Electrocardiographic Diagnosis of Ventricular Hypertrophy in the Presence of Right Bundle-Branch Block

By Richard W. Booth, M.D., Te-Chuan Chou, M.D., and Ralph C. Scott, M.D.

Forty-nine autopsied cases of complete and incomplete right bundle-branch block were studied with correlation of the electrocardiographic and anatomic diagnosis of ventricular hypertrophy. Twenty-six (53 per cent) of the cases showed anatomic evidence of right ventricular hypertrophy. The cases were studied for right ventricular hypertrophy according to the criteria of Barker and Valencia and of Milnor. Milnor's criteria resulted in frequent overdiagnosis while those that met Barker and Valencia's criteria usually but not invariably showed anatomic right ventricular hypertrophy. The electrocardiographic diagnosis of left ventricular hypertrophy was masked by the bundle-branch block in anatomically proved cases.

The presence of right bundle-branch block appears to make the diagnosis of ventricular hypertrophy more difficult, but there have been few reports concerning this problem in the recent English literature. Those studies that have been made since the introduction of the 12-lead electrocardiogram have been mostly confined to comparisons between the electrocardiogram and clinical findings. However, Caruso and associates, studied the pathology in 24 cases of right bundle-branch block and their conclusions conflicted with those arrived at on clinical grounds alone. A review of the literature reveals a considerable difference of opinion, not only as to the significance of right bundle-branch block, but also regarding its role in obscuring the diagnosis of left and right ventricular hypertrophy. It has been contended by Barker and that the diagnosis of left ventricular hypertrophy, though rendered more difficult, can often still be made. These concepts have also been upheld by Laham and associates.

The purpose of the present communication is to re-evaluate the accuracy of the diagnosis of ventricular hypertrophy in the presence of right bundle-branch block in a series correlating the electrocardiographic with the postmortem findings.

From the Cardiac Laboratory, Cincinnati General Hospital, and the Department of Internal Medicine, College of Medicine, University of Cincinnati, Cincinnati, Ohio.

Methods and Material

All the Cincinnati General Hospital postmortem records from the years 1950 to 1955 inclusive, comprising over 3,000 autopsies, were reviewed and those cases with an electrocardiographic diagnosis of right bundle-branch block were analyzed. The cases accepted were those in which a 12-lead electrocardiogram was available within 6 months of death, and where there was no pathologic evidence of myocardial infarction. Cases with infarction were excluded because of its possible effect upon the voltage of the QRS complexes.

The diagnosis of right bundle-branch block was made when the following criteria were present: (1) an S wave in lead I, (2) primary and secondary R waves in leads from the right precordium with the R' exceeding the initial R wave in height, i.e., RsR', sR', (3) delay in the onset of the intrinsicoid deflection in the right precordium greater than 0.05 second, (4) an S wave in V, or V, (5) no initial Q waves over the right precordium. The block was deemed incomplete if the QRS interval measured 0.08 to 0.11 second inclusive, and complete if the QRS interval measured 0.12 second or greater.

The material was then analyzed as to the presence of associated right ventricular hypertrophy.

*A few cases from Jewish Hospital, Cincinnati, Ohio, were also reviewed.

†The distinction between incomplete and complete right bundle-branch block listed above is based upon duration of the QRS and is the one used by Barker and Valencia. If the criteria of onset of intrinsicoid deflection in V, are used, i.e., 0.05 to 0.075 for incomplete and 0.08 second or greater for complete right bundle-branch block, some shifting of records from incomplete to complete status and vice versa will occur. We believe, however, that this in no way changes the conclusions we have reached.
<table>
<thead>
<tr>
<th>No.</th>
<th>Case &amp; Age</th>
<th>Cardiac diseases</th>
<th>Heart Wt./Gm.</th>
<th>RV Thickness dilated</th>
<th>LV Thickness dilated</th>
<th>RVH</th>
<th>LVH</th>
<th>Electrocardiogram</th>
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<tr>
<td>1</td>
<td>E.J./40</td>
<td>RHD-MS</td>
<td>480</td>
<td>7</td>
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<td>2</td>
<td>M.S./73</td>
<td>RHD-MLMS,ASHD</td>
<td>405</td>
<td>3</td>
<td>13</td>
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<td>3</td>
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<td>4</td>
<td>19</td>
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<td>E.D./70</td>
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<td>11</td>
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<td>12</td>
<td>E.P./72</td>
<td>ASHD,RHD-AS,MLMS</td>
<td>595</td>
<td>3</td>
<td>15</td>
<td>Yes</td>
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</table>

**Table 1.—Electrocardiographic and Postmortem Analysis, from Cases Showing Right Bundle-Branch Block**
as defined by Barker and Valencia, i.e., right ventricular hypertrophy is present if R' in the precordial leads is greater than 10 mm. in height in incomplete right bundle-branch block, and 15 mm. in height in complete right bundle-branch block.

