CLINICAL PROGRESS

Atrial Septal Defect
Electrocardiographic, Vectorcardiographic, and Catheterization Data

By Yu-Chen Lee, M.D., and Leonard Scherlis, M.D.

The Electrocardiogram is an important aid in the evaluation of right ventricular hypertrophy in both congenital and acquired heart disease. There have been more recent reports on the vectorcardiogram as offering additional information in the diagnosis of right ventricular hypertrophy.1-8

Most of the published electrocardiographic criteria are of value in instances of marked right ventricular hypertrophy. Minimal degrees of right ventricular hypertrophy, however, are often difficult to diagnose on the basis of the electrocardiogram alone. In addition, the presence of conduction delays of the right bundle-branch-block type may make the diagnosis of accompanying right ventricular hypertrophy difficult. This is often the problem in atrial septal defects where “incomplete right bundle-branch block” or “terminal conduction delays” are often described.

Cardiac catheterization and cardiac surgery have permitted a more complete evaluation and diagnosis of congenital cardiac lesions, and have afforded the opportunity for the correlation of electrocardiographic, vectorcardiographic, hemodynamic, and anatomic findings in atrial septal defects. The importance of the electrocardiogram9-24 and vectorcardiogram1-8, 24-31 as tools for the diagnosis of atrial septal defect has been stressed by many authors. However, there is no agreement regarding the correlation of the morphology of the electrocardiogram and the magnitude of either the shunt flow or the right ventricular pressure in atrial septal defects.

In this report is presented an analysis of the electrocardiographic and vectorcardiographic tracings, and anatomic and hemodynamic data in 70 patients with atrial septal defects studied at the University Hospital of the University of Maryland. Sixteen of these patients were also evaluated at varying intervals after surgery with electrocardiograms and vectorcardiograms.

Material and Methods

The present study is based on a series of 70 patients in whom the diagnosis of atrial septal defect was confirmed by right heart catheterization. In addition to the usually accepted clinical criteria, there was an increase of at least 2 volumes per cent oxygen saturation in the right atrial blood samples as compared with superior or inferior vena caval blood samples. All instances of atrial septal defects with accompanying pulmonic stenosis, interventricular septal defect, or coarctation of the aorta were excluded.

Repair of the defects under direct vision was carried out in 32 patients by R. Adams Cowley, M.D. These anatomic defects included 16 patients with uncomplicated ostium secundum, eight instances of ostium primum, two ostium secundum with mitral stenosis, one ostium secundum with rheumatic mitral insufficiency, and five with ostium secundum and partial anomalous pulmonary venous drainage.

The electrocardiograms included 12 conventional leads and V3R. They were analyzed for rate,
rhythm, P-R interval, amplitude and duration of the P wave, QRS duration, frontal plane axis, R and S wave amplitude, and R:S ratio in aVR, V₃₉ and V₆; intrinsicoid deflection in V₃₉, V₁, and V₆; configuration of V₃₉ and V₁; and configuration of P wave in V₃₉ and V₁. The electrocardiographic diagnosis of right ventricular hypertrophy was evaluated upon the basis of the criteria of Sokolow and Lyon, Barker and Valencia, and Milnor and Bertrand. Vectorcardiograms were recorded by the cube reference system of electrode placement. Polaroid photographs were taken of the oscilloscopic screen of a Technicon Vector-Scope or a Sanborn Visoscope. Of the 70 patients studied, 33 were male and 37 female. The ages of the patients ranged from 8 weeks to 52 years as shown in table 1.

In 16 patients, electrocardiograms and vectorcardiograms were recorded for periods up to 4 years after surgical repair.

**Results**

**Analysis of the Preoperative Electrocardiograms**

Rhythm

Sixty-four patients had normal sinus rhythm. There were four patients with atrial fibrillations, two of whom (27 and 35 years of age) had secundum defects and mitral stenosis. The other two patients had uncomplicated ostium secundum defects and were 35 and 54 years of age. Two patients, 5½ months and 29 years of age, had A-V nodal rhythm.

P Waves

In adults, the P wave was considered enlarged if 0.11 second or more in duration, or more than 3.0 mm in amplitude. In children, P waves more than 0.08 second in duration and 2.5 mm. in amplitude were considered enlarged. There were 18 instances of atrial hypertrophy. In 24 patients, the P waves were diphasic in V₃₉ and V₁.

