The Vectorcardiogram in Right Bundle Branch Block  
Correlation with Cardiac Failure and Pulmonary Disease  

John M. Fedor, Abe Walston, II, M.D., Galen S. Wagner, M.D., and John Starr, M.D.

SUMMARY  Frank vectorcardiograms (VCG) and clinical records of 243 patients with right bundle branch block (RBBB) were compared. The patients were classified into three categories on the basis of VCG criteria. The first category included 100 patients with a normal frontal axis, and the second category included 44 patients with concomitant left anterior hemiblock. The third category consisted of 99 patients with RBBB and myocardial infarction. The VCGs were classified into three types according to the QRS configuration in the transverse plane. In type I the initial forces were anterior and counterclockwise and the afferent limb crossed the midline posterior to E point; in type II the initial forces were anterior and counterclockwise and the afferent limb crossed the midline anterior to or through E point; and in type III the entire transverse loop was clockwise and anterior to E point. The patients were further classified according to the presence or absence of cardiac failure or severe pulmonary disease.

In patients with RBBB and a normal axis, cardiac failure or severe pulmonary disease was found in five of 49 patients with type I, 17 of 31 with type II, and 18 of 20 with type III pattern. In patients with RBBB and left anterior hemiblock, significant disease was found in one of 17 with type I, five of 16 with type II, and eight of 11 with type III pattern. These data show that, in patients with RBBB, the position of the afferent limb in the transverse plane can be used to predict cardiac failure or severe pulmonary disease.

SINCE THE DESCRIPTION BY WILSON ET AL. in 1932 of the electrocardiographic features of experimentally-produced right bundle branch block (RBBB), a wide spectrum of electrocardiographic (ECG) and vectorcardiographic (VCG) patterns have been observed to be associated with RBBB. Delay in the right bundle branch produces QRS prolongation with delayed terminal forces directed rightward and anteriorly. The extent to which the classical pattern of isolated RBBB can be modified by coexisting cardiac or pulmonary disease has not been adequately characterized in man. Previous studies have shown that in patients with RBBB, figure-of-eight, and clockwise transverse plane VCG patterns may be seen in the setting of coexisting left bundle branch block, left ventricular hypertrophy, right ventricular hypertrophy, and pulmonary disease. Although several recent texts have suggested that correlations exist between VCG patterns and the presence of specific disease states in patients with RBBB, corroborative clinical data have not been published. The purpose of this study was to correlate the types of RBBB with the clinical findings of either cardiac failure or severe pulmonary disease in a large number of patients.

Methods  
The study population consisted of 243 patients with VCG criteria for right bundle branch block (RBBB) seen at either Duke University Medical Center or the Durham V. A. Hospital. The VCG criteria for RBBB used for inclusion into the study were: A) QRS duration ≥ 120 msec, B) conduction delay with anterior and rightward displacement of the terminal position of the QRS. From the original group of 270 patients, 27 patients were excluded because of the presence of either idiopathic hypertrophic subaortic stenosis or Wolff-Parkinson-White syndrome, making a study population of 243 patients. The mean age of the patients was 57 (range 18–69); there were 215 males and 28 females. A large portion of the study population was obtained from the Veterans Administration Hospital which explains the male predominance.

Data Collection  
Vectorcardiograms were recorded using the Frank lead system on a Hewlett-Packard model 1507A vectorcardiograph. Chest electrodes were placed at the fourth intercostal space as recommended for the supine position. Photographs of the frontal, horizontal, and left sagittal planes were taken directly from the oscilloscope screen on Polaroid type 107 film. A calibration of 1 mV per 2–4 cm deflection was used, depending on the size of the VCG loop. To facilitate analysis the initial forces were enlarged using a calibration of 1 mV per 10 cm and photographed with the P and T loops excluded. The VCG loops were interrupted each 2.5 msec and all measurements were made by hand from the Polaroid prints of the VCG loops. A standard 12 lead ECG was recorded on each patient using a Hewlett-Packard automatic cardiograph (model 1515B). Right and left heart catheterizations were performed on 41 patients as part of their diagnostic evaluation.

