Electrocardiogram in corrected transposition of the great vessels of the bulbo-ventricular inversion type

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Twenty cases of corrected transposition of the great vessels of the bulbo-ventricular inversion type, either lone or combined with other intracardiac anomalies, were analysed. Rhythm and/or atrio-ventricular conduction disturbances were common to all groups of cases. QRS pattern changes were found to be related both to ventricular inversion and to ventricular hypertrophy.

Isolated corrected transposition and corrected transposition with systemic ventriculo-atrial regurgitation give rise to tracings suggestive of systemic ventricular hypertrophy.

Corrected transposition of the great vessels with pulmonary stenosis or pulmonary artery hypertension is usually accompanied by the electrocardiographic signs of a venous-ventricular hypertrophy, with a characteristic inversion of the normal precordial pattern.

The conventional criteria of ventricular hypertrophy may be applied in corrected transposition of the great vessels but are less reliable than in cases without ventricular inversion.

The so-called electrocardiographic pattern of ‘ventricular inversion’ in this anomaly is related not only to the inverted position of the ventricles but to a greater extent to the predominant, anatomically left, venous-ventricular hypertrophy which re-establishes the normal weight ratio between the anatomically right and anatomically left ventricles.

Most cases of corrected transposition of the great vessels correspond to the so-called bulbo-ventricular inversion forms, in which the transposition of the great vessels is associated with an inversion of the ventricles. The classical electrocardiographic criteria of normality or ventricular hypertrophy have been established on hearts without ventricular inversion. Thus, the question arises whether these conventional criteria keep their diagnostic value in the particular case of corrected transposition of the great vessels with ventricular inversion. To answer this question the electrocardiograms of 20 cases of corrected transposition of the great vessels either lone or combined with other intracardiac anomalies were analysed. This also offers the opportunity to consider to what extent the electrocardiographic features of corrected transposition of the great vessels may be related to the associated lesions.

Material and methods
Twenty patients, 13 men and 7 women, ranging in age from 4 to 40 years with corrected transposition of the great vessels of the bulbo-ventricular inversion type were studied. They included cases with no other intracardiac anomaly (Group I, 3 cases) and cases in which the transposition was combined with lesions usually resulting in hypertrophy either of the systemic ventricle (systemic ventriculo-atrial regurgitation, Group II, 5 cases), or of the venous ventricle (pulmonary stenosis, Group III, 3 cases), or of both (ventricular septal defect with pulmonary stenosis or pulmonary artery hypertension, Group IV, 9 cases). The diagnosis of the malformation was established by catheterization and angiography. Clinical and laboratory data are summarized in the Table. The criteria used for the diagnosis of corrected transposition of the great vessels of the bulbo-ventricular inversion type, were: (1) the anterior position of the aorta in relation to the pulmonary artery at catheterization and/or angiography; (2) the particular course of the venous catheter placed into the right pulmonary artery, failing to overcross the retrograde arterial catheter on the antero-posterior view; and (3) the peculiar shape of the ventricular cavities on angiography; the venous ventricle from which the pulmonary artery arises is triangular, smooth, and morphologically left; the systemic ‘arterial’ ventricle from which the aorta arises is trabeculated, ‘V’ shaped, and morphologically right. The conventional electro-
TABLE Clinical and laboratory data of 20 cases of corrected transposition of the great vessels

<table>
<thead>
<tr>
<th>Group, case, No., Age and sex</th>
<th>Pressures (mm. Hg)</th>
<th>Associated lesions</th>
<th>Shunt</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean pulm. art. wedge</td>
<td>Mean pulm. vein wedge</td>
<td>Syst. ventricular atrial regurgitation</td>
</tr>
<tr>
<td>I 1 24 F</td>
<td>NR</td>
<td>NR</td>
<td>25</td>
</tr>
<tr>
<td>2 12 M</td>
<td>8</td>
<td>NR</td>
<td>20</td>
</tr>
<tr>
<td>3 11 M</td>
<td>NR</td>
<td>20</td>
<td>23</td>
</tr>
<tr>
<td>II 4 7 M</td>
<td>12</td>
<td>NR</td>
<td>18</td>
</tr>
<tr>
<td>5 11 M</td>
<td>NR</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>6 28 F</td>
<td>20</td>
<td>NR</td>
<td>33</td>
</tr>
<tr>
<td>7 30 F</td>
<td>14</td>
<td>NR</td>
<td>23</td>
</tr>
<tr>
<td>8 38 M</td>
<td>26</td>
<td>NR</td>
<td>95</td>
</tr>
<tr>
<td>III 9 20 F</td>
<td>NR</td>
<td>NR</td>
<td>17</td>
</tr>
<tr>
<td>10 19 M</td>
<td>NR</td>
<td>17</td>
<td>160</td>
</tr>
<tr>
<td>11 27 M</td>
<td>6</td>
<td>12</td>
<td>127</td>
</tr>
<tr>
<td>IV 12 4 M</td>
<td>20</td>
<td>NR</td>
<td>68</td>
</tr>
<tr>
<td>13 6 M</td>
<td>NR</td>
<td>16</td>
<td>100</td>
</tr>
<tr>
<td>14 40 M</td>
<td>NR</td>
<td>NR</td>
<td>110</td>
</tr>
<tr>
<td>15 22 M</td>
<td>NR</td>
<td>NR</td>
<td>114</td>
</tr>
<tr>
<td>16 12 F</td>
<td>8</td>
<td>NR</td>
<td>20</td>
</tr>
<tr>
<td>17 16 M</td>
<td>NR</td>
<td>NR</td>
<td>110</td>
</tr>
<tr>
<td>18 6 F</td>
<td>24</td>
<td>NR</td>
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<tr>
<td>19 12 F</td>
<td>NR</td>
<td>13</td>
<td>95</td>
</tr>
<tr>
<td>20 36 M</td>
<td>NR</td>
<td>10</td>
<td>110</td>
</tr>
</tbody>
</table>