P-R Intervals

The P-R interval was prolonged in 13 tracings; seven in adults ranging from 0.21 to 0.28 second, and six in children under 13 years of age, with a range of 0.15 to 0.23 second. The criteria of Alimurung and Massell were used for children.

**QRS Complex**

The QRS duration was less than 0.10 second in 41 patients, 0.10 to 0.11 second in 24, and 0.12 second or greater in five patients. Among the last group, three were proved at surgery to have ostium secundum, and one to have an ostium primum defect. The remaining one was thought to have ostium primum. The configuration of the QRS complex in lead V₁ is shown in table 2.

Among the 65 electrocardiograms in which the QRS duration was less than 0.12 second, 42 tracings revealed R or R' in V₁, 5 mm. or more in amplitude and an R:S ratio greater than 1. Of the five records with QRS duration of 0.12 second or longer there were three tracings with rsR' configuration in V₁, one with Rs, and one with qR. The amplitude of R or R' was 5, 10, 14, 18, and 19 mm. in these five records.

**QRS Axis**

The mean QRS axis was 0° to +90° in 19 patients, in six of whom the diagnosis of ostium secundum defect was confirmed at surgery (fig. 1). In 36, the mean QRS axis was from +90° to +140°, with the diagnosis of secundum defect confirmed at operation in 16 cases. The axis was −150° in one patient found to have an ostium secundum defect at surgery. In one instance of ostium primum confirmed at surgery, the axis was −100°. The axis ranged from −10° to −90° in 13 cases. Eight of these patients had ostium primum lesions at surgery. One patient had a secundum defect complicated by rheumatic
mitral insufficiency. The remaining five patients were thought to have ostium primum lesions.

The presence of electrocardiographic evidence of right ventricular hypertrophy was evaluated upon the basis of the following criteria18, 32, 33, 35 (table 3): (1) qR in V3R or V1. (2) R in V1 equal to or more than 7 mm. (3) R in V1 equal to or more than 5 mm. (4) R/S in V1 equal to or more than 1. (5) Intrinsicoid deflection in V1, 0.035 to 0.05 second. (6) P enlargement. (7) rsR' in V3R or V1 with intrinsicoid deflection 0.05 to 0.075 second. (8) Criterion 7 + R' more than 10 mm. (9) Axis of +110° to -90°. (10) R in aVR equal to or more than 5 mm. (11) R/S in V5 equal to or more than 1.

**Catheterization Data**

The average of the right ventricular systolic pressure was lowest, 29 mm. Hg, among the rsR group; and highest, 60 mm. Hg, among those with qR, qRs, R, or Rs configuration in V1. The average right ventricular systolic pressure was higher in the rsR' as compared to the rsr' group (table 4).

In the group with normal axis (0 to +90°),

![Graph showing the distribution of the mean QRS axis in the electrocardiogram.](image-url)
the average right ventricular systolic pressure was 31 mm. Hg and ranged from 18 to 60 mm. Hg.

In the group with right axis deviation (+90° to -90°), the pressure ranged from 25 to 120 mm. Hg, with an average of 47 mm. Hg. In the group with left axis deviation (-10° to -90°) the average right ventricular pressure was 49 mm. Hg and ranged from 25 to 90 mm. Hg.

The ratio of pulmonary blood flow to systemic blood flow was greatest in those patients who had QRS complexes of the rSR' type in V1 and lowest in the rS and rsr' types (table 4).

The relationship between right ventricular pressure and the amplitude of the R wave in V1 was determined (fig. 2). The amplitude of the R wave in V1 tends to increase as the right ventricular pressure increases. However, many exceptions existed; for example, in one patient, the right ventricular pressure was 120 mm. Hg and the height of the R wave in V1 was only 3 mm.

Analysis of Preoperative Vectorcardiograms
The preoperative vectorcardiograms included five normal tracings, 49 instances of right ventricular hypertrophy, 15 with terminal conduction delay and one classified as miscellaneous.

The vectorcardiograms with right ventricular hypertrophy were divided into three general groups although considerable variation exists in each subgroup.