Recently Milnor listed additional criteria for the diagnosis of right ventricular hypertrophy. These criteria are: (1) a QRS duration of less than 0.12 second, plus either (2) a mean frontal plane axis from +110° to ±180° or —91° to ±180° or (3) an R/S or R'/S ratio in V6 greater than 1.0, providing the R or R' wave in V6 is greater than 0.5 millivolt. Our material has been evaluated with the use of these criteria.

The diagnosis of left ventricular hypertrophy was made according to the combined criteria as listed by Scott and associates: (1) left axis deviation with R, plus S, exceeding 25 mm.; S-T depressed greater than 0.5 mm. in the lead I or a T wave less than 1 mm. in lead I, (in the absence of digitalis) (2) a negative deflection in aVL greater than 14 mm., (3) an R wave in lead aVL greater than 11 mm. in a horizontal heart, (4) an R wave aVF greater than 20 mm. in a vertical heart, (5) an R wave in V6 or V4 greater than 26 mm., (6) R in V6 or V4 plus the S in V4 exceeding 35 mm., (7) QRS duration exceeded 0.10 or 0.11 second; T-wave inversion in V6 or V4 with S-T depression (in the absence of digitalis); (8) delay in the onset of the intrinsicoid deflection between 0.05 and 0.07 in V6 or V4. If one or more of the criteria were met, left ventricular hypertrophy was considered to be present. This method of diagnosis has been found to be accurate in 96 per cent of the cases (in the absence of right bundle-branch block) in patients with proved left ventricular hypertrophy. Combined ventricular hypertrophy was considered to be present only if the criteria for both left and right hypertrophy as listed above were met.

The electrocardiograms were divided into 2 main groups, incomplete and complete right bundle-branch block, according to the criteria previously given. The onset of the intrinsicoid deflections in leads V1 and V6 and the height and duration of the R' in V6 and V4 were measured, as were the depth of the S in V1, V5, V6 and the height of the R in V2 and V6. The presence or absence of Q waves in the left precordial leads was noted and all tracings were analyzed for a possible diagnosis of left and right ventricular hypertrophy, according to the criteria previously mentioned.

The pathologic criteria for right ventricular hypertrophy were that the heart weight exceeded the normal as determined by Zeek and the right ventricle was 5 mm. or greater in thickness if not dilated, or 4 mm. if a definite statement of dilatation was found in the protocol. Left ventricular hypertrophy, was considered to be present if the heart weight was increased and the left ventricle measured 14 mm. or greater or 13 mm. if a definite statement of dilatation was found in the protocol. The ventricular measurements were made by different prossectors over a period of years, but the technic was reasonably constant and is listed as the greatest thickness from epicardium to endocardium.

**Results**

As indicated in table 1 cases were analyzed for age, pathologic cardiovascular diagnosis, body size, heart weight, left and right ventricular thickness at postmortem examination, and in addition whether or not ventricular dilatation was present.

In this series of 49 cases, unselected as to anatomic diagnosis (table 2), there were only 6 (12 per cent) normal hearts. Four (8 per cent) had isolated right ventricular hypertrophy, 15 (30 per cent) had isolated left ventricular hypertrophy and 22 (45 per cent) showed combined left and right ventricular hypertrophy. In addition, there were 2 hearts with increased weights but normal measurements of the ventricular walls.

*Not listed because of space limitation.*

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### Footnote for Table 1

RHD, rheumatic heart disease; MS, mitral stenosis; MI, mitral insufficiency; ASHD, arteriosclerotic heart disease; HCVD, hypertensive cardiovascular disease; AS, aortic stenosis, IASD, intratral septal defect; IVSD, interventricular septal defect, PDA, patent ductus arteriosus; IWNM, increased weight with normal wall measurements; MVI, mitral valve involvement; OI.D., onset of intrinsicoid deflection; CP, cor pulmonale; ALCA, anomalous left coronary artery; FF, focal fibrosis; COA, coarctation of aorta; FAH, focal amyloidosis of the heart; PE, pulmonary embolus; EPC, fibrinous pericarditis; MPE, multiple pulmonary emboli.
TABLE 3.—A Correlation of the Electrocardiographic Diagnosis with the Postmortem Findings in 49 Cases of Right Bundle-Branch Block

<table>
<thead>
<tr>
<th>Electrocardiographic Diagnosis</th>
<th>Postmortem findings</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total</td>
</tr>
<tr>
<td>Incomplete right bundle-branch block</td>
<td>28</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
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<td></td>
<td></td>
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<td></td>
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<tr>
<td>Complete right bundle-branch block</td>
<td>21</td>
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<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>49</td>
</tr>
</tbody>
</table>

RVH, right ventricular hypertrophy; LVH, left ventricular hypertrophy; CVH, combined ventricular hypertrophy; No Hy., no ventricular hypertrophy; IWNM, increased heart weight with normal ventricular measurements.

