Hemodynamic and Anatomic Correlation of Electrocardiogram in Double-Outlet Right Ventricle

By Ehud Krongrad, M.D., Donald G. Ritter, M.D., William H. Weidman, M.D., and James W. DuShane, M.D.

SUMMARY
Scalar electrocardiograms of 31 patients with double-outlet right ventricle (19 with and 12 without pulmonary stenosis) were correlated with the hemodynamic and anatomic findings. Atrioventricular conduction defects, intraventricular conduction delay, and counterclockwise frontal-plane loops were found less often than formerly reported. Counterclockwise frontal-plane loops were found with, as well as without, pulmonary stenosis. The presence or absence of left ventricular hypertrophy did not correlate with the severity of pulmonary vascular obstructive disease. Electrocardiographic evidence of left atrial enlargement was difficult to interpret. Additional intracardiac malformations could not be appreciated from the electrocardiographic findings. The predictive value of the electrocardiogram for the diagnosis of double-outlet right ventricle was considered to be low. In patients with double-outlet right ventricle without pulmonary stenosis, the electrocardiogram was not helpful in predicting the severity of pulmonary vascular obstructive disease.

Additional Indexing Words:
Double-outlet right ventricle Electrocardiogram

With the development of surgical technics for correction of the less common types of congenital heart disease, accurate anatomic and physiologic diagnoses are now necessary. Origin of both great vessels from the right ventricle, double-outlet right ventricle (DORV), is one of these types that is amenable to total correction. If the anatomic and hemodynamic situation could be predicted by electrocardiography, this might serve as an important tool to alert the clinician to the presence of DORV.

Earlier reports suggested that the presence of DORV, with and without pulmonary stenosis, might be suspected from the electrocardiogram,\(^1\),\(^2\) however, others were unable to confirm these observations.\(^3\),\(^4\)

This report presents the electrocardiographic experience in patients with DORV with and without pulmonary stenosis seen at the Mayo Clinic from September, 1961 to May, 1970. None of these patients was cited in earlier reports from this institution.\(^1\)

Materials and Methods
Of the 31 patients with DORV described in this report, there were 12 without pulmonary stenosis (group 1) and 19 with pulmonary stenosis (group 2).

The diagnosis was made according to previously described criteria.\(^1\),\(^5\) In all patients the ventricular septal defect was infracristal. None of the patients included in the study had had a previous ventriculotomy or atriotomy.

The diagnosis was confirmed by cardiac
catheterization including selective angiography and by operation in 15 cases; the 16 other cases were diagnosed by cardiac catheterization including selective angiography, by operation, or by autopsy in various combinations (table 1).

The electrocardiograms (ECG) were obtained with a paper speed of 50 mm/sec. Seven patients in group 1 and two patients in group 2 were receiving cardiac glycosides at the time the ECG was recorded.

The ECG interpretations were made according to criteria suggested by Namin. In addition and for the purpose of this study, the diagnosis of left ventricular hypertrophy (LVH) in the presence of severe right ventricular hypertrophy (RVH) was made when the height of the QRS complex in lead V6 exceeded 15 mm. Calculations of frontal P and QRS axes were made from the standard and unipolar scalar electrocardiographic leads.

Results

The hemodynamic and ECG findings are presented in tables 1 and 2.

Rhythm Disorders and Atroventricular (A-V) Conduction Defects

Normal sinus rhythm was present in all 12 patients in group 1 and in 16 of the 19 patients in group 2. Of the three others, one had atrial fibrillation (pt 22), one had a wandering atrial pacemaker (pt 26), and one had a low atrial pacemaker (pt 17). Eight of the 31 patients (26%) had a prolonged A-V conduction time. Five were in group 1 and three were in group 2.

P Wave Analysis

In the 28 patients with regular sinus rhythm, the mean atrial vector was anterior, inferior, and to the left. Evidence of atrial enlargement was present in nine of the 12 patients in group 1 (table 3). Tricuspid valve insufficiency was present in five of the seven patients with ECG evidence of combined atrial enlargement (fig. 1). Nine of the 19 patients (47%) in group 2 had ECG evidence of atrial enlargement. In seven there was a pattern of right atrial enlargement and in two, combined atrial enlargement.

Ventricular Complex and Intraventricular Conduction Delay

Three of the 12 patients in group 1 (25%) had a frontal plane counterclockwise QRS loop with right inferior initial forces while five had a clockwise loop and four had a figure-of-8 loop with right inferior and counterclockwise initial forces (fig. 2, upper). There was a wide scatter of mean QRS axes in this group of patients, ranging from +160° to −170°. The mean frontal T vector was +63°.