Type 1
There were 21 instances of type 1 right ventricular hypertrophy. (fig. 3). The initial

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Table 4
Catheterization Data and QRS Configuration in V1

<table>
<thead>
<tr>
<th>QRS complex</th>
<th>rS</th>
<th>rsr'</th>
<th>rsR'R</th>
<th>Rs</th>
<th>qR</th>
<th>qRS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>6</td>
<td>10</td>
<td>32</td>
<td>22</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RV Systolic pressure (mm. Hg)</td>
<td>29</td>
<td>30</td>
<td>44</td>
<td>60</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PBF*</td>
<td>2.3</td>
<td>2.4</td>
<td>4.5</td>
<td>2.5</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*PBF, pulmonic blood flow; SBF, systemic blood flow.

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The initial inscription of the QRS sE loop (centrifugal) is normal and is directed to the right and anteriorly, and then to the left and slightly posteriorly. The returning or centripetal loop turns abruptly to the right in a clockwise or counterclockwise direction, and crosses the centrifugal loop before returning to the E point. This group also includes those tracings with a terminal appendage but without slowing. The direction of inscription of the QRS sE loop in the sagittal plane was clockwise in six, counterclockwise in 10, initially clockwise and terminally counterclockwise in three, and initially counterclockwise and terminally clockwise in two. The direction of inscription of the QRS sE loop in the frontal plane was clockwise in 11, counterclockwise in seven, and initially counterclockwise and terminally clockwise in three.

Type 2
In this group, there were 11 records. The initial inscription of the QRS sE loop in
the horizontal plane is similar to type 1. After the centrifugal inscription to the left, the loop is then inscribed in a clockwise direction anteriorly and to the right. (fig. 4A).

The direction of inscription of the QRS sE loop in the sagittal plane was clockwise in two, counterclockwise in eight, and initially counterclockwise and terminally clockwise in...
one. The direction of inscription of the QRS sE loop in the frontal plane was clockwise in seven, counterclockwise in two, and initially counterclockwise and terminally clockwise in two.

Figure 4A

C.N., 15 years old. Ostium secundum. The preoperative vectorcardiogram represents type 2 right ventricular hypertrophy as described in the text. The preoperative electrocardiogram revealed rsR' in V1, QRS 0.08 second.

Type 3

There were 17 instances of type 3 right ventricular hypertrophy (fig. 5). In the horizontal plane, the initial portion of the QRS sE loop is directed almost immediately to the
left with a small or absent septal deflection. It is then quickly inscribed to the right and anteriorly in a clockwise direction. The main portion of the QRS sE loop is markedly displaced to the right and anteriorly. In the sagittal plane, the direction of inscription was clockwise in two, counterclockwise in 12, and initially counterclockwise and then terminally clockwise in three. In the frontal plane, the direction of inscription of the QRS

Figure 4B
C.N., 4 months after repair of atrial septal defect. The vectorcardiogram reveals type I right ventricular hypertrophy. No significant electrocardiographic changes are noted.
s: loop was clockwise in 10, counterclockwise in three, and initially counterclockwise and terminally clockwise in four.

Among the 15 instances of terminal conduction delay, there were eight with additional evidence of right ventricular hypertrophy; four were type 1, three were type 2, and one was unclassified. The remaining seven revealed evidence of terminal conduction delay without right ventricular hypertrophy (fig. 7A).

Among the eight proved and five additional
suspected instances of ostium primum, there was no uniform configuration of the QRS sE loop in the horizontal projection. However, the QRS sE loop in the sagittal and frontal plane was characterized by certain distinctive features. The QRS sE loop was displaced.
markedly superiorly, and often to the left (fig. 6). The direction of inscription of the QRS sE loop in the frontal plane was counterclockwise in all patients.

In table 5 are summarized the right ventricular pressure, ratio of the pulmonic blood flow to systemic blood flow, QRS duration, and the various types of vectorcardiograms recorded in the 70 patients studied. The average right ventricular pressure was lowest among those who had normal vectorcardiograms and highest among those who had type 3 right ventricular hypertrophy. No significant differences were found among the group who had terminal conduction delay and types 1 and 2 right ventricular hypertrophy.

The vectorcardiograms were then further divided according to the presence of rsR' or rSr' in V1. These are tabulated in table 6.

Analysis of Postoperative Electrocardiograms and Vectorcardiograms

Sixteen patients had follow-up electrocardiograms and vectorcardiograms 4 months to 4 years after surgical repair. Excluded from the present series are those patients not followed for at least 4 months. The results are summarized in table 7. Fourteen patients were proved to have secundum type defect, confirming the preoperative diagnosis. The defect was thought to be completely closed except in one who had incomplete closure of the defect, and required reoperation.