Incomplete Right Bundle-Branch Block. As noted in table 3, 28 cases met the criteria for incomplete right bundle-branch block. There were 8 cases that fulfilled Barker and Valencia’s criteria for co-existing right ventricular hypertrophy, i.e., right precordial R’ greater than 10 mm. At autopsy, of these 8 cases 2 had right ventricular hypertrophy, 1 had left ventricular hypertrophy, 3 had combined ventricular hypertrophy, 1 had no hypertrophy, and there was 1 with increased weight but normal measurements.

In addition to the 8 cases mentioned above, there was 1 case that also met the electrocardiographic criteria for left ventricular hypertrophy, but had right ventricular hypertrophy at postmortem examination and 1 that showed combined ventricular hypertrophy on the electrocardiogram but only left ventricular hypertrophy postmortem. At autopsy the remaining 18 cases with no electrocardiographic evidence of ventricular hypertrophy showed left ventricular hypertrophy in 5, combined ventricular hypertrophy in 11, no hypertrophy in 1, and there was 1 case with increased weight but normal wall measurements. Of the 5 cases of congenital heart disease all had incomplete block and 3 showed associated right ventricular hypertrophy on electrocardiogram. All had anatomic right ventricular hypertrophy.

Twenty-six of our cases of incomplete right bundle-branch block were also analyzed according to Milnor’s criteria. Three of these had isolated right ventricular hypertrophy; 7 isolated left ventricular hypertrophy; 12 combined ventricular hypertrophy, 1 no ventricular hypertrophy, and 3 with increased weight and normal ventricular wall measurement at postmortem examination. Fifteen of the 26 met Milnor’s criteria for right ventricular hypertrophy on the electrocardiogram. The diagnosis was correct in 9 (60 per cent) and erroneous in 6 (40 per cent). In addition 7 cases with combined ventricular hypertrophy were missed as was 1 case of isolated right ventricular hypertrophy, although this was admittedly a borderline one with a frontal plane axis of +102°.

Complete Right Bundle-Branch Block. The 21 cases of complete right bundle-branch block (table 3) revealed 3 with electrocardiographic evidence of associated ventricular hypertrophy, i.e., R’ greater than 15 mm. over the right precordium. At autopsy 1 had right ventricular hypertrophy, 1 left ventricular hypertrophy, and 1 combined ventricular hypertrophy. The remaining 18 cases that showed no electrocardiographic evidence of ventricular hypertrophy showed left ventricular hypertrophy in 7, combined ventricular hypertrophy in 7, and no ventricular hypertrophy in 4.

Comment. Left ventricular hypertrophy, although present anatomically as an isolated finding in 15 cases, could be interpreted on the electrocardiogram in only 1 case. The bundle-branch block was equally effective in masking an enlarged left ventricle whether it was complete or incomplete.

A total of 11 cases met the criteria of Barker and Valencia for associated right ventricular hypertrophy. It would appear that the right ventricle was indeed enlarged in 7 of these cases for a total of 64 per cent, although
in 4 of these left ventricular hypertrophy was also present.

**Discussion**

An analysis of our data leads to the conclusion that in cases unselected as to postmortem etiologic diagnosis, the presence of right bundle-branch block, either complete or incomplete, does not necessarily indicate right heart hypertrophy or even heart disease at all. In 28 cases of incomplete right bundle-branch block there were 2 with normal hearts and in 21 with complete right bundle-branch block there were 4 with normal hearts. In fact, isolated left ventricular hypertrophy was far more common than isolated right ventricular hypertrophy in our series. These findings are at some variance with those of Mounsey and co-workers who analyzed clinically 16 cases of emphysema and thought that an RSR' was usually, but not invariably, associated with right ventricular hypertrophy. Friedland and associates found right ventricular hypertrophy in 70 per cent of cases with incomplete right bundle-branch block and it has been stated by Levine that persistent incomplete right bundle-branch block is presumptive evidence of right ventricular hypertrophy.