NR, not recorded or not estimated.
SR, sinus rhythm; NR, nodal rhythm; AF, atrial fibrillation; CSR, coronary sinus rhythm; QRS, QRS duration in second; AQRSF and A frontal plane direction of the means QRS and T vectors; WBi, White-Bock index; V3R and VAT, V1 and VAT, V6 and VAT, QRS pattern ventricular activation time in milliseconds for each of these leads. ‘q’, ‘q’ wave smaller than 0·5 mm.

cardiographic criteria of lone or combined ventricular hypertrophy are those described in the publications of Cabrera (1958), Lenègre, Caruso, and Chevalier (1954), Dack (1960), Beregovich et al. (1960), Elliott, Taylor, and Schieber (1963), and Ruttenberg et al. (1966).

Results

Group I: Isolated corrected transposition of the great vessels (Cases 1, 2, and 3 in Table and Fig. 1). The rhythm was sinus in two cases; there was a complete atrioventricular block in Case 3. The frontal plane QRS axes were at −50°, −30°, and +10°, with a well-defined R1-S3 pattern and a White-Bock (1918) index greater than +17 in the 3 cases. The right praeordial leads had a ‘QS’ or ‘rS’ pattern, and the left praeordial ones had an ‘Rs’ or ‘RS’ pattern. The index of Sokolow-Lyon (1949) was within the upper limits of normal. ‘RS’ diphasic complexes in V2, V3, or V4 were present in Case 2. There was a ‘q’ wave in V6 or V7 in two cases.

Group II: Corrected transposition of the great vessels with systemic ventriculo-atrial regurgitation (Cases 4, 5, 6, 7, and 8 in Table and Fig. 2 and 3). There was a disordered rhythm and/or impaired atrioventricular conduction in three cases: a first degree atrioventricular block, with intermittent atrioventricular dissociation in

FIG. 1 Case 1. Corrected transposition of the great vessels, without associated intracardiac anomalies.
Case 5 (Fig. 3); a nodal rhythm with increased ventriculo-atrial conduction time in Case 6; and atrial fibrillation with complete atrioventricular block in Case 8. The mean frontal plane QRS axis pointed leftward, with a well-defined R1-S3 pattern and a White-Brock index greater than +17 in the 3 cases with a normal pulmonary artery pressure. The morphology of the right praeocardial leads was ‘QS’ or ‘rS’ in 4 cases, and ‘QR’ only in Case 8, which had severe pulmonary artery hypertension. There was a ‘q’ wave in V6 or V7 in 2 cases.

**Group III: Corrected transposition of the great vessels with pulmonary stenosis** (Cases 9, 10, and 11, in Table and Fig. 4 and 5). The rhythm was sinus in the 3 cases; there was a first degree atrioventricular block with intermittent atrioventricular dissociation in Case 11. The mean frontal plane QRS and T axes were normal. In the 2 cases with severe pulmonary stenosis, the right praeocardial leads had a ‘QR’ or ‘qRs’ pattern. The ventricular activation time was longer on the right than on the left prae-
cordial leads (counterclockwise rotation of the horizontal vectorcardiogram). 'RS' diphasic complexes in V2, V3, or V4 were present in the 3 cases. There was a 'q' wave in V6 or V7 in one case.