Data Analysis  
The patients were divided into three categories on the basis of VCG criteria. The first category included 100 patients with "isolated RBBB" and a normal mean frontal axis (between +12° and +90°). The second category included 44 patients with RBBB and left anterior hemiblock (LAH). The VCG diagnosis of LAH was established when the frontal plane loop revealed rightward and inferior initial forces with a superior and leftward afferent limb, resulting in a counterclockwise loop with a mean frontal axis between +12° and −60°. Mean axis was determined as the angle of
the point of maximum voltage in the frontal plane. The third category consisted of 99 patients with RBBB and both VCG criteria and clinical evidence for myocardial infarction; these patients were analyzed separately. Eighteen of the 99 patients with myocardial infarction had concomitant left anterior hemiblock. The VCG diagnosis of anterior and diaphragmatic myocardial infarction was established using the criteria of Starr et al.10,11

The VCGs of patient categories 1 and 2 were classified into three types according to the QRS configuration in the transverse plane (fig. 1). In type I, the initial forces in the transverse plane were anterior and counterclockwise and the afferent limb crossed the midline posterior to E point (fig. 1, top panel). This configuration represents the classical RBBB pattern and gives a RSR in lead V1 of the standard ECG.5 In type II the initial forces were anterior and counterclockwise, and the afferent limb crossed the midline anterior to or through E point thus forming a figure-of-eight loop (fig. 1, middle panel). In type III the entire transverse loop was clockwise and anterior to E point (fig. 1, lower panel).

Analysis of the clinical records of patients in categories 1 and 2 allowed further classification of the patients according to the presence or absence of either cardiac failure or severe pulmonary disease. This classification was done only in the presence of specific clinical data as outlined below; unsupported clinical impressions were not sufficient basis for inclusion. Patients were classified as having cardiac failure when three of the following clinical signs were observed by at least two physicians: 1) ventricular (S3) gallop, 2) crepitant pulmonary rales, 3) peripheral edema, 4) cardiomegaly, 5) increased jugular venous pressure, 6) cephalization of pulmonary blood flow and Kerley B lines on chest roentgenograms, and 7) therapeutic response to diuretics. Severe pulmonary disease was established when, in addition to the clinical symptoms of pulmonary disease, pulmonary function studies revealed the vital capacity and forced expiratory one second volume to be less than 60% of the predicted (2 sp from normal), and arterial blood gases showed the pO2 to be <65 mm Hg and pCO2 >50 mm Hg.

Statistical analyses of mean frontal axis and pulmonary systolic pressures for the three VCG types were done using Student's t-test for unpaired data on an IBM 1130 computer.12 The determination of statistical significance of the number of patients in each group who had disease was done using Chi square analysis.

**Results**

Ninety-nine patients with RBBB also had VCG criteria and clinical evidence for myocardial infarction. Only the VCG classifications were applied to these patients. Therefore the final study population consisted of 100 patients with "isolated" RBBB, and 44 patients with RBBB and left anterior hemiblock. From these 144 patients there were 29 with cardiac failure, 18 with severe pulmonary disease, and seven with both diseases, bringing the total number of patients meeting the clinical criteria for cardiac failure or severe pulmonary disease to 54.

The relationship of VCG type to the presence of cardiac failure or severe pulmonary disease for patients with "isolated" RBBB and RBBB with left anterior hemiblock is shown in table 1. In patients with isolated RBBB, significant

**TABLE 1. The Relationship of VCG Type and Presence or Absence of Significant Cardiac and/or Pulmonary Disease**

<table>
<thead>
<tr>
<th>VCG pattern</th>
<th>N</th>
<th>Cardiac or pulmonary disease</th>
<th>Insignificant cardiac or pulmonary disease</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Insignificant cardiac or pulmonary disease</td>
<td></td>
</tr>
<tr>
<td><strong>Isolated RBBB</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>49</td>
<td>5</td>
<td>44</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>II</td>
<td>31</td>
<td>17</td>
<td>14</td>
<td>NS</td>
</tr>
<tr>
<td>III</td>
<td>20</td>
<td>18</td>
<td>2</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td><strong>RBBB with Left Anterior Hemiblock</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>17</td>
<td>1</td>
<td>16</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>II</td>
<td>16</td>
<td>5</td>
<td>11</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>III</td>
<td>11</td>
<td>8</td>
<td>3</td>
<td>&lt;0.005</td>
</tr>
</tbody>
</table>

*P derived from Chi square analysis.
RBBB and left anterior hemiblock classified according to VCG criteria.
N = number of patients; RBBB = right bundle branch block.
disease was found in five of 49 cases (10%) with type I pattern as compared to 17 of 31 (55%) with type II, and 18 of 20 (90%) with type III. Chi square analysis revealed that the difference between the number of patients in the disease category with type I and type III patterns was statistically significant ($P < 0.005$). Similarly in patients with RBBB and LAH, significant cardiac or pulmonary disease was found in one of 17 (6%) with type I pattern, five of 16 (33%) with type II, and eight of 11 (72%) with type III. Chi square analysis of this group revealed that differences in the number of patients in the disease category was significant for VCG types I, II, and III ($P < 0.005$).

