Negative P Waves in Lead I in Dextroversion: Differential Diagnosis from Mirror-Image Dextrocardia

With a Report of a Successful Closure of a Ventricular Septal Defect in a Patient with Dextroversion Associated with Agenesis of the Right Lung

By M. Mirowski, M.D., Catherine A. Neill, M.D., Henry T. Bahnson, M.D., and Helen B. Taussig, M.D.

In congenital dextrocardia the problem is frequently whether dextroversion or a mirror-image dextrocardia is present. In the former the atria preserve their usual relationship in the frontal plane but the left ventricle lies anterior to the right. In mirror-image dextrocardia there is atrial inversion and the right ventricle lies anterior to the left.

The electrocardiographic findings are generally considered to be extremely useful in the differential diagnosis. A negative P wave in lead I has been repeatedly emphasized as the most constant and valuable electrocardiographic sign of mirror-image dextrocardia. Positive P waves in lead I are considered to be the characteristic feature of dextroversion; indeed, some authors believe this to be the "sine qua non" that the heart is dextroverted.

These concepts are based fundamentally on the analysis of the direction of the spread of atrial depolarization in both conditions. Inasmuch as the atria are transposed in mirror-image dextrocardia, the mean spatial vector of atrial activation is directed to the right, forward, and downward, producing negative P waves in lead I. In dextroversion, on the other hand, the atria are normally located in the frontal plane and the excitatory process, originating on the right side, spreads to the left, forward, and downward; therefore, the P waves are positive in lead I. As early as 1889, before the electrocardiographic era, Waller recognized that the distribution of the cardiac potentials in mirror-image dextrocardia was diametrically opposite to that in normal subjects.

These criteria are undoubtedly of diagnostic help in the majority of cases. Nevertheless, cases with mirror-image dextrocardia and upward P waves in lead I, as well as cases of dextroversion with negative P waves in lead I have been reported. For this reason Burgemeister spoke about the "confusing behavior" of the P waves in these conditions and Keith and co-workers stated that in the diagnosis of dextrocardia "electrocardiography has proved deceiving to a great extent since it has introduced diagnostic criteria which are confusing or inaccurate."

Recently we have had the opportunity to study a child with dextroversion of the heart associated with a ventricular septal defect and agenesis of the right lung in whom the P waves in lead I were inverted. In this case it was possible to correlate the electrocardiographic findings with the anatomic position of the ventricles. A careful study of the unipolar leads, according to the method of Sodi-Pallares, based on Wilson's concept of semidirect leads indicated that the hypertrophied left ventricle lay anterior to the right ventricle. Furthermore, these electrocardiographic studies showed that inversion of the P waves in lead I is not necessarily limited to cases of mirror-image dextrocardia as distinct from dextroversion and, indeed, as an isolated finding, is not diagnostic of this condition.

A brief case report is given prior to the
discussion of the electrocardiographic differential diagnosis.

Case Report

J.W. A 7-year-old girl was hospitalized for closure of her ventricular septal defect. She had a normal familial, prenatal, and natal cardiac history, but a cardiac murmur was detected in the neonatal period. Growth and development were impaired, and she had frequent upper respiratory infections and a wheeze and stridor on excitement or exertion. Cyanosis was never noted but at the age of 5 the patient had an episode of congestive heart failure. Since then she had been maintained on digitalis. In the National Children’s Cardiac Hospital, Miami, Florida, the diagnosis of dextroversion of the heart with ventricular septal defect and agenesis of the right lung was made.

The blood pressure was 100/60 mm. Hg. The hemoglobin was 14.6 Gm, and the hematocrit value was 43 per cent. She was underweight and underdeveloped, and showed a slight pectus excavatum. The heart was enlarged, the apex beat being in the right anterior axillary line in the fifth intercostal space. A systolic thrill was palpable in the second right intercostal space; a systolic thrill was also readily felt in the back, just below the spine of the right scapula. A grade V/VI long, harsh systolic murmur was audible, maximal in the second right intercostal space, and a grade II/VI mid-diastolic murmur was audible at the apex. The second sound to the right of the sternum was loud and no definite splitting was detected. No breath sounds were audible over the right hemithorax. The liver was on the right side and was not enlarged. The spleen was not palpable. The extremities showed no cyanosis, clubbing, or edema.