Caruso and associates also have indicated that they consider an rR' or rsR' suggestive of right ventricular hypertrophy. Their conclusions were reached after postmortem studies, but their series was limited to cases with proved right ventricular hypertrophy. This perhaps may account for different results.

Laham and associates analyzed on clinical grounds 62 cases of right bundle-branch block, about half complete and half incomplete. They found 38 (61 per cent) with right ventricular hypertrophy, 21 (34 per cent) with left ventricular hypertrophy and 3 (5 per cent) with combined ventricular hypertrophy. Our pathologic correlation revealed right ventricular hypertrophy as an isolated lesion in only 4 cases (8 per cent), left ventricular hypertrophy in 15 (30 per cent), about the same as Laham, but combined ventricular hypertrophy was present in 22 (45 per cent), a much higher figure. This probably reflects the difficulty in making the clinical diagnosis of combined ventricular hypertrophy. In all, a total of 26 of our 49 cases (53 per cent) had an enlarged right ventricle either isolated or combined with enlargement of the left.

After analyzing our cases we agree, therefore, with Myers and associates who think that right bundle-branch block is merely suggestive, but certainly not pathognomonic, of right ventricular hypertrophy. In unselected cases right bundle-branch block is associated with enlargement of the left ventricle, either alone or in combination with right ventricular hypertrophy in too many cases to render it a significant sign of an isolated right ventricular lesion.

On the other hand the views of Said and Bryant and Camerini and Davies that incomplete right bundle-branch block is often found in normal subjects in no way detracts from its significance and the ability of this disturbance to obscure the electrocardiographic evidence of left ventricular hypertrophy remains a fact. An attempt was made to correlate the electrocardiographic diagnosis of right, left, or no ventricular preponderance with the ratio of right ventricular to left ventricular thickness in millimeters. Such an analysis failed to reveal the expected relationship that increasing mass of one ventricle would produce a more frequent electrocardiographic diagnosis of preponderance of said chamber.

In addition, if one attempts to relate the height of the R' wave over the right precordium to the ratio right ventricle/left ventricle thickness in millimeters, there can be noted no correlation between the voltage produced over the right precordium and the relative preponderance of the left or right ventricle.

Torner-Soler and associates considered that the blood pressure in the right ventricle correlates with the height of the R in right ventricular hypertrophy or the R' in incomplete right bundle-branch block especially in congenital heart disease. On the other hand, Scott and associates found that in their series of 28 cases of chronic cor pulmonale that had been catheterized, that incomplete
right bundle-branch block might occur at all levels of mean pulmonary artery pressure.

In essence, the genesis of the voltage of the R' wave remains obscure with neither ventricular mass nor mean pressure always producing a predictable effect.

The Diagnosis of Right Ventricular Hypertrophy. The diagnosis of right ventricular hypertrophy co-existing with right bundle-branch block, complete or incomplete, is not obscured in the opinion of Barker. On clinical grounds he thought that in nearly every instance of this combined hypertrophy-block pattern, as Torner-Soler terms it, right ventricular hypertrophy can be demonstrated.

Laham and associates also considered that a tall R' over the right precordium, which was present in 24 of 38 cases, believed to have on radiologic and clinical grounds right ventricular hypertrophy, was a valuable diagnostic sign. They added that this was true especially in cases that had a condition capable of overloading the right ventricle. They also thought that over the age of 40 the R' can be over 10 mm. without right ventricular hypertrophy. Although our postmortem cases range in age mostly above 40 we would certainly agree that the criteria for right ventricular hypertrophy in the electrocardiogram can be met and right ventricular hypertrophy not be found on postmortem examination in older patients who do not have a condition producing right ventricular over-loading.

In the study of Torner-Soler et al. 3 cases met the criteria for right ventricular hypertrophy and right bundle-branch block. All 3 had marked right ventricular hypertension when catheterized. In an over-all analysis of their cases it was noted, however, that the height of the R wave corresponded to the degree of right ventricular hypertension much better in congenital than in acquired heart disease.

Of our 11 cases that met the criteria for associated right ventricular hypertrophy, 7 (64 per cent) had right ventricular hypertrophy at postmortem examination. It is interesting to note that Caruso and associates in 24 cases of right ventricular hypertrophy confirmed post mortem found only 3 cases with an R' in the electrocardiogram greater than 10 mm. Although we had only 4 cases of isolated right ventricular hypertrophy, 3 of these 4 did have tall R' waves over the right precordium. These criteria obviously consistently miss the diagnosis of right ventricular hypertrophy when it is combined with left ventricular hypertrophy.

