The Scalar Electrocardiogram, Vectorcardiogram, and Exercise Electrocardiogram in the Assessment of Congenital Aortic Stenosis

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AORTIC STENOSIS is a relatively common congenital cardiac deformity. At the Congenital Heart Clinic of The Children’s Hospital Medical Center, Boston, this clinical diagnosis has been made in 252 instances in approximately 7,000 patients with the diagnosis of congenital heart disease seen over the past 10 years, representing an incidence of 3.6 per cent.

Congenital aortic stenosis may be of critical severity; it is the one lesion that may lead to sudden death in relatively asymptomatic children and adolescents. Since the advent of the pump-oxygenator, successful surgical relief of aortic obstruction has become possible at relatively low risks.1-3 Accurate assessment of the severity and the anatomic nature of the defect is possible by combined left and right heart catheterization, a time-consuming and even potentially dangerous procedure. It seemed desirable, therefore, to search for clinical means of selection of patients for cardiac catheterization and eventual surgery.

It is generally accepted4,5 that the electrocardiogram is the single most valuable clinical tool in the assessment of the severity of congenital aortic stenosis. Reynolds et al.,6 however, pointed to the occurrence of critical, and even fatal, aortic stenosis in patients with relatively normal electrocardiograms. Since the appearance of this report, we have seen 11 patients with high-grade aortic stenosis, as proved by cardiac catheterization, who have normal electrocardiograms. This is not surprising in view of the reported inaccuracy of the electrocardiogram in the diagnosis of left ventricular hypertrophy.7,8

It is the purpose of this publication to report further on the use of electrocardiographic techniques in the quantitative assessment of aortic stenosis. Ninety-five consecutive patients with aortic stenosis, in whom accurate left and right heart catheterization data were available, have been studied by means of 12-lead electrocardiograms. In 40 of them, vectorcardiograms were obtained by the cube system.9 Thirty-three patients underwent exercise tolerance tests with a variable resistance bicycle ergometer, with recording of lead V₆ precordial electrocardiograms.10 The relative merits of each technique are discussed.

Electrocardiographic Studies

Methods and Materials

The 12-lead scalar electrocardiograms of 95 consecutive patients aged 6 weeks to 20 years with isolated congenital aortic valvular or subvalvular stenosis were analyzed. All patients underwent right and left heart catheterization, the left ventricle being entered on every occasion either by the retrograde arterial approach11,12 or by transthoracic puncture.13 Because of the variability of recording techniques and the absence, in some cases, of simultaneous cardiac output determinations, it was decided to use the peak systolic gradient across the aortic valve as the criterion of severity, although the aortic valve area is a preferable index.

The electrocardiograms were taken within 36 hours of left heart catheterization and the analysis was directed principally toward detection of left ventricular hypertrophy although note was also made of the direction of the QRS and T mean
frontal plane vectors, of the presence or absence of P mitrale, and of the ventricular activation time in lead V6.

The electrocardiogram was designated as showing no left ventricular hypertrophy, if the voltages in precordial leads, reflecting predominantly left ventricular depolarization, did not exceed the 90th percentile of normals. If voltages fell between the 90th percentile and the maximum, the electrocardiogram was described as showing probable left ventricular hypertrophy, and if the voltages exceeded the maximum of normals, the electrocardiogram was designated as showing left ventricular hypertrophy.

Left ventricular hypertrophy with "mild strain" was defined as left ventricular hypertrophy with ST- and T-wave abnormalities confined to leads V5 and V6. If ST- and T-wave abnormalities were present in other leads in addition, the electrocardiogram was designated as left ventricular hypertrophy with "severe strain." Since the oldest patient in this series was 20 years old, and most of the others were children less than 15, the finding of ST- and T-wave changes was considered in each instance to be due to left ventricular hypertrophy. Coronary artery disease and other factors, making interpretation of ST- and T-wave changes difficult, were not thought to be present.

