>
<td>Isolated</td>
<td>14</td>
<td>12</td>
<td>6</td>
<td>—</td>
<td>Milnor*</td>
<td>Included</td>
</tr>
<tr>
<td>Bialostozky et al. 1957</td>
<td>4 mos.-65 yrs.</td>
<td>45-600</td>
<td>↑Thickness</td>
<td>Prep.</td>
<td>4</td>
<td>4</td>
<td>8</td>
<td>—</td>
<td>Braunwald*</td>
<td>—</td>
</tr>
<tr>
<td>Hollman 1958</td>
<td>1 mo.-15 yrs.</td>
<td>Not given</td>
<td>Not given</td>
<td>Isolated</td>
<td>—</td>
<td>—</td>
<td>29</td>
<td>—</td>
<td>Hollman*</td>
<td>Included</td>
</tr>
<tr>
<td>Evans and Short 1958</td>
<td>15-52 yrs.</td>
<td>280-700</td>
<td>↑Thickness</td>
<td>Both</td>
<td>—</td>
<td>—</td>
<td>10</td>
<td>—</td>
<td>Evans and Short*</td>
<td>Included</td>
</tr>
</tbody>
</table>

RV: right ventricular; LV/RV: left ventricular/right ventricular ratio; Prep.: preponderant; MS: mitral stenosis; CP: cor pulmonale; Cong.: congenital; IRBBB: incomplete right bundle-branch block (rSR'); CRBBB: complete right bundle-branch block; LBBB: left bundle-branch block.

*See text.

‡See text (2 cases with CYH).
trocardiographic pattern of RVH. Tracings showing RBBB were excluded. Twelve adult cases that fulfilled these criteria and had autopsy examination including a determination of heart weight and ventricular wall thickness were analyzed. Eight of the 12 demonstrated RVH anatomically. A surprising finding in the study was that 4 cases had anatomic LVH.

Bialostozky and associates\(^{32}\) found the electrocardiographic criteria for RVH as proposed by Braunwald and associates\(^{40}\) to be positive in 14 of 16 autopsy-proved cases of isolated RVH.

Phillips\(^{33}\) has recently made some interesting observations on the electrocardiographic changes in cor pulmonale secondary to pulmonary emphysema. Employing different sets of electrocardiographic criteria used by previous authors,\(^{40, 43, 44, 54, 55}\) he analyzed the electrocardiograms in 18 cases in which autopsy disclosed anatomic RVH. He emphasized that the electrocardiographic criteria for the diagnosis of RVH depend upon 2 basic changes in the electrical forces: their orientation toward the right and their orientation anteriorly. He postulated that a pattern of evolution of the electrocardiogram of cor pulmonale occurs in 3 stages. In the first stage, the major electromotive forces are directed leftward and posteriorly (normal electrocardiogram). In the second stage there is rightward but still posterior direction of the major electromotive force (RAD, prominent S waves in left precordial leads). In the third stage there is anterior as well as rightward direction of the mean spatial vector (tall R waves over the right chest as well as RAD). Patients in this last category of his series had the heaviest hearts with the thickest right ventricular walls. He concluded that those electrocardiographic criteria for RVH that depended primarily on anteriorly directed forces tended to detect only the more advanced forms of RVH, whereas those criteria that were based on the rightward (although still posterior) direction of the mean spatial vector tended to detect lesser degrees of RVH. Some 30 per cent of his cases, however, were missed by all criteria.

Evans and Short\(^{36}\) have reported the clinical and pathologic features in 11 patients with congenital heart disease and pulmonary hypertension. Only 10 of these, however, had electrocardiograms for comparison with the pathologic findings. Eight cases exhibited RVH in the electrocardiogram and 2 CVH. At autopsy the 8 cases had either isolated or preponderant RVH and the 2 had CVH although the RVH was proportionately greater.

Other studies with anatomic and electrocardiographic correlations have been carried out but sufficient specific details have not been given in these reports to be included in table 2. A brief consideration of some of these studies will now be made.

Thomas\(^{57}\) studied at autopsy 50 hearts from coalminers with pneumoconiosis, employing the method of Herrmann and Wilson\(^{3}\) for dissecting and weighing the right and left ventricles. The total heart weights ranged from 180 to 800 Gm. He considered an LV/RV ratio below 1.40 and above 2.20 as abnormal. While there were many small hearts in the series, right ventricular preponderance by actual weight was present even in the smallest hearts. It was noted that hypertrophy was first evident in the thickening of the muscle around the base of the outflow tract, particularly the crista supraventricularis. Only 18 of these cases had electrocardiograms available and a detailed correlation was not given. Four cases with electrocardiographic patterns of RVH had LV/RV ratios below 1.00 while 2 cases with LV/RV ratios of 1.00 and 1.02 did not display RVH in the electrocardiogram. One of these cases had a small heart, the right and left ventricles each weighing 91 Gm. Thomas suggested that the LV/RV ratio must be below 1.00 before the electrocardiographic pattern of RVH can be expected. He concluded that while there is a great degree of RVH usually found in the advanced cases of pneumoconiosis, the electrocardiographic patterns are a poor index of early RVH.

Fraser and Turner\(^{58}\) have conducted an extensive survey of the electrocardiogram in mitral valve disease including 200 cases in their operation series and 38 cases in their post-

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mortem series. We are here concerned only with the latter group. They encountered 13 patients with anatomic RVH but no evidence of this electrocardiographically. All 14 cases with R/S > 1 in right ventricular leads had anatomic RVH. All 11 cases with R > 7 mm in V1 irrespective of the R/S ratio had marked anatomic RVH. Four of their cases showed a Q in V1, and the right ventricular thickness ranged from 9 to 12 mm. in these cases. Of especial interest in this study was the occurrence of 11 cases of incomplete RBBB and at autopsy all had definite RVH. In this study there were 18 patients in which the thickness of the right ventricle was 8 to 12 mm. and in this group the height of the R wave in leads over the right ventricle varied between 1 and 15 mm. and the R/S ratio from 0.1 to 11.0.

These workers found a striking lack of correlation between the electrocardiographic and anatomic evidence of RVH. They noted particularly the poor correlation between the better developed electrocardiographic signs of RVH and the more pronounced degrees of anatomic RVH. They concluded that electrocardiographic signs in mitral stenosis do not occur until hypertrophy is marked and that in mitral stenosis anatomic hypertrophy is frequently less severe than in congenital heart disease.

Fraser and Turner also discussed some of the difficulties encountered in accurate correlation between electrocardiographic and anatomic findings. They pointed out that the electrocardiographic signs of RVH are not in direct proportion to the thickness of the ventricular wall beneath the exploring electrode. Of considerable interest were the findings of Fraser and Turner of anatomic and electrocardiographic RVH in occasional cases of predominant mitral insufficiency. Studies by Bentivoglio and associates56, 60 have also confirmed the phenomena of RVH in some cases of mitral insufficiency. The RVH has been attributed to disproportionate rise in the left atrial pressure with ensuing pulmonary hypertension and pulmonary vascular changes.59

It thus appears that the accuracy of the electrocardiographic diagnosis of RVH has varied from 97 to 100 per cent in the cases of congenital heart disease45, 47, 56 to a low of 23 per cent in our series of isolated RVH.50 Most workers agree that the electrocardiographic criteria for RVH are more frequently positive in RVH due to congenital heart disease than to acquired heart disease. On the other hand, it has been pointed out that the criteria of Sokolow and Lyon46 are too broad and would include as RVH many instances of normal hearts, especially in children.40, 44 In fact, modified criteria should be used for the diagnosis of RVH in infancy and early childhood.

**Right Ventricle Hypertrophy in Infancy and Childhood**

The electrocardiographic diagnosis of RVH in infancy is difficult because of the normal right ventricular preponderance found in this age group. While it has been generally accepted that the right ventricle in the newborn is of approximately the same or of even greater thickness than the left ventricle, there is considerable lack of agreement as to the exact age when the adult proportions between the right and left ventricles are reached.

Barry and Patten61 stated that after birth the left ventricle begins to assume its characteristic preponderance and by the fourth year of life adult proportions are attained. Lepeschkin62 commented that the right ventricular wall has become normal at the end of the first year, although his graph shows that the major change has occurred by the sixth month. Keen63 found the adult proportions between the left and right ventricle to be present by the third to fifth month although there was a progressive increase in the left-to-right ratio up to 12 months. According to Hollman64 the ratio of the right to the left ventricular thickness is 6 to 7 at birth, after 3 months the left ventricle starts to become dominant, and by the age of 6 years the normal adult left ventricular dominance is largely established. On the other hand Edwards65 stated that by the end of the third postnatal month the adult disproportion between the thickness of the 2 ventricles is established.

Sodi-Pallares and associates66 have pre-
sent recent evidence that in infants there is in fact no right ventricular predominance in the parietal thickness, but that the right ventricular free wall is thinner than the left and there is no great difference between the ventricles as in adults. As age progresses, the increase in thickness is more important in the left ventricular wall than in the right. The electrocardiographic pattern of right ventricular preponderance normally found in the newborn and infant undergoes a gradual transition with increasing age toward the adult type of electrocardiogram, which is characterized by left ventricular dominance.

There is a more or less gradual progression of the mean electrical axis from approximately +120° in the first month of life to between +60° and +90° at the age of 6 months, where it remains throughout the remainder of childhood. The R waves are small in leads I and II during the first month and increase to approximately adult amplitude between the ages of 3 and 6 months. While the older reports concluded on the basis of the standard-lead changes that the adult proportions between the right and left ventricles were reached by about the third to sixth month of life, Ziegler has emphasized that regardless of the extremity-lead pattern no definite conclusion may be made concerning the relative size of the 2 ventricles.