Twelve of the 19 patients in group 2 had clockwise frontal loops, five had figure-of-8 loops, and two had counterclockwise loops (fig. 2, lower). The mean frontal QRS vector was +123° for the patients with clockwise loops. The mean frontal T vector was +48°.

No predominant pattern was observed in the QRS configuration in lead V1. The most common configuration in group 1 was RSR and RS patterns (59%) while the most common in group 2 was rR and qR patterns (58%). Five in group 2 and two in group 1 had a Q wave in V1. All 31 patients had ECG evidence of RVH. In addition, six in group 1 (50%) and eight in group 2 (42%) had evidence of LVH.

Because the prediction of the size of the ventricular septal defect in patients with DORV is of surgical importance and because this defect provides the only egress of blood from the left ventricle, an attempt was made to correlate the left ventricular work load, as reflected by the ECG, with the size of the defect as estimated at operation and autopsy. It is expected that LVH will be found on the ECG in the presence of increased pulmonary blood flow (absence of pulmonary vascular disease [PVOD] or pulmonary stenosis), mitral insufficiency, or small restrictive VSD. No correlation was found among any of these parameters. In only three of six patients with anatomic LVH proved at autopsy was combined ventricular hypertrophy reflected by the ECG; in the other three patients the ECG showed only isolated RVH. An attempt to change the criteria and to diagnose LVH in the presence of an R wave in V6 of 20 mm or more did not improve the correlation with the hemodynamic or pathologic findings.

Intraventricular conduction delay was present in 16 of the 31 patients (51%). One patient in group 1 showed evidence of complete right
# Table 1

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**Group 2: With pulmonary stenosis**

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Abbreviations: C = cardiac catheterization + angiography; S = surgery; A = autopsy; RV = right ventricle; MPA = main pulmonary artery; FA = femoral artery; MVB = mixed venous blood.

*As estimated during operation or autopsy.

†At operation.
Table 2

Electrocardiographic Findings in 31 Patients with DORV

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<th>Age (yr)</th>
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<td>+40</td>
<td>90</td>
<td>RVH</td>
</tr>
<tr>
<td>25</td>
<td>3</td>
<td>140</td>
<td>0.18</td>
<td>0.06</td>
<td>+150</td>
<td>CW</td>
<td>qR</td>
<td>21/0</td>
<td>24/14</td>
<td>+90</td>
<td>60</td>
<td>CVH</td>
</tr>
<tr>
<td>26</td>
<td>12</td>
<td>75</td>
<td>2</td>
<td>—</td>
<td>—</td>
<td>CW</td>
<td>rR</td>
<td>21/1</td>
<td>8/8</td>
<td>+70</td>
<td>60</td>
<td>RVH</td>
</tr>
<tr>
<td>27</td>
<td>5</td>
<td>120</td>
<td>0.08</td>
<td>0.06</td>
<td>+150</td>
<td>CW</td>
<td>R</td>
<td>18/0</td>
<td>6/4</td>
<td>+80</td>
<td>70</td>
<td>RVH</td>
</tr>
<tr>
<td>28</td>
<td>6</td>
<td>100</td>
<td>0.13</td>
<td>0.06</td>
<td>+100</td>
<td>CW</td>
<td>Rs</td>
<td>19/9</td>
<td>16/0</td>
<td>+50</td>
<td>50</td>
<td>CVH</td>
</tr>
<tr>
<td>29</td>
<td>10</td>
<td>80</td>
<td>2.5</td>
<td>0.14</td>
<td>+135</td>
<td>S</td>
<td>rR</td>
<td>23/8</td>
<td>3/5</td>
<td>+20</td>
<td>115</td>
<td>RVH</td>
</tr>
<tr>
<td>30</td>
<td>14</td>
<td>90</td>
<td>0.18</td>
<td>0.08</td>
<td>+130</td>
<td>CW</td>
<td>qR</td>
<td>17/0</td>
<td>4/3</td>
<td>+40</td>
<td>90</td>
<td>RVH</td>
</tr>
<tr>
<td>31</td>
<td>10</td>
<td>110</td>
<td>0.16</td>
<td>0.08</td>
<td>+100</td>
<td>CW</td>
<td>rR</td>
<td>8/1</td>
<td>12/1</td>
<td>+30</td>
<td>70</td>
<td>RVH</td>
</tr>
</tbody>
</table>

Abbreviations: RVH = right ventricular hypertrophy; CVH = combined ventricular hypertrophy.