Two patients had ostium primum defects, one of which was completely closed during open-heart surgery. The diagnosis had been made prior to surgery in each instance.

Electrocardiogram

Among the 13 patients who had satisfactory closure of a secundum type defect, there was slight to marked decrease in the degree of right axis deviation, and a gradual decrease in the height of R or R' or increase in the amplitude of S in the right precordial leads. The configuration of QRS in V1 changed from Rs to rs in one, R to rsR' in one, qRs
Table 7
Analysis of Postoperative Electrocardiograms and Vectorcardiograms

<table>
<thead>
<tr>
<th>No.</th>
<th>Patient</th>
<th>Age</th>
<th>Interval postop.</th>
<th>Axis</th>
<th>QRS duration</th>
<th>QRS V1 pattern</th>
<th>Height of QRS V1</th>
<th>VCG</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>S.B.</td>
<td>8</td>
<td>4 yr.</td>
<td>+125</td>
<td>0.10</td>
<td>rsR'</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>2</td>
<td>J.F.</td>
<td>5</td>
<td>8 mos.</td>
<td>+95</td>
<td>0.09</td>
<td>rsR'</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>3</td>
<td>J.J.</td>
<td>5</td>
<td>1 yr.</td>
<td>+100</td>
<td>0.10</td>
<td>rsR'</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>4</td>
<td>H.L.</td>
<td>38</td>
<td>1½ yr.</td>
<td>+80</td>
<td>0.10</td>
<td>rsR'</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>5</td>
<td>P.M.</td>
<td>3</td>
<td>2½ yr.</td>
<td>+138</td>
<td>0.07</td>
<td>Rs</td>
<td>8</td>
<td>10</td>
</tr>
<tr>
<td>6</td>
<td>J.M.</td>
<td>21</td>
<td>2 yr.</td>
<td>+50</td>
<td>0.11</td>
<td>rs'r's'</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>7</td>
<td>C.N.</td>
<td>15</td>
<td>4 mos.</td>
<td>+100</td>
<td>0.08</td>
<td>rsR'</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>8</td>
<td>O.B.</td>
<td>22</td>
<td>2 yr.</td>
<td>+110</td>
<td>0.12</td>
<td>rsR'</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>9</td>
<td>Z.B.</td>
<td>21</td>
<td>1 yr.</td>
<td>+110</td>
<td>0.10</td>
<td>rsR'</td>
<td>0.5</td>
<td>1</td>
</tr>
<tr>
<td>10</td>
<td>J.O.</td>
<td>33</td>
<td>2 yr.</td>
<td>+100</td>
<td>0.08</td>
<td>R</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>J.P.</td>
<td>34</td>
<td>2½ yr.</td>
<td>+120</td>
<td>0.11</td>
<td>rs'r'</td>
<td>0.5</td>
<td>3</td>
</tr>
<tr>
<td>12</td>
<td>E.H.</td>
<td>27</td>
<td>1½ yr.</td>
<td>+90</td>
<td>0.10</td>
<td>rs'r'</td>
<td>1</td>
<td>3.5</td>
</tr>
<tr>
<td>13</td>
<td>E.C.</td>
<td>14</td>
<td>1 yr.</td>
<td>+45</td>
<td>0.10</td>
<td>Rs'r's'</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>14</td>
<td>C.R.</td>
<td>4</td>
<td>1½ yr.</td>
<td>+120</td>
<td>0.08</td>
<td>qRs</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>15</td>
<td>L.W.</td>
<td>16</td>
<td>2 yr.</td>
<td>+105</td>
<td>0.08</td>
<td>Rs</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>16</td>
<td>S.H.</td>
<td>43</td>
<td>1½ yr.</td>
<td>+110</td>
<td>0.10</td>
<td>qr'</td>
<td>2</td>
<td>13</td>
</tr>
</tbody>
</table>

Note: Patients 6 and 15 had septum primum defects. All others had secundum defects. Age given at time of operation. RVH, right ventricular hypertrophy; TCD, terminal conduction delay; TL, terminal loop.

The initial electrocardiogram in each instance was recorded prior to surgery and the latter at the time specified after surgery.

to rs in one, rsR' to rs'r' in two, Rs'r's' to RS in one, and remained as rSR' in six and as RS in one. The QRS duration showed little if any change postoperatively. No electrocardiographic changes were noted in the patient whose defect was not completely closed. In the one patient who had successful repair of an ostium primum defect, the QRS in V1 changed from rs'r's' to RS. No change was noted in the QRS duration or the left axis deviation. No electrocardiographic changes were noted in the remaining patient with ostium primum whose defect was not closed.