The precordial electrocardiogram in normal infants from birth to 3 years has been shown by Ziegler to include (1) evidence of a normal degree of relative right ventricular preponderance, consisting of large amplitude R waves with late onset of the intrinsicoid deflection in right precordial leads, (2) presence of upright T waves in leads from the right side and inverted T waves in left chest leads during the first 24 hours of life followed by a gradual progression to inverted T waves in right precordial leads and upright T waves in left precordial leads during the subsequent 2 to 4 days. The average amplitude of R in V₁ exceeds that of lead V₆ from birth to approximately 6 months; they are nearly equal from 6 months to 1 year; beyond the age of 1 year the amplitude of R in V₆ exceeds that of lead V₁. The R wave is dominant in V₁ (R/S ratio greater than 1) in the majority of infants during the first 1 to 3 years of life. After the age of 3 the majority of children will show an R/S ratio less than 1 in V₁, although occasionally this may not occur until the age of 5 or even older. The majority of normal children will have a dominant S wave in V₁ by the age of 5. Thus the right ventricular dominance of the normal infant gradually gives way to left ventricular dominance by the age of 3 years although this may be normally delayed in some cases until 5 years or even older.

Lepeschkin has observed that the R/S ratio in precordial leads, the degree of RAD, and the relative thickness of the right ventricular wall show an almost identical course during growth. Ziegler has cautioned against attempting to make too close a correlation between the electrocardiogram and the anatomic measurements in infants and children.

The diagnosis of abnormal right ventricular enlargement in the newborn and the infant under the age of 3 is thus attended by certain difficulties not present in the adult. Nevertheless, electrocardiographic criteria for the diagnosis of RVH in infants have been well defined. The more important of these proposed criteria may be briefly summarized: (1) a single-peaked RS deflection followed by a positive T wave in right precordial leads after the first 48 hours of life; (2) a monophasic R or a qR pattern with an upright (or inverted) T wave in right precordial leads; (3) an rSR' (with R' greater than 10 mm.) or a notched or double-peaked R, the amplitude of which is 85 to 100 per cent of the RS and followed by an inverted T wave in right precordial leads; (4) delay in the onset of the intrinsicoid deflection of 0.03 second or more in right precordial leads; (5) a dominant late R wave in aVR in a vertical heart; (6) RAD of +120° or greater; and (7) so-called "barrage" type of Donzelot with pattern of RVH plus RBBB extending across the entire precordium. Since an upright T wave may occur normally in V₁ at...
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age 12, there is a group between 1 and 12 years of age in whom RVH may be suspected from the presence of an upright T in V1.38

Goodwin70 studied 36 children from 3 to 14 years of age with congenital heart disease and presumed RVH. He suggested that the important signs of RVH are an R/S ratio greater than 1 with a ventricular activation time greater than 0.03 second in V1 and a Q/R ratio less than 1 in aV1r. Three of these cases came to autopsy and RVH was found in each.

Hollman,64 however, pointed out that the R/S ratio in V1 may be over 1.0 in normal children up to the age of 10 years. In infants under 3 months the R/S ratio is normally quite high.64 The R/S ratio in V5 in this age group normally, however, is always 0.6 or greater while in RVH it is 0.5 or less,64 this being of particular value in the diagnosis of RVH in the very young. Hollman64 has proposed criteria for the diagnosis of RVH from 1 month to 15 years of age, which include (1) the presence of a Q wave in V1; (2) onset of intrinsicoid deflection in V1 of 0.04 second or greater in the absence of RBBB; (3) R/S or R/Q in aV1r greater than 1.0; (4) P wave of 3 mm. or more in lead II or 2.5 mm. or more in any other lead; (5) electrical axis of over +120°; and (6) R/S in V1 and V5 which equal or exceed the following ratios:

<table>
<thead>
<tr>
<th>R/S</th>
<th>V1</th>
<th>7.0</th>
<th>4.5</th>
<th>2.5</th>
<th>2.0</th>
<th>1.5</th>
</tr>
</thead>
<tbody>
<tr>
<td>R/S</td>
<td>V5</td>
<td>0.5</td>
<td>0.7</td>
<td>0.8</td>
<td>0.9</td>
<td>0.9</td>
</tr>
</tbody>
</table>

Hollman evaluated the accuracy of these criteria in 29 autopsy-proved cases of isolated RVH in children ranging from 1 month to 15 years. He found 2 or more criteria present in 25 cases (85 per cent). In the remaining 4 cases 1 criterion was satisfied in each and he classified them as having probable RVH.

The rSR' Pattern in Right Precordial Leads

The rSR' pattern in right precordial leads with a QRS duration less than 0.12 second may occur in normal subjects,74,75 in RVH,42,76 in right ventricular dilatation,42,76 in so-called right ventricular diastolic over-

77 load,77 in incomplete right bundle-branch block (IRBBB),79,78 in IRBBB and RVH,74,78 in coronary artery disease,74 and in anterolateral peri-infarction block.31,33

The secondary r wave encountered in some normal subjects has been attributed to normal variation in the order of ventricular activation,75 activation of the pulmonary conus,74 or crista supraventricularis.80 In fact, it has been suggested that in the majority of healthy young adults final activation occurs in the base of the septum or right ventricle.81 The electrocardiographic pattern characterized by late secondary R waves in aV1r and in right chest leads is indistinguishable from or identical with certain types of IRBBB as described by Barker and Valencia78,81 Some workers believe that the rSR' pattern may represent an intermediate stage in the development of electrocardiographic signs of RVH and that it is not necessarily associated with a conduction defect.82,83 Evidence has been presented to suggest the concept that the rSR' observed in atrial septal defect84 as well as in certain other congenital and acquired lesions is due to hypertrophy of the outflow tract or crista supraventricularis rather than to IRBBB.80,85-87

Camerini, Goodwin, and Zoob49 encountered the rSR' in V4R in 5 cases and rSR' in 2 cases of anatomically proved RVH. They considered the rSR' pattern not to be diagnostic while the rSR' pattern was suggestive of RVH. Myers and co-workers42 encountered 9 cases that satisfied their criteria for IRBBB among 40 cases of autopsy-proved RVH. The findings of Fraser and Turner58 have already been cited. Barker and Valencia78 have proposed that in the presence of the pattern they refer to as IRBBB (rSR') if R' is 10 mm. or greater, RVH coexists. In complete right bundle-branch block (CRBBB) if R' is 15 mm. or greater, RVH is also present.88 Caruso and associates46 in their study of 58 cases of isolated RVH proved at autopsy, encountered 24 with IRBBB (rSR'). Of these, only 3 had an R' greater than 10 mm. Milnor and Bertrand83 found in their study of atrial septal defect that in 2 of 5 cases with RSR'
there was isolated RVH while in the other 3 there was CVH. They further observed that conduction delay must play a part in the pathogenesis of the RSR' in V1 in some instances but in the absence of direct evidence, it was not justifiable to assign the site of conduction delay to the "bundle branches."

In a controlled study (but one without autopsy correlation) Dodge and Grant 79 analyzed the electrocardiogram before and after the development of RBBB (QRS duration of 0.12 second or longer in this study). In none of the control tracings was there evidence of RVH. After the occurrence of RBBB, however, these workers found that R' in V1 ranged from 4 to 12 mm. in 90 per cent of their 80 cases and was 20 and 23 mm. in 2 cases. They thus demonstrated the range of deformity that RBBB may produce in the absence of electrical evidence of RVH. They concluded that the magnitude of the deflection of the last 0.04 second must exceed these values in RBBB before the diagnosis of RVH plus RBBB can be made.

Fowler 80 has observed the occurrence of tall secondary R waves in right precordial leads in 5 of 7 cases that developed induced complete RBBB as a complication of right heart catheterization. The R' in V1 ranged from 12 to 20 mm. in height and was 15 mm. or more in 3 instances. In all of these cases, the RBBB disappeared within 24 hours and in none was there any electrocardiographic evidence of RVH either before or after the RBBB.

Vector studies of the rSR' pattern have shown what Grishman and associates 80-88 believed to be a differential pattern in the horizontal plane: namely, clockwise rotation with a slowly inscribed terminal phase indicating RVH with RBBB. More recent studies, however, suggest that there are no loop rotations that may be considered as exclusively representative of RBBB.

Booth, Chou, and Scott 85 evaluated the accuracy of the electrocardiographic diagnosis of ventricular hypertrophy in the presence of RBBB in a series of 49 cases, correlating the electrocardiogram with the postmortem findings. The electrocardiographic diagnosis of RBBB was made by conventional criteria including a secondary R wave in right precordial leads exceeding the initial R wave in height. The block was deemed complete if the QRS duration was 0.12 second or greater and incomplete if the QRS duration measured 0.08 to 0.11 second, inclusive. The tracings were analyzed as to the presence of associated RVH by the criteria of Barker and Valencia. 78 The electrocardiographic diagnosis of LVH was made by means of current criteria. 27 In this series of 49 cases, unselected as to anatomic diagnosis, 4 (8 per cent) had isolated RVH, 15 (30.5 per cent) had isolated LVH, 22 (45 per cent) had CVH, 6 (12.5 per cent) had normal hearts, and 2 had increased heart weight but normal ventricular wall measurements.

The correlation of the electrocardiograms with the autopsy findings is shown in table 3. Twenty-six of our cases of IRBBB were analyzed according to Milnor's criteria 44 for the diagnosis of associated RVH. Fifteen of the 26 met Milnor's criteria for RVH on the electrocardiogram. The diagnosis was correct in 9 (60 per cent) and erroneous in 6 (40 per cent). In addition 7 cases of anatomic CVH were missed as was 1 case of isolated RVH. A total of 11 cases met the criteria of Barker and Valencia 78 for associated RVH. The right ventricle was enlarged in 7 of these cases (63.5 per cent) although in 4 of these LVH was also present. Unexpected findings in this study were the high incidence (37 cases) of anatomic LVH and the consistency with which the diagnosis was missed in the electrocardiogram. The electrocardiographic diagnosis of LVH was obscured in all by the RBBB. One case that fulfilled the electrocardiographic criteria for LVH revealed isolated RVH at autopsy. The results of this study are in agreement with Myers, 12, 76 who indicated that RBBB is associated with enlargement of the left ventricle, either alone or in combination with RVH, in too many cases to render it a significant sign of an isolated right ventricular lesion.