**Group IV: Corrected transposition of the great vessels with ventricular septal defect and pulmonary stenosis or pulmonary artery hypertension, with or without atrial septal defect and systemic ventriculo-atrial regurgitation** (9 cases in Table and Fig. 6 and 7). Sinus rhythm was present in 6 cases. In Case 19 there was a coronary sinus rhythm (Fig. 6). Two cases had a complete atrioventricular block. The frontal QRS axis pointed rightward in 7 cases; in 2 it was at ±0° and +70°. The White-Bock index was less than −14 in 6 cases. The mean frontal plane T axis lay between +50° and +100°. The right praecordial leads had a 'qR' or 'QR' pattern in 6 cases, and an 'rsR' or 'rR' pattern in 2 cases. The index of Sokolow-Lyon was normal. 'RS' diphasic complexes in V2, V3, or V4 were present in 6 cases. These diphasic complexes were larger than 50 mm. in one case only (Case 12). The ventricular activation time was longer on the right than on the left praecordial leads in all cases except one.

**Discussion**

The characteristic electrocardiographic signs of corrected transposition of the great vessels are of two types (Anderson, Lillehei, and Lester, 1957; Schiebler et al., 1961; Walker et al. 1958): (1) disordered rhythm and/or atrioventricular conduction, and (2) a peculiar QRS morphology with deep 'Q' waves in lead III and aVF, and inversion of the normal praeordial pattern, with 'qR' complexes on the right praeordial leads and 'RS' complexes on the left praeordial leads.

Disordered rhythm and/or atrioventricular conduction are found with almost equal frequency in each of the 4 groups of this series. Thus they appear to be an attribute of corrected transposition of the great vessels of the bulbo-ventricular inversion type, independently from any other intracardiac malformation.

On the contrary, QRS morphology differs considerably from one group to another.

Both isolated corrected transposition and corrected transposition with systemic ventriculo-atrial regurgitation usually showed an electrocardiographic pattern suggestive of systemic ventricular hypertrophy, which makes it impossible to differentiate the latter...
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from the former group (Badawi et al., 1961; Cumming, 1962; Gasul, Graettinger, and Bucheleres, 1959; Goodman and Kuzman, 1961; Hashiba et al., 1965; Keck et al., 1965; Kjellberg et al., 1955; Lev, Licata, and May, 1963; Lieberson et al., 1969; Platzer, 1955; Rotem and Hultgren, 1965; Rovelli and Ladelli, 1962; Ruttenberg et al., 1966; Schiebler et al., 1961). In both groups the electrocardiogram reflects the predominance of the systemic ventricle, which is in fact an anatomically right ventricle. This unusual situation recalls that found in Fallot’s tetralogy or trilogy which should have a similar weight ratio between the anatomically right and left ventricles, independently from their respective position. It is not possible in these two groups either to suspect the ventricular inversion from the pattern of the praecordial or endocavitary leads, or from the vectorcardiogram. Indeed, in Case 5, the endocavitary electrocardiogram recorded from the anatomically right systemic ventricle has a ‘QS’ morphology. Hashiba et al. (1965) obtained an ‘rS’ complex from the anatomically left venous ventricle of a case of isolated corrected transposition. In Case 1, the horizontal vectorcardiogram has a normal counterclockwise rotation (Fig. 8), and in this case a transient aspect of ‘complete right bundle-branch block’ had been observed during an attack of sinus tachycardia (Fig. 9).

In Group III, in which corrected transposition was combined with lesions usually resulting in hypertrophy of the venous ventricle, the electrocardiogram was either almost normal (Case 9, with mild pulmonary stenosis, ‘balanced’ biventricular hypertrophy; Fig. 4 and 10), or showed the signs of venous-ventricular hypertrophy with inversion of the normal praecordial pattern (severe pulmonary stenosis).

In Group IV, in which corrected transposition of the great vessels was associated with lesions usually resulting in combined ventricular hypertrophy (ventricular septal defect with pulmonary stenosis or pulmonary artery hypertension and bidirectional shunt), the ‘typical’ QRS electrocardiographic features were commonly observed. The electrocardiographic signs of systemic-ventricular hypertrophy were masked in this group by the signs of venous-ventricular hypertrophy in all but one case (Case 12: AQRS at ±9°; RS diphasic complex larger than 50 mm. in V4; ‘R’ wave larger than 25 mm. in V6 in presence of a prominent ‘S’ wave). Yet, in opposition to what is usually observed in Fallot’s tetralogy or trilogy there were no
negative T waves in lead III and in the right praecordial leads. It seems, therefore, that the classical electrocardiographic pattern of corrected transposition results from the combination of ventricular inversion with a predominant anatomically left venous-ventricular hypertrophy. This anatomically left venous-ventricular hypertrophy re-establishes the normal weight ratio between the anatomically right and anatomically left ventricles.

We wish to thank Dr. Joseph Baragan for the advice and help he has given to one of us (F.F.) in the preparation of the manuscript.

References


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Br Heart J 1970 32: 165-171
doi: 10.1136/hrt.32.2.165

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