*Figure-of-8.
†In V1R.
bundle-branch block (CRBBB) with QRS duration more than 0.1 sec in the pediatric age group or more than 0.12 sec in adults. Fifteen (48%) showed the pattern of intraventricular conduction delay of the “incomplete” right bundle-branch block (IRBBB) type as manifested by polyphasic QRS in V1 and QRS duration of less than 0.1 sec. Table 3 presents

![Table 3](https://example.com/table3.png)

**Table 3**

**Final Electrocardiographic Diagnosis in 31 Patients with DORV**

<table>
<thead>
<tr>
<th>Anomaly</th>
<th>Group 1</th>
<th>Group 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>%</td>
<td>N</td>
</tr>
<tr>
<td>Prolonged A-V conduction</td>
<td>5</td>
<td>42</td>
</tr>
<tr>
<td>Right atrial enlargement</td>
<td>0</td>
<td>—</td>
</tr>
<tr>
<td>Left atrial enlargement</td>
<td>2</td>
<td>17</td>
</tr>
<tr>
<td>Combined atrial enlargement</td>
<td>7</td>
<td>58</td>
</tr>
<tr>
<td>Isolated RVH</td>
<td>6</td>
<td>50</td>
</tr>
<tr>
<td>RVH + LVH</td>
<td>6</td>
<td>50</td>
</tr>
<tr>
<td>IRBBB</td>
<td>7</td>
<td>58</td>
</tr>
<tr>
<td>CRBBB</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td>Frontal QRS loop</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Counterclockwise</td>
<td>3</td>
<td>25</td>
</tr>
<tr>
<td>Figure-of-8</td>
<td>4</td>
<td>33</td>
</tr>
<tr>
<td>Clockwise</td>
<td>5</td>
<td>42</td>
</tr>
</tbody>
</table>

Abbreviations: IRBBB = incomplete right bundle-branch block; CRBBB = complete right bundle-branch block.

**Figure 1**

Patient 9, 17½ years old, with severe PVOD (Qp/Qs = 0.5; Rp = 85.5 units-m²) and severe tricuspid incompetence; paper speed 50 mm/sec. There is clockwise frontal QRS loop with a mean axis of +100°. P-R interval was 0.18 sec, and ECG pattern is compatible with combined atrial enlargement in absence of increased pulmonary blood flow. Note absence of Q wave in lead V4 and voltage criteria compatible with CVH. Increased voltages over left precordial leads in absence of Q wave might still represent right ventricular potentials.
the cumulative final ECG diagnosis in the 31 patients.

Associated Anomalies

Associated cardiovascular malformations occurred in 17 of the 31 patients (55%) (table 4). Most frequent were tricuspid valve incompetence and atrial septal defect. Malformations of the A-V valve occurred in 25% of those in group 1 and subvalvar aortic stenosis occurred in 16% of those in group 2. There was no correlation between the ECG findings and any of these malformations. Three patients had DORV and A-V valve malformations but no ostium primum defect. In these three there was a counterclockwise frontal QRS loop in two and a clockwise frontal QRS loop in one. In two with subvalvar aortic ring, the ECG showed combined ventricular hypertrophy while in the third the pattern was that of RVH (fig. 3).

Discussion

In DORV, the right ventricle communicates directly with the aorta and maintains systemic pressure; ECG evidence of RVH was present in all 31 patients. Pressure overloading of the left ventricle might be expected if a very small ventricular septal defect is present. Left ventricular volume overloading might be
Table 4

Associated Anomalous of Hemodynamic Significance in 31 Patients with DORV

<table>
<thead>
<tr>
<th>Anomaly</th>
<th>Pt</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tricuspid incompetence</td>
<td>6</td>
<td>19</td>
</tr>
<tr>
<td>Atrial septal defect</td>
<td>6</td>
<td>19</td>
</tr>
<tr>
<td>A-V valve malformation</td>
<td>3</td>
<td>10</td>
</tr>
<tr>
<td>Subvalvar aortic stenosis</td>
<td>3</td>
<td>10</td>
</tr>
<tr>
<td>Blalock-Taussig anastomosis</td>
<td>3</td>
<td>10</td>
</tr>
<tr>
<td>Patent ductus arteriosus</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>Pulmonary incompetence</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Moderate-to-severe pulmonary</td>
<td>10</td>
<td>32*</td>
</tr>
<tr>
<td>obstructive disease (PVOD)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total with associated anomalies</td>
<td>17</td>
<td>55</td>
</tr>
</tbody>
</table>

*Nine patients in group 1 (75%), and one in group 2 (5%).