An electrocardiogram (fig. 1) showed negative P waves in leads I, II, III, and aV_{p}, upright P waves in aV_{R}, and isoelectric P waves in aV_{L} (AP = 120°). The rate was 85 per minute, the QRS duration was 0.08 second, the P-R interval 0.12 second. The AQRS was = 60° and the AT + 70°. The precordial leads showed rS complex in V_{6R}, qRS complex in V_{5R}-V_{3R} and qRs complex in V_{1}. The P waves were negative all across the precordium with the exception of V_{6R} where they were isoelectric. In spite of the negative P waves in lead I the electrocardiogram was interpreted as suggestive of dextroversion of the

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Table 1

<table>
<thead>
<tr>
<th>Catheter position</th>
<th>Oxygen content</th>
<th>Pressure (mm. Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Saturation (%)</td>
<td>Volumes (%)</td>
</tr>
<tr>
<td>Inferior vena cava</td>
<td>67</td>
<td>10.2</td>
</tr>
<tr>
<td>Superior vena cava</td>
<td>69</td>
<td>10.4</td>
</tr>
<tr>
<td>Right atrium, high</td>
<td>70</td>
<td>10.6</td>
</tr>
<tr>
<td>Right atrium, mid</td>
<td>68</td>
<td>10.3</td>
</tr>
<tr>
<td>Right atrium, low</td>
<td>73</td>
<td>11.1</td>
</tr>
<tr>
<td>Right ventricle, in</td>
<td>77</td>
<td>11.7</td>
</tr>
<tr>
<td>Right ventricle, mid</td>
<td>87</td>
<td>13.2</td>
</tr>
<tr>
<td>Right ventricle, out</td>
<td>85</td>
<td>12.9</td>
</tr>
<tr>
<td>Main pulmonary artery</td>
<td>86</td>
<td>13.0</td>
</tr>
<tr>
<td>Left pulmonary artery</td>
<td>87</td>
<td>13.2</td>
</tr>
<tr>
<td>Left femoral artery</td>
<td>92</td>
<td>13.9</td>
</tr>
</tbody>
</table>

*An analysis of the electrocardiographic interpretation follows this case report.
†Reproduced by courtesy of the National Children's Cardiac Hospital, Miami, Florida.

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heart with a hypertrophied left ventricle lying anteriorly and on the left of the right ventricle.

Roentgenogram (fig. 2) showed the heart to be lying entirely in the right hemithorax. No pulmonary markings were evident on the right. The pulmonary vascularity on the left was increased.

An angiocardiogram† showed a right superior vena cava entering the right atrium just a little to the right of the midline. From there the dye passed to the right to enter a normal trabeculated right ventricle and then into a large dilated left pulmonary artery, which crossed the midline to reach the left lung; no right pulmonary artery could be seen. From the left lung the dye entered two large left pulmonary veins, which joined and emptied into the left atrium. Thence the dye passed into the left ventricle, a rather large but otherwise normal appearing structure with smooth lining, and thereafter entered the aorta. After left ventricular filling re-opacification of the pulmonary artery was seen (fig. 3).

At cardiac catheterization the pressures in the right ventricle and the pulmonary artery were slightly elevated. There was a rise of about 2-volumes per cent in blood oxygen content at the ventricular level (table 1).

An anterolateral thoracotomy was performed through the right fourth interspace. There was a small space of loose areolar tissue, probably representing the right pleural cavity, but no lung or remnant of lung was found in it. There was a single left pulmonary artery and a single left pulmonary vein. The right atrium lay behind and to

![Figure 2](https://example.com/figure2.png)

**Figure 2**

Anteroposterior thoracic roentgenogram. The heart lies entirely in the right hemithorax. No pulmonary markings are evident on the right. The pulmonary vascularity on the left is increased.
Figure 3

Angiocardiogram. Left. The dye enters two large left pulmonary veins, which join and enter the left atrium. Right. The left atrium, left ventricle, and aorta are visualized.

Figure 4

Upper drawing: Schematic representation of the anterior aspect of the heart; bottom left: schematic representation of the relationship of the ventricles; bottom right: schematic representation of the relationship of the atria.