Vectorcardiogram

In the 13 patients who had ostium secundum defects that were successfully repaired, the postoperative vectorcardiogram revealed slight to marked improvement in the degree of right ventricular hypertrophy (fig. 4A and 4B), (table 7). The terminal conduction delay disappeared completely in two cases (fig. 7A and 7B). The degree of terminal conduction delay decreased somewhat in the remaining instances in which there had been terminal conduction delay prior to surgery. No significant vectorcardiographic change was noted in the two patients whose defects were
not repaired. In one patient who had apparently successful repair of a secundum type defect no significant change was seen 2 years postoperatively and the vectocardiogram remained as right ventricular hypertrophy type 2. In one patient who had successful repair of an ostium primum defect, the vectocardiogram in the horizontal plane changed from right ventricular hypertrophy type 1 to normal and the frontal projection revealed persistent counterclockwise rotation with superior displacement of the QRS sE loop.

Discussion

The electrocardiographic diagnosis of right ventricular hypertrophy in congenital heart disease may prove to be difficult when there is a minimal degree of right ventricular hypertrophy or associated terminal conduction delay of the right bundle-branch-block type. This is particularly true in patients with atrial septal defects as compared to other lesions producing right ventricular hypertrophy. The frequency of the rSR' pattern in right precordial leads has been stressed in patients with atrial septal defect. The incidence of this pattern was initially reported to be as high as 95 per cent and the configuration considered as almost diagnostic of atrial septal defect. In more recent studies the reported incidence of rSR' has been somewhat lower, ranging from 60 to 77 per cent.

In the present series of 70 patients, an rSR' configuration was found in 32 instances or 46 per cent. The incidence for both rSR' and rSr' was 60 per cent. The right precordial leads varied rather markedly in configuration (table 2).

A normal configuration, rS in V1 was associated with normal right ventricular pressures and the lowest PBF/SBF ratios. The right ventricular pressures and the PBF/SBF ratios in the rSr' group showed no significant differences from the rS group. In the rSR' group, the right ventricular pressures were moderately elevated and the PBF/SBF ratios were highest. The right ventricular pressure tended to be higher as the amplitude or R or R' in V1 increased. There were, however, many exceptions (fig. 3), and no quantitative assessment could be made by measuring the magnitude of R or R'.

The vectocardiogram has been reported to be highly characteristic in atrial septal defect and useful in both diagnosing right ventricular hypertrophy and in differentiating right ventricular hypertrophy from right bundle-branch block. There was no uniformity in the vectocardiogram in our series. It was, however, valuable in the diagnosis of right ventricular hypertrophy. In the two patients who had normal electrocardiograms, the vectocardiograms were distinctly abnormal and compatible with right ventricular hypertrophy. The vectocardiogram was also abnormal in eight of the 10 patients whose electrocardiograms showed rSr' in V1. This configuration presents considerable difficulty because it is also frequently found in normal persons. Camerini found an rSr' configuration in multiple right precordial leads in 40 of 50 normal subjects. Forty-nine of the 50 normal children studied with multiple right precordial leads by Blount and associates revealed rSr' in at least one of the right chest leads. In their report, this pattern was not observed in VSR but at a higher level or further to the right.

The configuration of the vectocardiogram varied in general with the altered hemodynamics found in atrial septal defects. As the right ventricular pressure increases, the QRS sE vector shifts gradually anteriorly and to the right and the direction of the inscription becomes clockwise. Elek and associates found that the vectocardiogram progressively rotates to the right as right ventricular work increases. Liebman and Nadas stated that the relationship between the vector loop and the ratio of right to left ventricular work correlated substantially better than with any other measure.