An attempt was made in this study 95 to correlate the electrocardiographic diagnosis

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Table 3
A Correlation of the Electrocardiographic Diagnosis with the Postmortem Findings in Forty-nine Cases of Right Bundle-Branch Block

<table>
<thead>
<tr>
<th>Electrocardiographic diagnosis</th>
<th>RVH</th>
<th>LVH</th>
<th>CVH</th>
<th>NVH</th>
<th>IWNM</th>
</tr>
</thead>
<tbody>
<tr>
<td>IRBBB (28 cases) plus</td>
<td>8</td>
<td>2</td>
<td>1</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>18</td>
<td>0</td>
<td>5</td>
<td>11</td>
<td>1</td>
</tr>
<tr>
<td>CRBBB (21 cases) plus</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>18</td>
<td>0</td>
<td>7</td>
<td>7</td>
<td>4</td>
</tr>
<tr>
<td>Total</td>
<td>49</td>
<td>4</td>
<td>15</td>
<td>22</td>
<td>6</td>
</tr>
</tbody>
</table>

IRBBB, incomplete right bundle-branch block; CRBBB, complete right bundle-branch block; RVH, right ventricular hypertrophy; LVH, left ventricular hypertrophy; CVH, combined ventricular hypertrophy; NVH, no ventricular hypertrophy; IWNM, increased heart weight with normal ventricular measurements.

The electrocardiographic diagnosis of CVH is ordinarily based upon the conventional criteria for isolated LVH and RVH in the precordial and unipolar extremity leads. In addition, RAD greater than +90°, marked "clockwise" rotation, and signs of RVH and LVH in the precordial leads. Only 4 of their cases satisfied these criteria. Levine and Phillips concluded that CVH is rarely detected in the electrocardiogram.

Pagnoni and Goodwin found that direct evidence of RVH (increased ventricular activation time and R>S in V1) was masked by LVH. These workers considered an R>Q in aVR, S>R in V3 with T-wave inversion in V1, together with signs of LVH, diagnostic of CVH, while the association of a vertical heart position with signs of LVH were highly suggestive of CVH. Sokolow and Edgar observed that when CVH was present anatomically, the electrocardiogram usually reflected the change in the ventricle with the major hypertrophy. They further found that when ventricular hypertrophy was minimal, a normal or borderline electrocardiogram was occasionally found. Lipsett and Zinn in a study of 73 patients with necropsy evidence of CVH found only 10 cases with electrocardiographic evidence of CVH and concluded that the diagnosis is difficult. They found that RVH resulting from cor pulmonale could be diagnosed more frequently than RVH resulting...
Table 4

<table>
<thead>
<tr>
<th>Author</th>
<th>Number of cases with anatomic CVH</th>
<th>ECG diagnosis</th>
<th>Accuracy of ECG diagnosis of CVH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Soulie et al.</td>
<td>27</td>
<td>4</td>
<td>15</td>
</tr>
<tr>
<td>1949</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Levine and Phillips</td>
<td>26</td>
<td>2</td>
<td>12</td>
</tr>
<tr>
<td>1951</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pagnoni and Goodwin</td>
<td>51</td>
<td>13</td>
<td>13</td>
</tr>
<tr>
<td>1952</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lipsett and Zinn</td>
<td>73</td>
<td>10</td>
<td>21</td>
</tr>
<tr>
<td>1953</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Levine and Whipple</td>
<td>8</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>1955</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fraser and Turner</td>
<td>22</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>1955</td>
<td></td>
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</tr>
</tbody>
</table>

CVH, combined ventricular hypertrophy; LVH, left ventricular hypertrophy; RVH, right ventricular hypertrophy.

from rheumatic or hypertensive heart disease when LVH was also present. They related this to the more significant RVH present in their cases with cor pulmonale. Whipple and Levine69 studied the electrocardiogram (and vecorecardiogram) in 9 cases of CVH proved at autopsy. It is of interest that they observed while LVH in a vertical heart occurred in 2 instances, there were no cases in which the precordial transition zone shifted significantly or prominent R waves in aVp occurred. Fraser and Turner58 encountered 22 cases of anatomic hypertrophy of both ventricles in their study of mitral valve disease. These workers found that considerable anatomic biventricular hypertrophy may exist in association with electrocardiograms showing RVH alone (11 cases) or without evidence of either RVH or LVH (6 cases). In 2 cases with electrocardiographic evidence of LVH alone the anatomic LVH was proportionately greater than the RVH, and the former seemed to mask the electrocardiographic signs of RVH. They suggested that from the electrical point of view RVH and LVH may "balance" each other and no ventricular hypertrophy be evident in the electrocardiogram.7 They found, however, no obvious relationship between the degree of hypertrophy in the ventricles and the presence or degree of electrocardiographic signs of biventricular hypertrophy. They concluded that electrocardiographic signs of CVH are uncommon in patients with rheumatic heart disease, even though such hypertrophy may be marked at autopsy.

Ventricular Hypertrophy in the Presence of Left Bundle-Branch Block

The electrocardiographic diagnosis of LVH in the presence of complete left bundle-branch block (LBBB) has been considered difficult, if not impossible,88 although some workers8 believe that this diagnosis can be made. We have recently studied this problem in our laboratory and compared the accuracy of the electrocardiographic diagnosis with the pathologic findings.100 All cases with the diagnosis of LBBB in which an autopsy had been performed were collected. All cases with pathologic evidence of myocardial infarction were excluded. The conventional electrocardiographic criteria of high voltage for LVH were employed and the pathologic criteria for ventricular hypertrophy have already been defined.

Twenty-eight cases of LBBB were studied. At autopsy all cases had cardiac hypertrophy. Only 10 of the 28 cases showed electrocardiographic evidence of LVH. At autopsy 3 of these had pure LVH and 7 had CVH. Eight cases of pure LVH at autopsy showed no electrocardiographic evidence of LVH. It would appear on the basis of this study that the precise electrocardiographic diagnosis of LVH in the presence of LBBB is difficult and unreliable. Massive RVH and LBBB rarely occur together, according to Wilson.20 We are planning to investigate this uncommon combination of findings.

Ventricular Hypertrophy and Myocardial Infarction

Myers101,102 has emphasized that ventricular hypertrophy occasionally presents electrocardiographic signs of LVH alone. Other workers, however, have concluded that RVH and LVH do not "balance" out to give a normal electrocardiogram.
cardiographic patterns that may mimic those of myocardial infarction. He presented 10 cases of LVH\textsuperscript{101} in which no evidence of infarction was demonstrated at autopsy, yet one or more of the following electrocardiographic features were encountered which suggested infarction: (1) deep Q waves, marked ST-segment depression, or sharp inversion of the T waves in left ventricular leads; (2) abnormal Q waves, bizarre ST-segment shifts, or cove plane inversion of the T waves in the transitional zone; (3) QS pattern or abnormal S-T elevation in right precordial leads.

In a related study Myers presented 15 cases proved at autopsy of RVH or right ventricular dilatation without myocardial infarction.\textsuperscript{102} The electrocardiograms in these cases presented one or more of the following features that resembled a pattern of myocardial infarction: (1) abnormal qR or QS patterns or sharp T-wave inversion in right precordial leads; (2) reduction in amplitude of the R waves or replacement by a QS deflection or change from an upright to an inverted T wave as the transition zone was reached; (3) persistence of normal Q waves in left ventricular leads accompanied by marked reduction in the R wave and exaggeration of the S wave.

Goodwin\textsuperscript{103} has studied the relationship between ventricular hypertrophy and myocardial infarction. His material was highly selected in that he included only those cases that had evidence at autopsy of myocardial infarction, RVH or CVH, and whose electrocardiograms displayed predominantly negative deflections in V\textsubscript{5} (rS, Qr, QS, or qrS). His 85 cases were grouped as isolated hypertrophy, isolated infarction, or both hypertrophy and infarction. In analyzing his tracings for distinguishing features, he found that the classic pattern of wide deep Q waves with S-T elevation and T-wave inversion in V\textsubscript{5} was encountered in infarction with or without hypertrophy but in only 1 case of isolated RVH. On the other hand the rS pattern in V\textsubscript{5} occurred in isolated infarction, isolated hypertrophy, or in both hypertrophy and infarction. Features favoring infarction included diagnostic evidence in other leads, a negative T wave in V\textsubscript{5}, a positive T wave in V\textsubscript{1}, or S-T elevation greater than 2 mm. By contrast, a vertical heart position and "right atrial" P waves (pointed P waves of 2.5 mm. or more) suggested isolated or dominant RVH. Right atrial P waves were not encountered in any case of infarction, and a vertical heart was only seldom encountered in infarction (12 per cent) while rather commonly encountered in the cases of hypertrophy without infarction (66 per cent).

Goodwin\textsuperscript{103} pointed out that while the commonest cause of a deep S wave in V\textsubscript{5} is isolated or dominant RVH, it may also occur in anterior infarction with or without RVH. Thus, any electrocardiogram showing the rS pattern in V\textsubscript{5} should be interpreted with caution and the possibility of a concealed infarct considered. On the basis of this study he also concluded that the classic patterns of anterior infarction (QS with S-T elevation and T-wave inversion) were less common when RVH was present than when infarction occurred in the absence of RVH. The most important factors concerned with the production of the rS pattern in anterior infarction were thought to be associated RVH and changes in the healing of the infarct.

Burch and associates\textsuperscript{104} have recently reported a correlative study of the electrocardiogram (and spatial vectorcardiogram) with the findings at autopsy in 59 patients with myocardial infarction. Of 33 cases with posterior myocardial infarction, 18 had proved ventricular hypertrophy. The 1 case with anatomic RVH showed electrocardiographic RVH; 6 cases with anatomic LVH showed electrocardiographic LVH in 4; 11 cases of anatomic CVH showed 1 with electrocardiographic CVH, 7 with electrocardiographic LVH, and 1 with RBBB. There were 2 cases in this group of posterior infarction with the electrocardiographic criteria of RVH without anatomic evidence of RVH.

Of 26 cases with anterior myocardial infarction 21 had anatomic ventricular hypertrophy. One case with anatomic RVH displayed electrocardiographic RVH; 7 cases
with anatomic LVH displayed electrocardiographic LVH in 3; 13 cases of anatomic CVH showed 2 with electrocardiographic CVH, and 5 with electrocardiographic LVH. There was 1 instance in this group with anterior infarction of a false diagnosis of LVH. The anterior infarct was missed electrocardiographically in 1 of the hearts with ventricular hypertrophy.