Discussion

Negative P waves in lead I with a right-lying heart suggest the diagnosis of mirror-image dextrocardia. Nevertheless, this diagnosis was discarded after analysis of the unipolar leads (fig. 1). The following findings were thought to indicate that the left ventricle lay anteriorly and to the left; the qRS complexes in V_{5R}, the qRs complexes in V_{3R}, and the qR complexes further to the left were all interpreted as those characteristic of the free left ventricular wall. In addition, the variation in potential of the trabecular zone of the right ventricle (rS) appeared in the precordial leads only in V_{6R}, but could also be observed in aV_{R}, suggesting that the right ventricle lay on the right, posteriorly, and inferiorly. The high voltage of the R waves in pulmonary artery. The distribution of the coronary arteries appeared normal (fig. 4). A right ventriculotomy was made during cardiopulmonary bypass, and a defect in the membranous septum was seen and closed (fig. 5).

Postoperatively no significant cardiac murmurs were audible. The patient's course was complicated by severe respiratory distress, which necessitated tracheotomy. Thereafter she did well and was discharged 3 weeks after operation in satisfactory condition.
V₁ and V₅R, as well as of the S waves in V₆R, the deviation of the mean electrical axis to the left (\( \Delta QRS = -60^\circ \)), and the extension of the left ventricular complexes over most of the precordium were all findings consistent with the diagnosis of left ventricular hypertrophy.

The possibility that the qRs and the qR patterns in the precordial leads reflected the variation in potential of the hypertrophied right ventricle rather than the left should be considered, since these configurations can be observed in both conditions.¹,² Three objections could be raised to the hypothesis that the ventricular complexes in V₅R and further to the left correspond to the right ventricle. The first concerns the position of the mean manifest electrical axis of the QRS (\(-60^\circ\)), which, even corrected for mirror-image dextrocardia, would still be \(-120^\circ\), a rather unusual position in right ventricular hypertrophy (with the possible exception of chronic cor pulmonale).³ The second objection is more important and is concerned with the interpretation of the presence of the q waves in leads V₅R to V₆R. Sodi-Pallares⁴ has studied the significance of the q waves in the right precordial leads of normally placed hearts, in the absence of myocardial infarction and usually in the presence of right ventricular hypertrophy (qR complex). He relates such q waves to right atrial dilatation, which allows transmission of the negative intra-atrial potential to the anterior thoracic wall. In our case this explanation seems quite improbable, not only because there is no other evidence of right atrial hypertrophy, but especially because the q waves are clearly seen in V₅R, quite far from the right atrium, even if it were admitted that it is inverted and lying on the left. On the contrary, the significance of these q waves, as well as of the r waves in

**Figure 5**

*Surgical exposure through the right anterolateral thoracotomy. The ventricular septal defect is exposed through a right ventriculotomy.*

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V_{6R} and aV_{P} will be perfectly clear if related to the first septal vectors of a dextroverted heart (fig. 6). Finally, the absence of the usual septal patterns (RS) can be noted on our tracing, with an abrupt change from rS to qRS complexes, a frequent occurrence in left ventricular hypertrophy and reported by Portillo and co-workers as a useful sign in the differential diagnosis between right and left ventricular hypertrophy in congenital dextrocardia.

The electrocardiogram was therefore interpreted as suggestive of dextroversion, as it showed an anteriorly placed hypertrophied left ventricle and a posteriorly placed, apparently normal, right ventricle. This diagnosis was confirmed at operation.

From a theoretical viewpoint another possibility could be considered in the electrocardiographic differential diagnosis, namely, that the ventricles are situated as in dextroversion and only the atria are inverted (mixed dextrocardia, type IIIa of Areilla and Gasul) and Grant also stated that no case of dextroversion has so far been reported in which the atria were abnormal in position. On the other hand, Areilla and Gasul cited one such case in their studies of 50 cases of congenital dextrocardia and at least two other cases, confirmed by postmortem studies, have been reported. It seems therefore that this might be a cause of error but it is an extremely rare one.

Since the negative P waves in lead I in our case were clearly not due to atrial inversion as proved at operation, some other explanation must be sought. After exclusion of technical errors and obvious arrhythmias, the other recognized causes of inverted P waves in lead I in dextroversion include (1) atrial ectopic pacemakers; (2) possibly, marked right atrial enlargement; (3) the rare cases in which dextroversion is associated with inversion of the atria; (4) to a lesser extent, situs inversus with levocardia has been pointed out by others; indeed, the occurrence of ectopic rhythms seems to be the explanation for the "confusing behavior" of the P waves.