Results

Table 1 illustrates the distribution of electrocardiographic evidence for left ventricular hypertrophy in the 95 patients, arbitrarily divided into two groups, one with a peak systolic gradient of less than 40 mm. Hg and one in which the gradient was 40 mm. Hg or over. Fifty-one (74 per cent) of the 69 patients with significant aortic stenosis (gradient over 40 mm. Hg) showed definite evidence of left ventricular hypertrophy. In contrast, six (24 per cent) of 26 patients with gradients of under 40 mm. Hg did show evidence of left ventricular hypertrophy, whereas the electrocardiograms of the remainder of this group were within normal limits.

Table 2 expresses the analysis of the results in percentage form, the figures indicating the percentage of electrocardiograms showing evidence of left ventricular "strain pattern" for different peak systolic gradients. Using only the presence of ST- and T-wave changes as criteria for identification of patients with gradients of 40 mm. Hg or over, there is a 30 per cent (21 of 69) incidence of "false negatives" and a 12 per cent incidence of "false positives" (3 of 26 cases). The discrepancy between the electrocardiogram and the pressure gradient in one of the "false positives" is probably explainable on the basis of a "damped" withdrawal pressure tracing and in fact, at surgery, severe, discrete, subvalvular stenosis was found. In the other two patients, technically good tracings showed peak systolic gradients of 38 mm. Hg and 20 mm. Hg, respectively, with corre-

Table 1

| Electrocardiographic Evidence for Left Ventricular Hypertrophy in 95 Cases with Aortic Stenosis |
|---------------------------------|--------------------------------------------------|------------------|
| "Mild" aortic stenosis (26 patients), peak systolic gradient 10 to 39 mm. Hg | Significant aortic stenosis (69 patients), peak systolic gradient 40 to 140 mm. Hg |
| No LVH | 8 | 6 |
| Probable LVH | 12 | 12 |
| LVH by voltage | 3 | 3 |
| LVH with mild ST, T changes | 6 (24%) | 19 (74%) |
| LVH with severe ST, T changes | 1 |
| Total | 26 cases (100%) | 69 cases (100%) |

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sponding cardiac outputs of 4.1 and 5.1 L./min./M.².

The mean QRS vector in the frontal plane was within the normal range in 84 instances. There was right axis deviation (+150°, +180°) in two patients, one of whom was a baby of 6 weeks; the other was a 13-year-old boy with an aortic gradient of 75 mm. Hg. Left axis deviation (ranging from −5° to −150°) was found in seven instances. All except one had significant aortic stenosis with gradients higher than 50 mm. Hg. The remaining patient showed multiple abnormalities in the horizontal plane vectorcardiogram as well, but had only a moderate gradient of 20 mm. Hg. These findings are difficult to understand. In general it is clear, however, that extreme leftward deviation of the frontal plane axis is found in patients with severe aortic stenosis, but this is by no means an obligatory relationship, as pointed out before. In the five patients in whom vectorcardiograms were available, a normally directed mean frontal plane axis was found in three, indicating that axis shift may have been due to factors other than left ventricular hypertrophy.

Using the angle between the mean QRS vector and the mean T vector in the frontal plane as a rough indication of the “ventricular gradient,” 31 patients showed an angle of greater than 60°. Twenty-eight of the 31 had T-wave abnormalities readily apparent on the electrocardiogram, and the fact that the “ventricular gradient” was abnormal contributed no further information. Thus, the calculation of the gradient does not seem to contribute information in this group of patients beyond that obtainable from the determination of the direction of the T vector alone.

P mitrale was present in seven patients. Not all had severe aortic stenosis but, in every one, significant mitral regurgitation was demonstrated at cardiac catheterization. The ventricular activation time, measured in lead V₆, varied from 0.02 to 0.06 second and showed no correlation at all with the severity of the stenosis.

In summary then, the most useful aspect of the electrocardiogram in the assessment of aortic stenosis is the detection of left ventricular “strain pattern.” The appearance of secondary ST- or T-wave abnormalities almost always reflects a peak systolic gradient of over 40 mm. Hg. Unfortunately, however, 30 per cent of cases with peak systolic gradients of over 40 mm. Hg had no ST- or T-wave changes and thus would not be detected by electrocardiographic means.