These authors concluded that the diagnosis of ventricular hypertrophy by the electrocardiogram was unreliable. They attributed this in part to the high incidence of combined hypertrophy with the electric effects of one chamber tending to neutralize the other. They also called attention to the observations of den Boer,105 that the difficulty in distinguishing LVH from anterior myocardial infarction may be due to the fact that in both conditions the resultant of the electric dipoles points backward and to the left.

**Electrophysiologic and Anatomic Considerations**

**General**

The exact electrophysiologic mechanisms that are responsible for the electrocardiographic phenomena that occur in ventricular hypertrophy are not entirely clear.7, 8, 20 Hypertrophy results in an increase in the ventricular muscle mass and in the length and thickness of the wall of the involved chamber.7, 20 There is also an increase in the total epicardial surface area and an increase in size of the individual muscle fibers.29 Perhaps there may be a diminution in the density of the juncture between the Purkinje fibers and the subendocardial muscle, as well as an increase in the path traversed by the excitation wave.20 It has also been suggested that the myocardial fibrosis which may occur in ventricular hypertrophy may perhaps produce an alteration in the more peripheral parts of the conduction system.31

An increase in the number of activation dipoles with oblique spread of the epicardial QRS vector,7, 106 increased voltage generated by the individual, hypertrophied muscle fibers,29 closeness of the hypertrophied and frequently dilated ventricular chamber to the chest wall,8 and increased surface area with increase in magnitude of vectors generated from this region7 have all been proposed as explanations for the increase in voltage that accompanies ventricular hypertrophy.

The increase in voltage, the increase in duration of the QRS complex, and the delay in the onset of the intrinsicoid deflection are ordinarily attributed to delay in the transmyocardial excitation wave because of increase in muscle mass and thickness of the free ventricular wall.7, 8, 107 So-called parietal wall conduction defects of a minor degree may play a role in some cases.31 The increased thickness and mass of the hypertrophied ventricle may permit it to generate forces that abnormally overbalance as well as outlast the forces produced by the opposite ventricle.20

The S-T depression and T-wave inversion that often accompany increased voltage of the QRS complex in ventricular hypertrophy have ordinarily been attributed to the increased thickness of the muscular walls8 with altered direction of recovery. These S-T segment and T-wave changes have been considered by some to be secondary to the large QRS complexes, while these alterations have been considered by others actually to represent primary changes due to myocardial disease.8, 20

While anatomic rotation of the heart has been invoked for various electrocardiographic changes encountered in ventricular hypertrophy,8, 25, 62, 88 Grant has shown in a careful anatomic-electrocardiographic study that there is rarely more than a 20° variation in the anatomic long axis of the left ventricle in either the normal subject or the subject with marked RVH or LVH, regardless of body build.7, 18

**Left Ventricular Hypertrophy**

The genesis of the electrocardiographic patterns that occur in LVH is still obscure. As already indicated, various explanations have been offered for the electrocardiographic changes encountered in ventricular hypertrophy but the precise electrophysiologic mechanisms must await further investigation.
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However, it may be well to review briefly some of the more cogent, currently accepted views as they apply to LVH.

First, however, let us consider the anatomic alterations that occur in LVH. It is well established from pathologic studies\textsuperscript{1-7, 108} that there is an absolute increase in muscle bulk or weight of the left ventricle, there is an increase in thickness of the free wall as well as of the interventricular septum, and an elongation of the left ventricular chambers. Furthermore, it has been demonstrated\textsuperscript{108} that the diaphragmatic and anterior portions of the left ventricle do not change appreciably in contour with the development of LVH, presumably because they are limited by the diaphragm and the outflow tract of the right ventricle respectively. However, the posterior and superior surfaces of the left ventricular wall are unrestrained and show the most marked alteration, becoming bowed and elongated.\textsuperscript{7, 108} In LVH the internal architecture of the left ventricle is not significantly altered\textsuperscript{33} aside from hypertrophy of the papillary muscles and the trabeculae carneae. Histologically there is an increase in size of the individual muscle fibers although a moderate degree of hypertrophy is required before it is apparent microscopically.\textsuperscript{109} In myocardial hypertrophy there is apparent failure of the blood supply to parallel the growth of the muscle fibers. The ratio of 1 capillary to 1 muscle fiber in the adult persists, no matter how large the heart becomes, producing what probably is relative coronary insufficiency in the enlarged heart.\textsuperscript{12, 110, 111} Some cases of LVH show a considerable increase in the interstitial connective tissue.\textsuperscript{33, 88}

When hypertrophy produces a symmetrical thickening of the wall of the left ventricle, it is ordinarily referred to as "concentric hypertrophy."\textsuperscript{112, 108} When hypertrophy becomes marked or is complicated by dilatation, the apical portion of the left ventricular chamber becomes thinner than the wall at the base. This condition is referred to as "eccentric hypertrophy."\textsuperscript{112, 108} When dilatation of the left ventricle occurs it has also been observed to involve more markedly the portions of the chamber that lie in front of the anterior leaflet of the mitral valve; thus the outflow tract and superior wall of the left ventricle may be more markedly stretched and bowed than the inflow tract and inferior wall.\textsuperscript{108}

The conventional electrocardiographic criteria for LVH include increase in voltage (tall R waves in left precordial leads, deep S waves in right precordial leads, tall R waves in aV\textsubscript{1}, in horizontal hearts, tall R waves in aV\textsubscript{P} in vertical hearts), delay in ventricular activation time in left precordial leads, slight increase in width of the QRS interval, and ST-segment and T-wave displacement opposite in direction to the major QRS deflection. While LAD may occur in LVH with tall R waves in lead I and deep S waves in lead III, normal axis deviation is common, and even RAD may be encountered.

Many explanations have been advanced for the genesis of the electrocardiographic alterations that occur in LVH. These have been well presented in several publications.\textsuperscript{8, 33, 62, 88} It will suffice here to summarize the more likely explanations.

The prolongation of the QRS as well as the delay in ventricular activation time is ordinarily attributed to the increased thickness of the left ventricular wall with the consequent increase in time for the activation process to spread through the myocardial wall. Wilson and Herrmann\textsuperscript{107} demonstrated many years ago that there was a relationship between increasing ventricular weight and thickness of the left ventricular wall and increasing duration of the QRS interval. These workers, however, believed that an increase in the QRS interval beyond 0.10 second should not in general be ascribed to increased size of the heart or increased thickness of the left ventricle alone but to an intraventricular conduction defect. This has also been affirmed by Bayley.\textsuperscript{112}

The propagation of excitation over the Purkinje network was estimated by Lewis and associates\textsuperscript{115-115} to be 4,000 mm. per second while in the ventricular walls only 400 mm. per second. More recent studies,\textsuperscript{8, 116, 117} however, give the rate of Purkinje propagation at
about 1,000 mm. per second and ventricular wall conduction at about 300 mm. per second. It would appear that marked increase in thickness of the ventricular musculature would result in slower transmyocardial spread of the activation wave. The exact depth of penetration of the Purkinje network into the myocardial wall is unsettled but it has been suggested that these fibers penetrate deeply into the muscular wall of the left ventricle,\textsuperscript{8, 118} although this has never been demonstrated anatomically in the human heart.\textsuperscript{119} In fact, Sodi-Pallares\textsuperscript{8} stated that the rate of conduction through the intramural muscle of the ventricular wall is not related to the thickness of the muscle but is rather a function of the greater or lesser penetration of the Purkinje network into the depths of the muscle. It has been pointed out\textsuperscript{116, 117} that in the peripheral and particularly in the basal parts of the endocardium the Purkinje fiber may be relatively unbranched or sparse.

It has been suggested that in LVH the Purkinje network does not keep pace with the increasing thickness of the left ventricular wall and does not penetrate as deeply as in the normal, proportionately, so that more of the spread of activation is through the more slowly conducting muscle cells. Another possible cause of the delay in intraventricular conduction is that, with the left ventricular enlargement, there is spreading out of the Purkinje arborization. This results in a decrease in density of the Purkinje network per unit volume of muscle, and the impulse does not reach all regions of the endocardial surface quite so rapidly because it must travel through the enlarged fibers lying between the Purkinje junction.\textsuperscript{88}

As the muscle fibers enlarge, they tend to outgrow their blood supply. The ratio of 1 capillary to 1 muscle fiber in the adult has been demonstrated to remain throughout life. Thus, in an enlarged heart there is a decrease in the number of capillaries per unit volume of myocardial tissue, which results in relative coronary insufficiency.\textsuperscript{12, 62, 110} Some of the muscle fibers die and are replaced by fibrosis.\textsuperscript{88} This replacement fibrosis of the myo-
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may be a decrease in the volume of the poorly conducting lung between the heart and the chest wall. Hypertrophy of the ventricular muscle also increases its area in relation to the area of short circuiting fluids surrounding the heart and contained in its cavities.

Hypertrophy of the individual muscle fibers results in an increase in their cross sectional diameter. This change results in a decrease in the internal resistance while not altering the external resistance. This decrease in turn tends to increase the voltage generated by them on excitation.

A recent study, however, casts some doubt on this last hypothesis. Uhley measured the transmembrane action potentials of the single cell in the hearts of normal control rats and in those with LVH secondary to induced hypertension. He found no significant difference in the cell potential between the 2 groups and concluded that increased voltage found in the electrocardiogram with LVH patterns is not the result of changes in single-cell generator amplitudes.

In the normal heart, the forces generated by the free wall of the left ventricle normally outlast those produced by the free wall of the right ventricle. The last portion of the left ventricle to be activated is the posterobasal region of the free wall. The terminal QRS forces therefore tend to be directed posteriorly and somewhat leftward.