As far as the negative P waves in lead I in the present case are concerned one would be tempted to relate them to the rightward rotation of the atria (fig. 4) and to a consecutive deviation of the mean P axis to the right. It is uncertain, however, that this deviation is sufficient to project the AP vector on the negative half of lead I. Moreover, the atrial displacement cannot explain the backward direction of the spatial P axis, responsible for the negative P waves in the precordial leads. The most plausible explanation is that the P waves configuration reflects an abnormal atrial activation due probably to a nodal or to a low left atrial rhythm. This hypothesis is supported not only by elimination of other possibilities but also by the relatively short P-R interval (0.12 second with a heart rate of 85) and by analysis of the postoperative electrocardiogram (fig. 7). This tracing, taken 2 weeks after surgery, shows quite pronounced changes in atrial activation. The position of the mean electrical axis of atrial activation in the frontal plane differs only slightly from the previous location (−150° versus −120°). Nevertheless, in the horizontal plane this axis, directed before the operation.
NEGATIVE P WAVES IN LEAD I

Figure 7
Electrocardiogram taken 2 weeks after surgery. Note particularly the appearances of positive P waves in almost all precordial leads. Leads $V_{SR}$ and $V_{3R}$ are taken with a 5 mm. standardization.

backward and to the right, points forward and to the right on the postoperative tracing, producing positive P waves in almost all precordial leads. It is more logical to attribute these changes to a different postoperative location of the atrial pacemaker than to any new distribution of the atrial potentials caused by the hemodynamic and positional changes after the closure of the ventricular septal defect.

It is obvious, therefore, that the analysis of atrial activation alone is not adequate to differentiate mirror-image dextrocardia and dextroversion. In mirror-image dextrocardia not only are the atria transposed but the ventricles are the mirror-image of the normal position, the right ventricle lying anteriorly and to the left of the left ventricle. In dextroversion the left ventricle remains on the left as in the normal but lies anterior to the right ventricle. Consequently, in both conditions the analysis of ventricular activation must be taken into consideration. According to this concept the electrocardiogram in dextroversion has to show not only upright P waves in lead I (in the absence of rhythm disturbances) but also the potential variations of the left ventricle on the left and anteriorly and those of the right ventricle on the right and posteriorly. In this case and in others recently studied in this clinic the use of Sodi-Pallares’ approach based on the semi-direct leads concept has usually made possible a precise diagnosis of the relative position of the two ventricles in patients with a right-lying heart. The presence of ventricular hypertrophy or of intraventricular conduction disturbances is generally not an obstacle to the correct determination of the spatial position of the ventricles. Moreover, when there is a discrepancy between the findings resulting from the analysis of atrial activation and those of ventricular activation, we believe that the latter are more reliable, since they are virtually independent of supraventricular disturbances of rhythm. The value of electrocardiography in the differential diagnosis between mirror-image dextrocardia and dextroversion can be greatly increased if emphasis is placed on the analy-

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sis of ventricular activation as revealed by the QRS complexes in the unipolar leads.

**Summary**

The electrocardiographic differentiation of dextroversion from mirror-image dextrocardia has frequently been based on the configuration of the P waves in lead I, the assumption being that P waves are upright in dextroversion and inverted in mirror-image dextrocardia.

A patient is presented with inverted P waves in lead I in whom an electrocardiographic diagnosis of dextroversion was made based on the analysis of the unipolar leads. This diagnosis was confirmed at surgery when a ventricular septal defect was successfully repaired.

The Sodi-Pallares method of analysis of the precordial leads is valuable in determining whether the left ventricle lies anteriorly and to the left as in dextroversion or posteriorly and to the right as in mirror-image dextrocardia. Because of the frequency of unusual atrial rhythms in patients with right-lying hearts, the configuration of the P waves is often unreliable. When there is a discrepancy between the result of analysis of atrial activation and that of ventricular activation, the latter should be used in determining whether dextroversion or mirror-image dextrocardia is present.

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_Circulation_. 1962;26:413-420
doi: 10.1161/01.CIR.26.3.413

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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