In the 60 patients with isolated RBBB who had not met the clinical criteria for cardiac failure and pulmonary disease, there were 26 patients with a normal cardiovascular system. Of these 26 normals, 21 had a type I pattern and five had a type II pattern. Thirty patients with RBBB and LAH did not have cardiac failure or significant pulmonary disease, and ten of these were normal. Of these ten normals, six had a type I pattern and four had a type II pattern. In the remaining 54 patients with cardiac or pulmonary disease of insufficient degree to satisfy the clinical criteria, 33 had a type I pattern, 16 had type II, and five had type III.

To determine if the correlation between clinical status and VCG pattern was supported by hemodynamic findings, the cardiac catheterization data were analyzed in 41 patients. In this group cardiac failure or severe pulmonary disease was found in one of 17 patients with a type I pattern, seven of 12 with type II, and 12 of 12 with type III (fig. 2). In figure 2 the position of the afferent limb (at the midline) is compared to the systolic pulmonary artery pressure. The amount of anterior displacement of the afferent limb shows a linear correlation with increasing pulmonary pressure ($r = 0.79$). Pulmonary artery pressure elevation was significantly more prevalent in patients with type II and type III patterns ($P < 0.001$). The mean systolic pulmonary pressure was $28.8 \pm 1.8$ (SEM) for type I, $35.5 \pm 2.4$ for type II, and $61.3 \pm 2.8$ mm Hg for type III patients. Only three patients with a type I VCG had a pulmonary artery systolic pressure $> 35$ mm Hg, and all type III patients had a pulmonary pressure $\geq 40$ mm Hg.

Only distribution of VCG type was analyzed in the patients with myocardial infarction (MI) (table 2). The distribution of patients according to VCG type in diaphragmatic MI was almost exactly the reverse of that seen in anterior MI. Type I and type II patterns were more prevalent in DMI and type III was more prevalent in AMI.

The location of mean frontal plane axis (VCG) did not correlate significantly with either VCG type or the presence of cardiac or pulmonary disease. The mean frontal axis (VCG) was $43 \pm 11^\circ$ (SEM) for type I, $31 \pm 9^\circ$ for type II, and $55 \pm 33^\circ$ for type III patients with isolated RBBB. The mean frontal axes for the three VCG types were not significantly different. However, six of 19 patients with isolated RBBB type III pattern had a mean frontal plane axis $>90^\circ$, and of these six patients, four had severe pulmonary disease (data not shown).

A comparison of the ECG patterns in the standard limb leads and in lead V1 revealed no ECG criteria which could consistently allow identification of the patients with type I, II, and III VCG patterns.

**Discussion**

Right bundle branch block has been observed as a benign finding in approximately one percent of the normal population as well as an accompaniment of various types of heart disease. Right bundle branch block alters the terminal QRS, frequently obscuring the electrocardiographic manifestations of certain disease processes. The vectorcardiographic method affords a two dimensional display of the QRS complex, allowing the changes of RBBB to be more precisely analyzed than is possible with the standard ECG. The broad spectrum of vectorcardiographic patterns seen in patients with RBBB suggests that important diagnostic information may be gained from comparing the various VCG patterns seen in RBBB to the underlying clinical state. Several texts have suggested that certain VCG patterns in RBBB may be associated with heart disease; however data have not been published in support of these hypotheses.

In the present study the position of the afferent limb in the transverse plane afforded an easily identifiable descriptor to correlate with the presence of cardiac failure or pulmonary disease. The purpose of this study was not to associate a VCG pattern with an etiological diagnosis but rather test the value of a specific VCG pattern in predicting the presence of underlying cardiac failure or pulmonary disease. The data show that of 66 patients (without infarction) with the type I VCG pattern only six (9%) had evidence of significant dis-

---

**Table 2.** RBBB with Myocardial Infarction

<table>
<thead>
<tr>
<th>VCG Type</th>
<th>Anterior MI</th>
<th>Diaphragmatic MI</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>6</td>
<td>16</td>
</tr>
<tr>
<td>II</td>
<td>4</td>
<td>14</td>
</tr>
<tr>
<td>III</td>
<td>51</td>
<td>8</td>
</tr>
</tbody>
</table>