In LVH the sequence of ventricular activation is the same as in the normal. In LVH it has been demonstrated by direct epicardial leads that there is delay in activation of the left ventricular surface, especially in the most basal and posterior regions. This delay accounts for the posterior and superior deviation of the terminal vectors in LVH. In other words, the ventricular activation in LVH is basically an exaggeration of the normal with the terminal forces having a more posterior and superior direction. The mean QRS vectors are increased in magnitude but not greatly altered in direction. The mean spatial QRS vector in LVH has the direction of a line from the center of the heart toward the free wall of the left ventricle. This vector may or may not undergo a directional rotation.

Dilatation of the left ventricular chamber may occur alone or in combination with LVH. Dilatation tends to lengthen the endocardial pathway traversed by the excitation wave. It also may result in further diminution of the density of the Purkinje fiber network. A further effect of dilatation may be to produce stretching of the conduction system, which reduces its cross sectional diameter and may cause direct mechanical injury resulting in a conduction disturbance. Dilatation also produces an increase in the epicardial surface area of the left ventricle and the free wall is carried closer to the chest wall. These features may result in QRS prolongation and increased amplitude.

Dilatation, however, results in an increase in the intracardiac blood volume, which acts as a shunt for the cardiac action currents and decreases the voltage of the electrocardiogram. Congestive failure is a common accompaniment of left ventricular dilatation, and edema of the lungs also serves to short-circuit the flow of current in the cardiac field and decrease the voltage. Another possible cause for diminished voltage in left ventricular dilatation is the damage and destruction of muscle fibers that may be present. The net effect of dilatation upon the electrocardiographic pattern is therefore difficult to predict. Some workers believed that the QRS changes attributed to LVH reflect only an increase in ventricular size whether due to hypertrophy, dilatation, or both. Barker suggested, however, that left ventricular dilatation is seldom present without antecedent LVH.

Increase in the depth of the Q waves in leads overlying the left ventricle have been attributed to hypertrophy of the interventricular septum with increase in the electromotive forces appearing during its activation from left to right. Grant however, has presented evidence that casts some doubt upon the validity of the concept of a septal Q wave.

The ST-segment and T-wave alterations
that occur in LVH may be secondary to the large QRS areas. It is thought that these alterations are related to the early onset of repolarization in the subendocardial region before activation has reached the epicardial surface.\textsuperscript{88} It has been postulated\textsuperscript{33} that the direction of repolarization in the normal heart is due to a gradient of pressure across the ventricular wall, the intramyocardial pressure being greater in the endocardial layers. Disappearance of this gradient in LVH has been offered as an explanation for the reversal of repolarization.\textsuperscript{33} Objection, however,\textsuperscript{121} has been raised to this latter explanation. Hypoxia of the endocardial layers, due to increasing intramyocardial pressure that opposes capillary blood flow, is thought not to be responsible for the direction of repolarization in the normal heart.\textsuperscript{126} The role of the temperature gradient in the process of repolarization in the normal heart is still not clearly defined.\textsuperscript{126} In these so-called secondary T-wave changes of LVH, the ventricular gradient is normal. In some cases of marked LVH the T-wave changes have also been considered to be secondary to a parietal wall conduction defect in the left ventricle.\textsuperscript{33} Primary T-wave changes with an abnormally directed ventricular gradient may develop in LVH and are generally considered to be due to myocardial ischemia or coronary insufficiency.\textsuperscript{33, 88, 121, 127}

It is probable that some tracings that have been regarded as characteristic of LVH are examples of incomplete LBBB.\textsuperscript{8, 81, 121} Wilson\textsuperscript{29} pointed out that incomplete LBBB can be excluded when there is a Q wave in one or more leads from the left precordium, but that the absence of Q waves is of no help. The electrocardiographic differentiation of incomplete LBBB from LVH may at times be difficult or impossible. Rasmussen and Moe\textsuperscript{128} suggested that the electrocardiographic patterns in many patients with LVH are due to retarded conduction in the left heart. Grant and Dodge,\textsuperscript{129} however, maintained that incomplete LBBB is exceedingly uncommon as a stable form of QRS prolongation.

The term left ventricular "strain" has become ambiguous. While some authors use the term synonymously with LVH,\textsuperscript{16, 19, 130} others reserve it for the S-T depression and T-wave inversion in left ventricular leads without increased voltage of the R waves. Still others use the term "LVH and strain" when both high voltage of the R waves and the characteristic ST-T changes are present. Grant\textsuperscript{33} stated that left ventricular strain should be diagnosed only when the S-T and T-vector changes are accompanied by increased magnitude of the QRS vector. Objection to the use of the term "strain" has been raised\textsuperscript{20, 33, 121} on the grounds that the term is obscure, that it gives the connotation of a struggling weak heart about to fail, and that it introduces a mechanical term for an electrical phenomenon. Furthermore, similar S-T and T-wave changes may be caused by a multiplicity of factors, such as ischemia, digitalis, electrolyte alterations.\textsuperscript{33, 131} Therefore, the term "strain" is not specific. As Kossmann\textsuperscript{131} has pointed out, alterations in the S-T segment and T waves alone can scarcely be taken as indicative of anything anatomic.

\textit{Causes of False-Positive and False-Negative Diagnoses of Left Ventricular Hypertrophy}

As has already been pointed out, the diagnosis of LVH can be reasonably accurately made from the clinical electrocardiogram. However, certain features must be taken into account and deserve emphasis. The age of the patient is important. In infants, children, and young adults, the voltage, particularly in the precordial leads, normally is greater than in older adults.\textsuperscript{22, 33, 37, 131, 132} Therefore, different criteria for the amplitude of the QRS complexes must be employed for the various age groups. In infants and children the voltage criteria generally accepted for the diagnosis of LVH\textsuperscript{38} exceed the maximum normal for their age.\textsuperscript{37}

Grubschmidt and Sokolow\textsuperscript{132} evaluated high voltage of the QRS (as defined by Sokolow and Lyon\textsuperscript{22}) as the sole manifestation of LVH in a clinical study of 101 cases. They concluded that in adults over the age of 25, it is
a reliable sign. However, in persons between 20 and 25 years, they stated that the sum of R in V3 or V6 and the S in V1 should be considered high voltage only when it exceeds 40 mm. rather than 35 mm.

Aside from age, the individual’s body build should be taken into consideration. Thin-chested or emaciated individuals with normal hearts may have high voltage in the precordial leads that would ordinarily satisfy the criteria for LVH. Conversely, obese individuals or thick-chested individuals may have normal or even low-voltage QRS complexes, despite anatomic LVH.

Pathologic conditions such as myocardial infarction, congestive heart failure, pericardial effusion, pleural effusion, anasarca, and pulmonary emphysema may all serve to reduce the amplitude of QRS voltage in proved cases of LVH.

Of interest is the demonstration that administration of potassium salts to patients with hypertensive heart disease and LVH resulted in a reduction in the QRS voltage. Dietary restriction of sodium and sympathectomy have also been shown to effect a reduction in QRS amplitude, which has not always been correlated with blood pressure reduction or change in heart size. These observations suggest that factors other than hypertrophy may contribute to the high voltage of the QRS complexes.

Bryant observed that incomplete LBBB may at times be confused with the pattern of LVH, or so-called left ventricular strain. He pointed out that the R wave in right chest leads and the Q wave in left chest leads may be absent or unusually small in some electrocardiograms that otherwise indicate LVH, and suggested that this may be due to incomplete LBBB. Conversely, some of the instances of false-positive diagnoses of LVH with delay in the onset of the intrinsicoid deflection may, in fact, be examples of incomplete LBBB.

Certain cases of LVH show a discrepancy between the voltages in the limb and precordial leads. When the mean QRS vector is directed markedly posterior, its projection on the frontal plane is foreshortened, with the result that the limb leads exhibit normal or even low-voltage QRS complexes while the precordial leads have high voltage and a transition zone displaced to the left. On the other hand, when the mean QRS vector is directed markedly leftward and parallel with the frontal plane, the transitional pathway for this vector passes through or near the precordial lead positions and small QRS complexes result while the limb leads display increased voltage of the QRS complexes.

A false-positive diagnosis of LVH can occur with mitral insufficiency and a giant left atrium.

**Right Ventricular Hypertrophy**

In the normal adult heart the free wall of the left ventricle is approximately 3 times the thickness of the right ventricular wall. The normal electrocardiographic pattern is dominated largely by the activation of the left ventricle. When LVH occurs the result is ordinarily an accentuation of the normal configuration of the ventricular complexes.

Lesser degrees of RVH produce no characteristic features in the electrocardiogram. In fact, the right ventricular wall may increase to double normal thickness, or even more, and still not produce sufficient increase in potential to counterbalance that produced by the normal free wall of the left ventricle. Since only seldom in acquired heart disease does the thickness of the right ventricle equal and, even more rarely, exceed that of the left ventricle, it is perhaps even more surprising that the electrocardiographic diagnosis of RVH can be made with as much certainty as has been possible. In congenital heart disease the degree of RVH is usually more marked than in acquired RVH of the adult, and therefore ordinarily produces more marked electrocardiographic evidence of RVH.