Utilizing the criteria of Milnor for the diagnosis of right ventricular hypertrophy, we found that this method will frequently make the diagnosis of right ventricular hypertrophy when it is not present anatomically, 6 cases (40 per cent) in our series. These criteria cannot be fairly judged for accuracy with regard to isolated right ventricular hypertrophy, as only 3 cases were in this group. When both ventricles were hypertrophied, however, the diagnosis could be made electrocardiographically in only 5 of 12 cases (42 per cent). It would appear, therefore, that in our series the usefulness of these criteria is limited by the fact that overdiagnosis of right ventricular hypertrophy may result.

Diagnosis of Left Ventricular Hypertrophy. Barker has indicated that he considers the diagnosis of left ventricular hypertrophy is difficult, although it can still be made, in the presence of right bundle-branch block. Laham and associates believed that they could make the diagnosis of associated left ventricular hypertrophy in 60 per cent of their cases on clinical grounds.

In our series this diagnosis was made only once by means of all available criteria. The one case that fulfilled the criteria at postmortem studies revealed isolated right ventricular hypertrophy. In our series, besides the 15 cases of isolated left ventricular hypertrophy there were 22 additional cases of combined ventricular hypertrophy or a total of 37 cases that had an enlarged left ventricle. The electrocardiographic diagnosis was obscured in all.

Summary and Conclusions

Electrocardiograms from 49 cases of right bundle-branch block that have come to autopsy have been reviewed and analyzed. The re-
RESULTS are discussed in terms of both the pathologic and electrocardiographic diagnosis of right and left ventricular hypertrophy.

The results indicate that in our series (1) of 49 cases of right bundle-branch block, unselected as to pathologic diagnosis, only 26 (53 per cent) showed anatomic evidence of right ventricular hypertrophy. (2) Those cases with electrocardiograms that met Barker and Valencia's criteria for co-existing right ventricular hypertrophy frequently, but not invariably, displayed right ventricular hypertrophy post mortem. The percentage being higher in congenital than in acquired heart disease. (3) Anatomic left ventricular hypertrophy, either isolated or combined, was consistently masked in the electrocardiogram by both complete and incomplete right bundle-branch block, (4) Utilization of the criteria proposed by Milnor for the diagnosis of associated right ventricular hypertrophy resulted in frequent overdiagnosis. The genesis of a tall R' in right bundle-branch block over the right precordium is discussed and it is concluded that at this time neither the muscle mass nor the mean pressure in the right ventricle or pulmonary artery can be consistently correlated with it.

SUMMARIO IN INTERLINGUA

Es passate in revista e analysate le electrocardiogrammas ab 49 casos de bloco de branca dextere pro le quaes reportos necroptic esseva disponibile. Le resultatos es discutite con referentia al diagnose tanta pathologic como etiam electrocardiographic de hypertrophia dextero- e sinistro-ventricular.

Le sequente observationes resulta del datos in nostre serie. (1) Inter 49 casos de bloco de branca dextere, solmente 26 (i.e. 53 pro cento) exhibiva signos anatomic de hypertrophia dextero-ventricular. (2) Le casos con electrocardiogrammas que satisfaceva le criterios de Barker e Valencia pro le co-existentia de hypertrophia dextero-ventricular exhibiva frequente—sed non invariablemente hypertrophia dextero-ventricular al necropsia. Le percentage esseva plus alte in morbo cardiac congenite que in morbo cardiac acquirite. (3) Hypertrophia sinistro-ventricular, isolate o combinate, ben que anatomicamente evidente, esseva uniformemente masche in le electrocardiogramma per complete e incomplete bloco de branca dextere. (4) Le uso del criterios proponite per Milnor pro le diagnose de associate hypertrophia dextero-ventricular resultava frequentemente in diagnoses falsemente positive. Le genese de un alte R' in bloco de branca dextere supra le precordio dextere es discutite. Es concludite que al tempore presente ni massa muscular ni le pression medie in le ventriculo dextere o in le arteria pulmonar pote esser correlazione con illo de maniera uniforme.

REFERENCES


The usefulness of esophageal leads in detection of atrial activity and in the differentiation of mechanisms of arrhythmias is illustrated by examples of a wandering pacemaker and of an atrial tachycardia with bundle-branch block simulating ventricular tachycardia. The recording of atrial and ventricular proximity potentials with the help of esophageal leads permits the demonstration of a different pharmacologic mechanism; by which quinidine and procaine amide suppress various types of ectopic impulse formation. Widening of P waves and QRS complexes following quinidine suggests that its action is mainly one of slowing of impulse transmission. Procaine amide on the other hand, does not affect the contour of the esophageal P waves, and hence appears to influence primarily the excitability of abnormal centers.

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