The rSR' pattern seen in VSR and V1 in atrial septal defects has long been considered as "incomplete right bundle-branch block." Lasser et al. stated that many of these cases

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are in reality right ventricular hypertrophy by vectorcardiogram. It has been stated\textsuperscript{16, 18-23} that the rSR' pattern seen in atrial septal defect is due to dilatation or hypertrophy of the right ventricle outflow tract rather than interruption of conduction in the right bun-

\textbf{Figure 7A}

\textit{J.J., 5 years old. Ostium secundum. Right ventricular pressure 40 mm. Hg. The vectorcardiogram before operation is characteristic of terminal conduction delay without evidence of right ventricular hypertrophy. The electrocardiogram is rSR' in \textit{V1}. QRS 0.10 second.}
Figure 7B

J.J., 1 year after repair of septal defect. The vectorcardiogram is now normal; the terminal conduction delay is no longer present. The electrocardiogram shows rSR' in V1. QRS duration remains 0.10 second. The height of R' has decreased.

dle branch for the following reasons: 1. Following successful repair of the atrial septal defect, there is a decrease in the voltage of R' in the right precordial leads. 2. As the right ventricular pressure increases, the rSR' configuration may change to R. As right ventricular pressure decreases, the R may change to rSR'. 3. Although the rSR' pattern is predominantly seen in atrial septal defect, it is also seen in cardiac disease of other types.35, 37-40

Following surgical correction of pulmonary valvular stenosis, Blount et al.39 reported that the high R wave in right precordial leads...
evolved to rSR' in three of the five patients after a pulmonary arterial approach. This also occurred in three patients after pulmonary valvotomy in a series of 46 patients reported by Landman.38 No vectorcardiograms were reported in these patients.

Our findings would support the conclusion that rSR' is frequently due to hypertrophy, since the electrocardiographic pattern of rSR' in many instances of atrial septal defect was seen in patients with vectorcardiogram demonstrating types 1 and 2 right ventricular hypertrophy without terminal conduction delay.

Although the QRS duration was 0.10 or 0.11 second in 24 patients with atrial septal defects, only eight of the vectorcardiograms in this group revealed terminal conduction delay of the right bundle-branch-block type. The vectorcardiograms revealed right ventricular hypertrophy without terminal conduction delay in the remaining 16. However, in only one instance of the five with QRS durations of 0.12 second or greater was there no definite terminal conduction delay.

In the presence of terminal conduction delay, the vectorcardiograms were sufficiently characteristic to permit the diagnosis of right ventricular hypertrophy in eight patients. In the remaining seven patients the terminal conduction delay predominated, and the presence of right ventricular hypertrophy could not be determined from the vectorcardiogram.

In two of the five patients who had terminal conduction delay in the vectorcardiograms prior to surgery, there was no evidence of terminal conduction delay following successful repair of the septal defect. In the remaining three patients, evidence of terminal conduction delay persisted despite disappearance of accompanying right ventricular hypertrophy. Serial electrocardiograms revealed gradual decrease in the height of the R or R' deflection in the right precordial leads.

In patients with atrial septal defects, the vectorcardiographic evidence of terminal conduction delay may be produced by hypertrophy of the outflow tract of the right ventricle or ischemia secondary to hypertrophy of the right ventricular outflow tract.

Following successful repair of atrial septal defects, the hypertrophy or ischemia of the right ventricular outflow tract may disappear in some patients but in others may be irreversible. The persistence of terminal conduction delay, despite successful surgery, may be due to organic lesions of the right bundle branch, so that no change would be expected after surgical repair. Davies and associates25 reported that there was no change in the complete right bundle-branch block observed in two patients who had improvement in other respects following repair of atrial septal lesions. In 1941, Blackford and Parker37 reported an instance of pulmonic stenosis with complete right bundle-branch block. They ascribed the intraventricular conduction delay to ischemia secondary to right ventricular hypertrophy.

Taussig,41 Toseano-Barboza,15 and Blount17 were among the first to stress the value of the electrocardiogram in differentiating between ostium secundum and ostium primum types of atrial septal defect. Left axis deviation, counterclockwise rotation, and superior displacement of the QRS sE loop in the frontal plane were described in 1956 by Toseano-Barboza et al.15 as characteristic electrocardiographic and vectorcardiographic findings in patients with ostium primum defects. Subsequently, many other reports have confirmed these findings.

There is no agreement, however, concerning the cause of these electrocardiographic phenomena in ostium primum. Some have ascribed the left ventricular hypertrophy to hemodynamically significant mitral insufficiency while others have attributed the left axis deviation to an alteration in the exciting pathway.15,31 Toseano-Barboza and associates15 stated that mitral insufficiency was not the direct cause of the left axis deviation because (1) newborn children with defects of the atrioventricular canal already demonstrated the diagnostic tracings; (2) the association of pulmonic stenosis did not obscure the unusual excitatory pattern; and (3) the electro-
cardiographic record did not correlate with the size of the left ventricle.