In the normal heart the right ventricle lies anteriorly to the left ventricle. The plane of the interventricular septum is relatively parallel to the frontal plane of the body. When RVH develops, there is an increase in the thickness of the free wall as well as an
increase in the epicardial surface area. As the hypertrophy progresses, the anterior surface of the right ventricle obliterates the retrosternal space and then comes to lie against the posterior aspect of the sternum and anterior chest wall. These rigid structures tend to limit any further forward enlargement of the right ventricle, so that any additional right ventricular enlargement must push the heart backward.88

With right ventricular hypertrophy there is also hypertrophy of the trabeculae carneae connecting the free wall of the right ventricle to the septal surface.13, 108 Because of these hypertrophied trabeculae carneae the capacity of the hypertrophied right ventricle may not be significantly increased. With the onset of right ventricular dilatation, however, the trabeculae carneae network connecting the septum with the free wall becomes attenuated and appears to shift laterally. The capacity of the right ventricular chamber increases. This increase in effective inner surface area may be accomplished with little apparent change in outer contour of the heart.108 Further enlargement of the right ventricle must take place by enlargement of the free wall because the septal surface is fixed by the size of the left ventricle. As dilatation continues, first the inflow and then the outflow tracts enlarge. With marked RVH and dilatation the right ventricle extends beyond the septal margin and the heart comes to have a "double apex."1108 Of particular interest is the observation that in some cases of RVH due to congenital heart disease there is an actual increase in the number of myocardial fibers due presumably to hyperplasia occurring during the first few months after birth. This is in contrast to the finding in acquired RVH in which there is no increase in the number of myocardial fiber units.108

Grant33, 108 believed that when LVH is marked, RVH is a regular accompaniment. The explanation offered is that, since the interventricular septum consists almost entirely of left ventricular fibers, any increase in the size of the left ventricle automatically increases the septal surface of the right ventricle and, therefore, of the free wall of the right ventricle. Jones4 however, found in his autopsy study that in hypertension, in the absence of failure, only the left ventricle hypertrophies but with development of congestive failure, the right ventricle hypertrophies progressively with the duration of failure. Similar observations have been made by Fulton.6

The electrocardiographic patterns of RVH occur less frequently than do the patterns of LVH and are also subject to greater variation.9 The QRS duration is not ordinarily prolonged because the pathway of activation through the right free wall is not, except in extreme RVH, as long as it is in the left free wall.98 Activation of the right ventricle is normally completed in about 0.06 second or less. Therefore, a normal QRS of 0.09 would not become prolonged until marked RVH had developed. In fact, ordinarily in uncomplicated RVH, the QRS interval should not be prolonged beyond 0.10 second.121

The genesis of the increased voltage in the right precordial leads in RVH may be similar to that already discussed as causative in the high voltage in left ventricular leads in LVH. These factors include increase in thickness of the right ventricular wall, increase in the epicardial surface area of the right ventricle, proximity of the hypertrophied and often dilated right ventricle to the anterior chest wall, together with decrease in amount of intervening lung tissue, and decrease in internal resistance of the hypertrophied muscle fibers in the right ventricular wall.

The mean spatial QRS vector tends to increase in magnitude and to point more directly toward the hypertrophied free wall of the right ventricle.121 Since the right ventricle lies anteriorly and slightly to the right of the left ventricle, the QRS forces developed from it normally are directed rightward and somewhat anteriorly.33 When RVH develops these rightward and anteriorly directed forces are increased in magnitude. This change results in right axis deviation (RAD) of the mean QRS vector, which has been stated to be the most common manifestation of RVH.33

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This mechanism presumably plays a prominent role in the RAD due to physiologic hypertrophy of the right ventricle in infants.82 It has been pointed out, however,33 that the right ventricle normally contributes very little to the QRS complex and even marked RVH influences the direction of the mean QRS vector only slightly.33 RVH results in marked RAD only in those subjects (children and young adults) where the mean QRS axis normally is more or less vertically directed. In older individuals whose normal mean QRS vector tends to be more horizontal, RVH much less commonly produces marked RAD.33 Conduction delay in the right ventricle has also been implicated as a cause of RAD as it permits the electrical forces of the lateral wall of the right ventricle to be virtually unopposed by those of the left ventricle.62 Dilatation of the right ventricle has been considered to cause clockwise rotation of the heart about its long axis and thereby also produces RAD.62 Of course, Grant’s studies7 deny the importance of anatomic rotation of the heart in the genesis of the electrocardiographic pattern of RVH.

The anteriorly directed initial forces in RVH are responsible for the increased amplitude and duration of the R wave in the right precordial leads. When the terminal forces in RVH become anteriorly directed, there results an R’ in the right precordial leads. If the terminal vector is directed rightward and superiorly, with no prolongation of the QRS, the so-called S1, S2, S3 syndrome is produced and is due to a conduction variation in the right ventricle.33 If the terminal vector is inferiorly directed (as well as rightward and anteriorly) an S in lead I and an R’ in V1 are present. As has already been discussed, this last pattern would be classed by many as IRBBB.

The S-T and T-wave alterations that may occur in the right precordial leads in RVH are generally considered to be secondary to the QRS abnormalities.88 In marked RVH the S-T and T vectors are directed leftward and posteriorly,33 opposite to the direction of the mean QRS vectors. Thus, the S-T segments tend to be depressed and the T waves inverted in leads II and III.

The precordial lead changes in RVH may be grouped into 4 main types, based principally upon changes in the right precordial leads.8, 62 The first group displays tall R waves (with small or absent S waves) with late onset of the intrinsicoid deflection. It has been suggested that this pattern may be attributed to slow spread of excitation in the hypertrophied wall of the right ventricle.62 Sodi-Pallares8 attributed this type of pattern to clockwise rotation of the heart about its longitudinal axis, so that the thickened free wall of the right ventricle lies relatively close to the anterior chest wall. This type of pattern is commonly encountered in pulmonary stenosis and is due to concentric RVH.134

A second type also presents increased voltage of the R waves in V1 but is preceded by a Q wave. The genesis of this qR pattern in right precordial leads in RVH has been and remains a somewhat confused problem. The proposed explanation135 of marked clockwise rotation about the longitudinal axis, so that right precordial leads are facing the epicardial surface of the left ventricle, seems highly unlikely in view of Grant’s studies.7

Initial depolarization of the interventricular septum from right to left in some of these cases has been suggested102 and catheterization studies by Fowler and co-workers136 would support this concept. Sodi-Pallares and associates8 have offered another explanation for this pattern in RVH. They believe that the right atrium is markedly enlarged with the result that this dilated chamber transmits to the right precordial leads the electrical effects of vectors that originate in the high basal portions of the interventricular septum. These workers have studied at autopsy 42 cases that exhibited this pattern and found enlargement of the right atrium in all cases and concomitant hypertrophy or dilatation of the right ventricle in all but one.

Lepeschkin62 has suggested that the Q wave corresponds to electrical forces, due to activation of the septum from right to left, which
are no longer opposed by those due to radial activation of the lateral wall of the right ventricle, as these appear later and are perhaps smaller due to decreased density of transitions between the conducting system and myocardium as a consequence of dilatation. Bayley\textsuperscript{21} offered still another explanation for the Q wave in V\textsubscript{1} in RVH. He believed it was due primarily to apical activity of the right ventricle when its greater dimension and proximity to the xiphoid process creates a larger, negative solid angle at V\textsubscript{1} and V\textsubscript{2}.

The third type of pattern that may occur is that of small r waves and deep S waves in the right precordial leads (and even in some cases in all precordial leads, including V\textsubscript{6}). This pattern is encountered primarily in chronic cor pulmonale and is thought to be due principally to the anatomic position of the heart.\textsuperscript{8} While the right ventricle is hypertrophied the heart is displaced downward in the chest owing to the low position of the diaphragm in pulmonary emphysema.\textsuperscript{8} The QRS voltage is decreased because the precordial electrodes are relatively far removed from the ventricles.\textsuperscript{8} Another explanation for this pattern is that there are right ventricular dilatation and clockwise rotation of the heart around its long axis displacing the rS pattern, which is thought to arise from the early right paraseptal ventricular region, to the left.\textsuperscript{8, 62}

The fourth category is the rSR' pattern in right precordial leads. This topic has already been discussed. Only a few additional points now need emphasis. This pattern, when it occurs in RVH, is ordinarily associated with right ventricular dilatation. This dilatation is thought to cause stretching and in some other way interfere with the right ventricular conducting network and result in a slowing of right ventricular conduction.

Grant\textsuperscript{38, 134} has grouped the electrocardiographic changes in right ventricular enlargement into 3 main patterns and has formulated the concept that each of these depends upon the sequence in which the various regions of the right ventricle are activated during the QRS interval and whether right ventricular dilatation or hypertrophy is present. The septal portion of the right ventricle contributes its electric forces during the first 0.04 second of the QRS interval and the free wall of the right ventricle contributes its electric forces during the second half of the QRS interval.

1. When the septal and paraseptal portions of the right ventricle are involved in the hypertrophy, abnormal right ventricular forces are developed during the initial 0.04 second of the QRS interval. These forces are directed anteriorly and give rise to the abnormally tall and broad initial R waves in V\textsubscript{1}. This pattern is encountered in pulmonic stenosis where there is concentric RVH.

2. When the inflow region of the free wall of the right ventricle (the portion that rests on the diaphragm) is predominantly hypertrophied or dilated, the terminal QRS forces point inferiorly and anteriorly. These produce right axis deviation in the standard leads and the transitional QRS complexes in the precordial leads are displaced to the right.

3. When the outflow portion and crista regions of the free wall are principally involved, the terminal forces point anteriorly and rightward. These produce an S wave in lead I and an R' in V\textsubscript{1}. This pattern is encountered in atrial septal defect where there is marked dilatation of the free wall of the right ventricle with sparing of the septal region. Grant believed that this RSR' pattern in atrial septal defect was not IRBBB but a type of parietal block possibly due to fibrosis that is encountered in marked dilatation.

This neat distinction, based upon selective changes in the right ventricle as suggested by Grant, has to our knowledge not yet been subjected to careful correlation with autopsy and electrocardiographic studies.

The diagnosis of RVH in the presence of RBBB is fraught with considerable difficulty. The observations of Dodge and Grant\textsuperscript{79} and of Fowler\textsuperscript{89} have already been cited, as well as the autopsy study of Booth and associates.\textsuperscript{95} In fact, Barker and Valencia\textsuperscript{78} and Wilson\textsuperscript{20} showed an illustration of intermittent complete RBBB in which the secondary R wave in V\textsubscript{1} was 15 mm. Yet, during normal
intraventricular conduction, there was no electrocardiographic evidence of RVH. Similar observations have been made by Levine.\cite{levine1971} Bryant\cite{bryant1981} believed that if the activation of the free wall of the right ventricle was sufficiently delayed so as to follow the phase when the maximal forces were developed in the free wall of the left ventricle, that excitation of the right ventricular free wall alone might give rise to the relatively tall secondary R waves in the right precordial leads. He concluded that RVH in the presence of a major degree of IRBBB can be diagnosed from the electrocardiogram with little accuracy.