---

**Figure 2.** On the ordinate is expressed the position of the afferent limb as it crosses the midline at E point. This position is expressed as millivolts (MV) anterior or posterior from the E point using the transverse plane of the Frank lead system. On the abscissa is pulmonary artery systemic pressure in mm Hg. Patients with type I pattern are shown as circles, type II patients as squares, and type III patients as triangles. Patients with cardiac failure or severe pulmonary disease are shown as stippled circles (type I), squares (type II), and triangles (type III).
ease. However, in type II and type III patterns an increasing incidence of cardiac failure or severe pulmonary disease was found; 22 of 47 patients (47%) with type II and 26 of 31 patients (84%) with type III. Cabrera et al. reported a good correlation between magnitude of rightward anterior forces and ratio of right to left ventricular work in patients with RBBB and pulmonary hypertension from a variety of etiologies (mitral stenosis, atrial septal defect, and valvular pulmonary stenosis). Other studies have shown that patients without RBBB but with right ventricular hypertrophy from right ventricular pressure overload frequently demonstrate a vectorcardiographic pattern resembling the type III pattern identified here. This VCG pattern has been termed type A RVH. Therefore, it is not surprising that in patients with RBBB and right ventricular overload there would be a high incidence of type III pattern. Previous studies in patients with known right ventricular hypertrophy with and without RBBB suggest that the anterior displacement of the afferent limb may be manifestation of the accentuated right ventricular forces. Kulbertus et al. suggested that bilateral disease involvement of the conduction system may give rise to type II or type III patterns based on observations in which stimulation of the posterior wall during surgery produced clockwise and anterior transverse plane loops. This observation may in part explain those patients with RBBB with LAH who demonstrate type II patterns without evidence of myocardial disease. However, the QRS durations of types I, II, and III were not significantly different in this study, suggesting that coexistent left ventricular conduction defects were unlikely.

In 41 patients the comparison between pulmonary pressure and the VCG pattern showed that a greater number of patients with type II and type III VCG patterns had an elevated pulmonary artery pressure and clinical evidence of cardiac and pulmonary disease (fig. 2). Abildskov et al. also noted a direct correlation between the ECG characteristics of RVH and pulmonary pressures in patients with pulmonic stenosis. Scherlis et al. showed a regression of anterior forces and a conversion from clockwise to counterclockwise rotation (transverse plane) in patients following successful repair of congenital heart lesions with subsequent lowering of right heart pressures. These studies support our observations showing a good correlation ($r = 0.79$) between the level of pulmonary systolic pressure and anterior displacement of the afferent limb. The correlation shown here between anterior displacement of the afferent limb and the level of pulmonary pressure suggests that right ventricular overload may be an important factor in the genesis of the type II and type III VCGs. However, it is difficult to quantify a precise diagnosis of right ventricular hypertrophy in living patients because of A) the lack of precise methods to quantify right ventricular size in living patients, B) the difficulty in separating coexisting left and right ventricular hypertrophy, and C) the difficulty in separating the effects of right ventricular hypertrophy from right bundle branch conduction delays caused by stretch and elevated right ventricular pressure. The lack of correlation of mean frontal QRS axis with either VCG type or the incidence of cardiac failure or pulmonary disease in this study is supported by the findings of Baydar et al. showing no relationship between frontal QRS axis and the presence of ventricular hypertrophy.

The reason for the high incidence of type III patterns in patients with RBBB and anterior myocardial infarction is unknown. Left ventricular infarction and fibrosis may reduce the electromotive forces generated by the left ventricle, diminish the cancellation effect, and thereby accentuate the right ventricular forces. Left ventricular failure may cause elevation of pulmonary and right heart pressures as the left ventricular filling pressures rise, further augmenting right ventricular electromotive forces. Both of these factors may contribute to the high incidence of type III patterns in this group.

It is worthy of emphasis that many patients in this study demonstrated mild to moderate cardiac and/or pulmonary involvement but did not meet the strict clinical criteria required for inclusion into the cardiac failure or severe pulmonary disease categories. More precise methods of hemodynamic and clinical evaluation might have detected a higher incidence of cardiac failure and pulmonary disease. The two disease categories in this study are frequently associated with right ventricular hypertrophy and dilatation and it is tempting to speculate that the anterior displacement of the afferent limb in the setting of RBBB may be an indicator of early RVH.

In summary the data show the clinical usefulness of certain VCG patterns associated with RBBB. First, of the 66 patients with a type I pattern only 9% had cardiac failure or severe pulmonary disease whereas in the patients with a type III pattern (without infarction) 84% had cardiac failure or severe pulmonary disease. Second, none of the 36 patients with a normal cardiovascular system had a type III pattern (27 with type I, and 9 with type II). Third, differentiation between the three VCG types was not possible using the standard ECG. This study demonstrates that in patients with RBBB the position of the afferent limb in the transverse plane does possess predictive value in the recognition of underlying cardiac failure or severe pulmonary disease. The vectorcardiographic method allows this descriptor to be easily identified and extends the usefulness of the VCG in the diagnosis of heart disease.