On the other hand, Milnor and Bertrand, Cooley and McNamara, and Campbell and Missen favored the view that the left axis deviation is related to mitral insufficiency. Liang and Schwantje reported three patients with ostium primum defects whose electrocardiograms were interpreted as left ventricular hypertrophy. Significant mitral insufficiency was demonstrated at operation in each instance.

If the left axis deviation, superior displacement, and a counterclockwise rotation of the frontal plane QRS sE loop were to disappear following surgery, it would suggest strongly that these changes were due to left ventricular hypertrophy rather than congenital aberration of the left bundle branch. To our knowledge, however, no sufficient follow-up electrocardiographic and vectorecardiographic study of instances of ostium primum has been reported. In our one patient, left axis deviation persisted following successful surgery. No significant mitral insufficiency was noted in those who had ostium primum defects and underwent surgery. Thus, we believe that alteration of the conduction pathways is an important mechanism for the left axis deviation in instances of ostium primum.

Opinions differ as to the specificity and diagnostic value of the electrocardiographic abnormality. Witham and Ellison reported five instances of clinically diagnosed ostium primum defects only one of which had left axis deviation. Patients with incomplete or atypical ostium primum may not have the characteristic electrocardiogram. However, patients with atrial septal defect combined with ventricular septal defect, or high ventricular septal defect with involvement of the atrial septum, have been reported to have left axis deviation and superior displacement and counterclockwise rotation of the frontal plane QRS sE loop. We have also observed this characteristic electrocardiogram and vectorecardiogram in high ventricular septal defects. There was one instance of a secundum type defect as confirmed at surgery with superior displacement and counterclockwise direction of inscription of the frontal plane QRS sE loop. The electrocardiographic electrical axis in the frontal plane was $-150^\circ$. We have been unable to explain this isolated occurrence.

In the present study, each of the seven patients who were diagnosed as having ostium primum lesions and one patient as ostium secundum and mitral insufficiency had left axis deviation and a superior displacement of the QRS sE loop in the frontal plane. The direction of inscription of the QRS sE loop was counterclockwise in the frontal plane in all of these tracings.

Only one of the proved ostium secundum defects had left axis deviation, $-30^\circ$. This was complicated by mitral insufficiency on a rheumatic basis. The direction of inscription of the QRS sE loop in the frontal plane, however, is not uniform in the ostium secundum defect. Among the 55 patients who had normal or right axis deviation, there were 12 with counterclockwise rotation in the frontal plane. Therefore, the direction of inscription cannot be regarded as a sign for or against ostium primum defects. Despite the occasional exception, we believe that the left axis deviation and the superior displacement of QRS sE loop in the frontal plane are the most valuable features in differentiating ostium primum from ostium secundum defects.

**Summary**

The results of the electrocardiographic, vectorecardiographic, and hemodynamic studies of 70 patients with atrial septal defects are analyzed. Serial electrocardiograms and vectorecardiograms were obtained in 16 patients following surgery.

The incidence of the rSR' and rSr' patterns in the right precordial leads was 60 per cent.

To a limited extent, the configuration of the electrocardiogram is related to right ventricular pressure and PBF/SBF. However, no quantitative assessment could be made by determining the magnitude of R or R' in lead V1.

The vectorecardiogram is an important ad-
junction to the electrocardiogram in detecting right ventricular hypertrophy. As the right ventricular pressure increases, the QRS sE loop tends to shift more to the right and anteriorly.

Although it can be generally stated that the rSR' configuration in atrial septal defects is usually due to right ventricular hypertrophy, this configuration is also due to terminal conduction delay or terminal conduction delay associated with right ventricular hypertrophy in a significant number of instances.

The vectorcardiogram is useful in detecting terminal conduction delay in patients with rSR' in the right precordial leads. It was also of value in determining the presence of right ventricular hypertrophy in combination with terminal conduction delay.

The evidence of terminal conduction delay may disappear after successful repair of atrial septal defects.

Left axis deviation, superior displacement, and counterclockwise direction of inscription of the QRS sE loop in the frontal plane are valuable signs for differentiating ostium primum defects from ostium secundum defects. Each of the eight proved instances of ostium primum defects had these typical features.

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YU-CHEN LEE and LEONARD SCHERLIS

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