**Combined Ventricular Hypertrophy**

The electrocardiographic diagnosis of CVH is even more difficult than is the diagnosis of isolated or preponderant hypertrophy of either ventricle. Normally the free wall of the left ventricle is approximately 3 times the thickness and twice the weight of the free wall of the right ventricle. If both ventricles undergo hypertrophy, but retain their relative proportions to each other, the left ventricle remains preponderant and the electrocardiogram tends to display the pattern of left ventricular hypertrophy.\cite{wilson1970} In fact, Wilson\cite{wilson1970} doubted that hypertrophy which increases the masses of the 2 ventricles proportionately could be distinguished from preponderant hypertrophy of the left ventricle alone. In addition, the observation has been made that even in RVH the thickness of the right ventricular wall seldom approaches, and only rarely exceeds, the thickness of even the normal left ventricular wall and that it seems unlikely that when the left ventricle is greatly hypertrophied even the most marked RVH could significantly alter the QRS forces.\cite{barker1980}

It is an observed fact, however, that biventricular hypertrophy does, on occasion, give rise to highly suggestive, if not absolutely diagnostic, patterns. Barker\cite{barker1980} believed that in order to produce an electrocardiographic pattern of CVH there must be increased thickness of the free wall of the left and of the right ventricle to almost equal degree. The QRS interval may be normal\cite{levine1971} or slightly prolonged in CVH.\cite{levine1971} The prolongation is presumably due to the increased time required for the impulse to spread through the thickened portion of the free wall of the left ventricle. Since the increased voltages generated by the hypertrophied ventricles tend to counterbalance each other, the amplitude of the deflections in the electrocardiogram may not greatly exceed the normal. There may be S-T and T-wave changes secondary to the increased duration of the QRS interval. Slurring and notching of the QRS complexes may occur because of local delays in intraventricular conduction due to widespread myocardial fibrosis.

Many patterns purported to be of value in the diagnosis of CVH have been offered.\cite{levine1971, wilson1970, levine1970, wilson1970} Among the more useful are the presence of LVH in the precordial leads with a vertical heart (or right axis deviation $>90^\circ$); marked displacement of the transitional zone to the left with $S>R$ in $V_5$, $R>Q$ in $aV_R$ together with signs of LVH; tall R waves with late peaks and inverted T waves in both right and left precordial leads.

Dilatation of the right ventricle in bilateral ventricular enlargement has been invoked to explain the displacement of the transition zone to the left, with "clockwise" rotation of the heart producing right axis deviation.\cite{wilson1970} It has been suggested that such "clockwise" rotation does not occur when the right ventricular enlargement is subsequent to left ventricular failure, for in these cases the right ventricle expands upward without displacement of the dilated left ventricle backward.\cite{wilson1970} When in combined ventricular enlargement there is more hypertrophy than dilatation of the right ventricle, the pattern of increase in voltage in the right precordial leads is encountered.\cite{wilson1970}

The electrocardiographic pattern encountered in ostium primum defects (persistent common atioventricular canal) is of interest. There is LAD in the standard leads (especially of the initial forces) and an rSR pattern in the right precordial leads.\cite{levine1971, barker1980, wilson1970, wilson1970, wilson1970, wilson1970} The left precordial leads may also display evidence of LVH.
While it has been proposed that the LAD is due to the associated mitral insufficiency (resulting from the left mitral valve), others deny this and attribute this configuration to altered spread of conduction through the left ventricle, At autopsy these cases exhibit RVH and dilatation and may also show LVH. Since the introduction of the concept of ventricular overload, patterns of CVH, especially in congenital heart disease, have been described. These patterns are further considered in the section on ventricular overload syndromes.

Ventricular Overload Syndromes

The interesting concept of systolic and diastolic overloading of the heart was introduced by Cabrera and Monroy and extended by Sodi-Pallares. They have described what they believe are specific electrocardiographic features and have related them to characteristic anatomic alterations in the ventricles. Systolic overloading of the right ventricle is illustrated by cases of pulmonic stenosis and of pulmonary hypertension. The electrocardiogram displays an increase in voltage of the R waves in right precordial leads and (in advanced cases) T-wave inversion. At autopsy these cases are found to have thickening of the free wall of the right ventricle and slight or moderate dilatation of the outflow tract only.

Diastolic overloading of the right ventricle is illustrated by atrial septal defect and by tricuspid insufficiency. The electrocardiogram exhibits RBBB (complete or incomplete) and at autopsy there is dilatation of the right ventricle. The stretching of the conducting fibers and increase in length of the conduction pathway associated with dilatation have been considered as causative factors in the production of the electrocardiographic pattern.

Systolic overloading of the left ventricle is found in aortic stenosis and in systemic hypertension. The electrocardiographic pattern is characterized by ST-segment depression and T-wave inversion in left ventricular leads.

At autopsy these cases display a thick-walled left ventricle without significant dilatation of the cavity. Cabrera and Monroy regard these T-wave changes as "primary."

Diastolic overloading of the left ventricle occurs in aortic insufficiency and in patent ductus arteriosus. The electrocardiographic pattern is characterized by tall R waves with late peaks in left precordial leads and deep S waves in right precordial leads. The T waves are tall and peaked and may be preceded by an upward S-T displacement in the left precordial leads. At autopsy there is usually considerable dilatation of the left ventricular cavity, with slight thickening of the wall.

Systolic overload is thought to manifest itself principally at the time of ventricular repolarization. The effect then is to delay the onset of repolarization, with the T waves of the affected ventricle becoming flattened or negative. Diastolic overload lengthens the activation process of the involved ventricle. The electrocardiogram shows a delay in the onset of the intrinsicoid deflection in the precordial leads reflecting the involved ventricle.

Combined right and left ventricular overload patterns have been described in a variety of congenital heart conditions. Marsico and Sodi-Pallares found this electrocardiographic pattern in 56 per cent of their 32 cases of ventricular septal defect. These cases exhibited (1) RVH with systolic overloading of the right ventricle with tall R waves in V1 and V2; (2) LVH with or without diastolic overloading of the left ventricle characterized in V5 and V6 by tall or normal R waves when a small deflection would be expected due to the marked RVH, delayed onset of the intrinsicoid deflection, positive T waves; (3) deep Q waves in V5 and V6, suggestive of septal hypertrophy. The mean spatial QRS vector deviated to the right, forward, and upward.

Patent ductus arteriosus with moderate pulmonary hypertension also has been shown to demonstrate CVH, characterized by RVH with systolic overloading and left ventricular diastolic overloading with tall peaked T waves in V5 and V6. Ziegler has also de-
scribed what he considers several diagnostic patterns of CVH in infants, characterized by various combinations of right and left ventricular overload patterns. He has pointed out that in infancy with QRS or T evidence of RVH in right precordial leads, the occurrence of T-wave inversion in left precordial leads is strong presumptive evidence of associated LVH. Another observation made by Ziegler was that in infants with electrocardiographic evidence of LVH the presence of positive T waves in right precordial leads is evidence of probable RVH, if the R in V1 exceeds 40 to 50 per cent of the total amplitude of RS in that lead. Some of Ziegler’s observations have been supported by anatomic studies.

The concept of systolic and diastolic overloading of the ventricles has been of considerable value in the interpretation of the electrocardiogram in various forms of congenital heart disease. However, to our knowledge there have been no extensive, careful, autopsy-controlled correlations with these electrocardiographic patterns. In fact, such an evaluation might be difficult because the concept of systolic and diastolic overload implies a physiologic or hemodynamic, more than an anatomic, alteration in the ventricles. Nevertheless, certain empiric observations have been made. In general, when ventricular enlargement is due to increased flow (diastolic overload), there is ventricular dilatation, and when the enlargement is due to increased resistance to flow (systolic overload), there is ventricular hypertrophy. It has also been observed that in congenital heart disease dilatation is more often accompanied by minor conduction defects than is hypertrophy, especially in the right ventricle. Left ventricular dilatation is said to increase QRS amplitude, whereas LVH is prone to produce accompanying S-T and T-wave changes. The recent study of Selzer and associates, however, tends to discount the distinguishing electrocardiographic features between LVH and left ventricular dilatation. The right ventricular diastolic overload pattern, so commonly encountered in atrial septal defect, has been shown post mortem to be associated with right ventricular dilatation, no relationship could be detected between the RBBB and the thickness of the septum or the right ventricular wall. Objection has been raised to the concept of the overload syndrome in that the electrocardiogram cannot record hemodynamic or mechanical events but only those anatomic and conduction alterations that may accompany such changes. Kossmann has pointed out that minor degrees of LBBB may be causative in the production of the electrocardiographic pattern of so-called left ventricular systolic overload. Therefore, the whole problem of the overload syndrome, while of considerable clinical interest, is not necessarily on a firm structural basis. Sodi-Pallares, furthermore, admits that there are diagnostic limitations to these electrocardiographic patterns.

Discussion

It would appear that with the considerable uncertainty still surrounding the exact mode of production of the electrocardiographic phenomena in ventricular hypertrophy, any attempt to correlate largely empiric electrocardiographic patterns with the anatomic expression of this hypertrophy may well lead not infrequently to evidence of disparity in the results. Furthermore, it has been pointed out that in indirect leads, there is no necessary correlation between the amplitude or duration of the QRS complex and the magnitude of the transmural voltage or the thickness of the ventricular wall. The QRS deflections represent the net effect of two sets of electric forces (opposite in sign) generated, not only in the portion of the heart immediately underlying the exploring electrode, but in all other parts of the heart as well. In addition, it has been emphasized that because of the composite value of the ventricular deflections in semi-direct leads, it is difficult, or perhaps impossible, to ascribe to a precise portion of the electrocardiogram electrical activity arising in an equally precise area of the right ventricle or left ventricle.

Despite the problems inherent in such stud-
ies, certain significant facts emerge. With the use of conventional electrocardiographic criteria for the diagnosis of LVH in adults an accurate diagnosis in autopsy-proved instances of isolated LVH is possible in about 85 per cent.27 On the other hand, given an electrocardiogram demonstrating the pattern of LVH in an adult, the possibility of making a false-positive diagnosis ranges from about 10 per cent to 15 per cent.28, 29 This is at variance with the experience of Levine and Phillips,26 who found that LVH was invariably present at autopsy when diagnosed electrocardiographically.