Acknowledgment

The authors gratefully acknowledge the valuable contribution of Dr. Joseph C. Greenfield, Jr., for his stimulus and critical review of the manuscript during preparation. The Department of Medical Illustration at the Durham Veterans Hospital rendered valuable support. The secretarial assistance of Mrs. Brenda Haley is gratefully acknowledged.

References

The Conducting Tissues in Primitive Ventricular Hearts without an Outlet Chamber


SUMMARY We have studied the disposition of the cardiac conducting tissues in four hearts from situs solitus individuals possessing primitive ventricles without outlet chambers. These hearts correspond to the type of univentricular heart defined as common ventricle by Lev. All the hearts studied possessed normally positioned great arteries. Two groups, each consisting of two hearts, could be distinguished.

The first type possessed a small posterior ridge which divided the posterior portion of the primitive ventricle into right and left ventricular sinuses. The papillary muscles to the atrioventricular valves were separate structures and arose on each side of this posterior ridge. The conducting tissues in these hearts arose from an atrioventricular node situated in the atrial septum but deviated posteriorly. The atrioventricular bundle pierced the fibrous annulus posteriorly and descended on the posterior ridge, lying to its left side. A bifurcation was not identified, and bundle branches were not present.

The other two hearts had no posterior ridge. A common posterior papillary muscle supported both atrioventricular valves, and in one a marked anterior muscle bar produced obstruction of the pulmonary outflow tract. The connecting atrioventricular node was situated laterally in the right atrioventricular orifice, and the atrioventricular bundle descended into the right parietal wall of the primitive ventricle. A bifurcation of bundle branches was not observed.

The disposition of conducting tissue in these hearts differs from that found in “primitive ventricle with outlet chamber” in that the connecting atrioventricular node and bundle are situated anteriorly and are intimately related to the transposed pulmonary artery outflow tract in the latter anomaly. The surgical significance of these findings is emphasized.

DESPITE THE CONSIDERABLE INTEREST engendered by hearts which possess an apparently single ventricular chamber, relatively few investigations into the disposition of the conducting tissues in these anomalies have been published. Monckeberg1 demonstrated that in such hearts the atrioventricular bundle might be related either to a posterior ridge or from an unusually situated atrioventricular node from which it descended anteriorly. Visioli2 confirmed the first arrangement of conducting tissue in a case of what he termed "cor biloculare." We3 showed that the anterior distribution of conducting tissue was to be expected in primitive ventricular hearts with outlet chambers and transposed arteries, and our findings have subsequently been confirmed by Bharoti and Lev.4 Most univentricular hearts possess an outlet chamber.5 In those patients with univentricular hearts in which the outlet chamber is absent, the ventricle is unseptated, presenting as a huge ventricular septal defect. In this arrangement, the conducting tissue might be expected posteriorly, as found in the cases described by Monckeberg1 and Visioli.2

Traumatic heart block is a well-recognized complication when patients with hearts of this type are submitted to corrective surgery6,7 and this is borne out by experience in our own center. Of six cases submitted to surgery in Liverpool, two developed complete heart block; of these one survived with a permanent pacemaker. Three further patients developed variable rhythms with atrioventricular dissociation but without complete heart block, and two of these died. In view of this high incidence of rhythm disturbance in primitive ventricular hearts without outlet chambers, we have investigated the disposition of conducting tissues in the hearts of the three patients who died, and in a similar heart from our cardiopathological collection.

Definitions and Terms

Considerable controversy surrounds the nomenclature of univentricular hearts. Lev and his colleagues8 divided the

---

From the Department of Child Health and Royal Liverpool Children's Hospital, Liverpool, England.

Dr. Anderson is a British Heart Foundation Senior Research Fellow.

Address for reprints: Dr. J. L. Wilkinson, Department of Anatomy and Embryology, Institute of Child Health, Alder Hey Children's Hospital, Liverpool L12 2AP, England.

Received July 14, 1975; revision accepted for publication December 15, 1975.

The vectorcardiogram in right bundle branch block: correlation with cardiac failure and pulmonary disease.
J M Fedor, A Walston, 2nd, G S Wagner and J Starr

Circulation. 1976;53:926-930
doi: 10.1161/01.CIR.53.6.926

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1976 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/53/6/926

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/