expected in the presence of a large ventricular septal defect without pulmonary stenosis or pulmonary vascular disease (PVOD) since the pulmonary blood flow is increased and left ventricular blood volume also is increased. A systemic artery-to-pulmonary artery anastomosis might add to the work of the left ventricle. Left ventricular hypertrophy was noted at autopsy in all 18 cases reported by Mehrizi⁹ and in 12 of 13 reported by Mirowski et al.² and others.¹⁰ ¹¹

In all six of our cases in which the left ventricular size was estimated at autopsy, anatomic LVH was found, but ECG evidence of LVH was noted in only three of these six. Thus, the anatomic-electrocardiographic correlation of LVH in the presence of RVH in our patients with DORV is not satisfactory. ECG evidence of combined ventricular hypertrophy was found less frequently than reported by Mirowski et al.² in patients with DORV and pulmonary stenosis and somewhat less than reported by Engle et al.³ in patients with DORV without pulmonary stenosis.

In group 1 in our series, hemodynamic findings reflected little or no PVOD in two patients, moderate PVOD in five, and severe PVOD in three. When the presence of ECG voltage criteria for LVH, presence or absence of deep Q waves (> 4 mm) in V₆ or prolonged intrinsicoid deflection in V₆ was

![Figure 3](image_url)

**Figure 3**

Patient 13, 33 years old, large VSD; P axis of +15°. Vector analysis of standard and unipolar extremity leads indicate a figure-of-8 frontal QRS loop with mean axis of 110°. No evidence of LVH in presence of combined severe pulmonary stenosis, subvalvar aortic stenosis, and right ventricular systolic pressure of 180 mm Hg.
correlated with the hemodynamics, patients with hemodynamic findings favorable to total surgical correction could not be differentiated from those with severe PVOD.

The lack of a good correlation between the diagnosis of atrial enlargement as manifested by the ECG and by hemodynamic events has been previously noted.12

In group 1 patients, combined atrial enlargement was commonly associated with tricuspid valve incompetence without increase in pulmonary blood flow. Indeed, of the seven patients who had ECG manifestations of combined atrial enlargement, five had evidence of tricuspid incompetence with marked right atrial enlargement. Of these five, only one had a pulmonary-to-systemic blood flow ratio of more than 2.0.

Intraventricular conduction delay of the right bundle-branch block type was observed in 89% of the cases reported by Mirowski et al.,2 63% being complete, and 26% incomplete. We found complete right bundle-branch block in only one of 31 (3%) cases. The total incidence of intraventricular conduction delay of the incomplete type was 48% in our series. However, because some QRS prolongation can be caused by ventricular hypertrophy13, 14 and peripheral conduction delay15 or ventriculotomy,16 this pattern in the presence of RVH was not considered as necessarily reflecting a true incomplete right bundle-branch block (that is, prolonged conduction in the right bundle).

A high incidence of counterclockwise QRS frontal-plane loops in patients with DORV without pulmonary stenosis was noted in earlier reports and was considered to be of diagnostic importance.1 Although these loops are common in patients of this type, subsequent reports failed to confirm the high incidence.3, 17 The discriminative significance is further decreased by the wide spectrum of congenital heart defects in which this finding exists.18 It is of interest that this finding occurs in DORV with pulmonary stenosis as well. This was observed in two of our cases, as well as in a previously reported case.11

Six of our patients showed a counterclockwise superiorly oriented frontal-plane QRS loop, four of them in association with intraventricular conduction delay. This pattern was interpreted by Rosenbaum et al.19, 20 as reflecting left anterior hemiblock or left anterior hemiblock in association with right bundle-branch block. Histologic21 evidence of relative hypoplasia of the anterior superior left bundle in cases of A-V canal, electrophysiologic evidence22, 23 compatible with preferential activation of the postero-inferior left ventricular wall, as well as other experimental, clinical, and surgical observations24-26 are in agreement with such an ECG diagnosis.

As noted, right ventricular dilatation and hypertrophy, as well as stretch of the conduction system or ventriculotomy are known to produce the ECG pattern of “incomplete” or “complete” right bundle-branch block. Consequently, the diagnosis of left anterior hemiblock with incomplete right bundle-branch block in the presence of right ventricular dilatation and hypertrophy27 or after operation25 is not valid. It might reflect peripheral conduction delay or right ventricular hypertrophy and not necessarily a true incomplete right bundle-branch block.

The significance of left anterior hemiblock and right bundle-branch block and its natural history in the congenital form may be considerably different from those of the acquired form. Patients with the acquired form tend to develop complete heart block,28-30 virtually an unknown complication in the congenital form. No data are yet available on the natural history of the congenital form of counterclockwise superiorly oriented frontal-plane QRS loops.

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Hemodynamic and Anatomic Correlation of Electrocardiogram in Double-Outlet Right Ventricle

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