Emaciation, slender body build, and age appear to be some of the major factors resulting in a false-positive diagnosis of LVH. Obesity has been shown in some cases to obscure the electrocardiographic diagnosis of LVH. Thus the voltage criteria in LVH may be totally unreliable in subjects who are either markedly overweight or underweight. In addition, the present voltage criteria for LVH in adults may result in overdiagnosis in children and even in some normal young adults. Generally speaking, minimal or early LVH is more likely to be missed than marked LVH.

Of the various groups of electrocardiographic criteria for the diagnosis of LVH, delay in the onset of the intrinsicoid deflection was considered by Selzer to be the least reliable, having been present in many cases without anatomic LVH and absent in some with severe hypertrophy.29 In our study of autopsy-proved isolated LVH, delayed ventricular activation time was encountered in 26 per cent of the cases.27 Increase in voltage was present in 29 per cent of cases of isolated LVH.27 In Grant’s series31 high voltage occurred in over 90 per cent of cases with marked LVH. Selzer29 found high voltage to be present in most cases in his series but in that study the tracings were selected because they displayed LVH. He also found high voltage to be the most frequent cause of a false-positive diagnosis of LVH.

No close relationship can be demonstrated between any specific electrocardiographic abnormality and the ventricular weight, the left ventricular wall thickness, or the LV/RV wall thickness ratio. However, a significant association was found between increasing heart weight and increasing accuracy of diagnosis.27

The accuracy of electrocardiographic diagnosis of autopsy-proved preponderant or isolated RVH in different series varies widely (23 to 100 per cent). These reports, however, include heterogeneous groups with wide age ranges, varied etiologies of the RVH, and the utilization of various electrocardiographic criteria. To attempt to achieve some semblance of order from these studies, it may be observed that the electrocardiographic diagnosis of RVH is most accurate in congenital heart disease, less so in mitral stenosis and chronic cor pulmonale, and commonly undetected in RVH secondary to left heart failure. In most instances when the pattern of RVH is encountered in the electrocardiogram, the diagnosis is substantiated at autopsy, yet Walker and associates50 found that 4 of 12 patients presenting this pattern did not have RVH at autopsy. Lipsett and Zinn97 also found 5 of 17 cases with anatomic RVH to exhibit electrocardiographic evidence of RVH.

Anatomic CVH may present any of the following electrocardiographic patterns: (1) normal, (2) nonspecific abnormalities, (3) bundle-branch block, (4) preponderant hypertrophy of 1 ventricle, usually the left, or (5) a diagnostic pattern of biventricular hypertrophy. While a vertical axis in the presence of electrocardiographic evidence of LVH in the precordial leads has been proposed as highly suggestive of CVH, yet this pattern has been occasionally encountered in LVH alone.

Delay in the onset of the intrinsicoid deflection in right precordial leads in RVH and in left precordial leads in LVH has constituted an integral part of the conventional criteria in the electrocardiographic diagnosis of ventricular hypertrophy.20, 22, 42, 43 However, there has been increasing skepticism concerning the significance of the intrinsicoid deflection. This has stemmed largely from vectorcardiographic studies.44, 145-147 Milnor,44 in

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fact, stated that he does not make measurements of the "intrinsicoid deflection" because he believes that they are misleading in theory and of no value in practice. As he pointed out, it is probable that no precise meaning referable to a localized area of the myocardium can be assigned to the "intrinsicoid deflection." Actually, Wilson himself emphasized that the potential variations of every element of the epicardial surface contribute in some measure to the potential variations of an electrode placed upon the precordium. Therefore, although the exact significance and importance of the intrinsicoid deflection is at the present time still uncertain, it is an empirical observation that the onset is delayed in at least some cases of ventricular hypertrophy.

The value of axis deviation in the diagnosis of ventricular hypertrophy has undergone cycles of waxing and waning popularity. When the standard leads constituted the mainstay of electrocardiographic leads, right axis deviation (RAD) and left axis deviation (LAD) were given considerable importance in the diagnosis of respective ventricular hypertrophy. With the introduction of the unipolar extremity and chest leads, the importance attached to axis deviation diminished. In fact, Myers and Braunwald have indicated that axis deviation is of little or no value in the diagnosis of ventricular hypertrophy. Kossmann has stated that it is doubtful that RVH, except on rare occasions, can cause RAD by itself.

However, with the considerable interest at present in vectorcardiography and vector-electrocardiography, the evaluation of the frontal plane projection of the mean QRS axis in the scalar electrocardiogram has again gained favor. Milnor has placed considerable emphasis on RAD in the diagnosis of RVH. Phillips has demonstrated that RAD occurs earlier than the changes in the right precordial leads in the development of RVH in cor pulmonale. Grant has stated that RAD is the commonest manifestation of RVH. The relationship of LAD to LVH is perhaps somewhat less specific. Grant's studies of LAD in LVH have already been cited, including his belief that it is not the hypertrophy itself that produces the axis deviation but a conduction defect in the left ventricle. In our present state of knowledge, it would appear that abnormal degrees of axis deviation are of importance in the electrocardiographic evaluation of ventricular hypertrophy and, therefore, axis deviation should be determined.

That errors may occur in the electrocardiographic diagnosis of ventricular hypertrophy are well recognized and have been emphasized by Kossmann. The lack of agreement among various authorities in the electrocardiographic diagnosis of LVH has been specifically pointed out by Dimond.

It would appear appropriate to speculate about the possibility of future improvements in the electrocardiographic diagnosis of ventricular hypertrophy. Perhaps some of the proposed orthogonal electrocardiographic lead systems may simplify the electrocardiographic criteria and improve the diagnostic accuracy. Again, however, only an electrocardiographic-pathologic evaluation of such systems will establish their merit in this regard.

Summary

The correlation of the electrocardiogram with anatomic evidence of ventricular hypertrophy, while laden with numerous pitfalls, still remains the best available means of determining the accuracy of the electrocardiographic diagnosis of ventricular hypertrophy.

In 100 instances of isolated left ventricular hypertrophy (LVH) demonstrated at autopsy, a positive electrocardiographic diagnosis was made in 85 per cent by use of conventional criteria. However, in other studies designed to test the reliability of these criteria, it was found that a false-positive diagnosis was made in 10 to 15 per cent of the cases.

The electrocardiographic diagnosis of right ventricular hypertrophy (RVH) is more difficult. In electrocardiographic studies, confirmed by autopsies, the correlation has ranged from 23 to 100 per cent, while the number of false-positive diagnoses has been as high as 33 per cent. The correct electrocardiographic diagnosis is more frequent in
RVH due to congenital heart disease than to acquired heart disease.

The significance of the rSR' pattern in right precordial leads is discussed. Its occurrence in anatomic RVH and the problem of the electrocardiographic diagnosis of RVH in the presence of right bundle-branch block (RBBB) are reviewed.

Combined ventricular hypertrophy (CVH) is frequently missed in the electrocardiogram, the diagnosis having been made in only 8 to 26 per cent of cases proved at autopsy.

The unreliability of the electrocardiographic diagnosis of LVH in the presence of left bundle-branch block (LBBB) is documented.

The precise electrophysiologic phenomena that occur in ventricular hypertrophy are still largely conjectural. The more commonly accepted hypotheses are reviewed.

The lack of close correlation between ventricular wall thickness or respective ventricular muscle mass and the individual electrocardiographic patterns is emphasized. The possible explanations for some of these discrepancies are presented.

**Summario in Interlingua**

Le correlation del electrocardiogamma con evidentia anatomic de hypertrophia ventricular curre il riso de numeroso fallacia, sed illo remane nonobstante le meilior medio disponible pro determinar le accuratia del diagnosis electrocardiographic de hypertrophia ventricular.

In 100 casos de isolate hypertrophia sinistro-ventricular (HSV) demonstrar al necropsia, un positivo diagnosis electrocardiographic esseva facete 85 pro cento del vices per le application del criterios conventional. Tamen, in altrum studios planate pro testar le fidelitate de ille criterios, il esseva trovate que un diagnosis false-positive havava essite facete in inter 10 e 15 pro cento del casos.

Le diagnosis electrocardiographic de hypertrophia dexterovo-ventricular (HDV) es plus difficile. In studios electrocardiographic, necropicamente confarnate, le correlation ha variate inter 23 e 100 pro cento, et le numero del diagnoses false-positive ha essite alte, usque a 33 pro cento. Correcte diagnoses electrocardiographic de HDV es plus frequente quando le condition es le effecto de conenite morbo cardiae que quando illo es le effecto de acquirirte morbo cardiae.

Le signification del patrono rSR' in derivationes dexterovo-precordial es discuitate. Es revistate su occurring in HDV anatomic e etiam le problema del diagnose de HDV in le presentia de bloco de branea dexter.

Hypertrophia ambi-ventricular (HAV) escappa frequentemente al examine electrocardiographic. Iste diagnose esseva facete in solmente 8 inter 26 casos in que le condition esseva provate al necropsia.

Le basse fidelitate del diagnose electrocardiographic de HSV in le presentia de bloco de branea sinistre es documentate.

Le precise phenomenos electrophysiologic que occurre in hypertrophia ventricular remane in grande mesura conjectural. Le theorias le plus communemente acceptate es revistate.

Es signalate le manco de correlasion inter le spissitate del pariete ventricular o le respective massa muscular e le configuration electrocardiographic in le caso individual. Es presentate explicationes possibile pro certes de iste discrapantias.

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The treatment of established venous thrombosis and pulmonary embolism remains a problem. A survey of 80 cases of deep venous thrombosis treated by anticoagulants indicated a serious failure rate in the form either of recurrent venous thrombosis or of originally recurrent or recurrent pulmonary embolism. Heparin was more effective than Dicumarol in relieving symptoms of venous thrombosis, but was associated with a number of fatal pulmonary emboli in the presence of apparently adequate therapy. Venous ligation, especially at the level of the vena cava, supplemented by postoperative anticoagulant therapy seemed to offer the safest course with minimal morbidity.

KITCHELL

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The Correlation Between the Electrocardiographic Patterns of Ventricular Hypertrophy and the Anatomic Findings
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