Vectorcardiographic Studies

General Considerations

It has been suggested that the vectorcardiogram (VCG) is more accurate than the standard electrocardiogram in the diagnosis of various cardiac disorders. In fact, Wolff has shown, in 139 instances of left ventricular hypertrophy confirmed at autopsy, that the vectorcardiogram correctly indicated the diagnosis in 106, whereas the electrocardiogram did so in only 68 instances. Some of the reasons for the superior performance of the vectorcardiogram in the diagnosis of left ventricular hypertrophy may be found in the more accurate isolation and determination of the direction of instantaneous

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Table 2

**Incidence of Secondary ST- and T-Wave Abnormalities in Relation to Peak Systolic Gradient in 95 Cases with Aortic Stenosis**

<table>
<thead>
<tr>
<th>Gradient mm. Hg</th>
<th>10-19</th>
<th>20-29</th>
<th>30-39</th>
<th>40-49</th>
<th>50-74</th>
<th>75-70</th>
<th>100 and over</th>
</tr>
</thead>
<tbody>
<tr>
<td>ST, T abnormalities</td>
<td>0%</td>
<td>8%</td>
<td>28%</td>
<td>46%</td>
<td>76%</td>
<td>67%</td>
<td>78%</td>
</tr>
<tr>
<td>No ST, T abnormalities</td>
<td>100%</td>
<td>92%</td>
<td>71%</td>
<td>54%</td>
<td>24%</td>
<td>33%</td>
<td>22%</td>
</tr>
<tr>
<td>Total</td>
<td>26 cases (100%)</td>
<td>69 cases (100%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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vectors. It has been stated, that the bulk of the free left ventricular wall contributes its major influence during the later phases of the QRS complex. This is reflected in the spatial QRS loop by a displacement in a posterior, superior, and leftward direction.\(^{19,20}\)

Therefore the measurements of the 0.03 and 0.035-second vector, as well as the maximum QRS vector, as projected onto the horizontal plane, were principally studied in relation to left ventricular hypertrophy. Such a study of the direction of timed QRS vectors is quite difficult in the scalar electrocardiogram.

**Methods and Materials**

Forty cases with varying degrees of aortic stenosis, all of which belonged to the group of 95 described earlier, were studied with the cube reference system.\(^{21}\) This study was usually done on the same day the electrocardiogram was taken. Seven were also studied with the Frank lead system.\(^{22}\) As normal controls, 45 patients were randomly selected for this kind of analysis from the larger group of children previously studied by vectorecardiographic means.\(^{23}\)

Multiple QRS loops were recorded from the Sanborn vectorecardiograph 185 on XXX Kodak 35-mm film. These films were read in a Doelmat R Rollfilm reader, and the direction of selected vectors was determined with a protractor and scale. The QRS loops were interrupted each 0.0025 second, facilitating the selection of the appropriate instant to be studied. The zero-point was isolated by means of amplified detailed photographs. No vectors were studied beyond 0.06 second. Direction and configuration of T loops were also studied.

This manner of analysis corresponds to that previously used by Pipberger et al. and others,\(^{24,25}\) but differs from an earlier study from this laboratory on these same patients. The usual statistical methods and formulas have been used.\(^{26}\)

**Results: Vectorcardiogram in Aortic Stenosis as Compared to Normal Subjects**

**Horizontal Plane**

Analysis of data in the 45 controls indicates a wide spread in the individual measurements of the initial (0.01, 0.02 second) vectors as well as in the terminal vectors (beyond 0.04 second). The magnitude of this variation, as well as the considerable overlap between this group and the patients with aortic stenosis, precludes their use for comparison. A much smaller variation was found, however, in the direction of the 0.025, 0.03, 0.035 second, and maximum QRS vectors. The standard deviations, for the observations at these instants, were so much reduced from the wide spread found in the initial and final vectors that an artifact, related to the reference system, clearly suggests itself.

Actually, these data do confirm the postulations by Pipberger,\(^{27}\) that the poor representation of cardiac vectors along the anteroposterior or Z axis causes considerable variation in measurements of vectors that are mainly projected upon this axis. For this reason, initial and final vectors, determined by the cube system, appear to contain significant errors.\(^{28}\) In fact, these extreme variations have led to the development of corrected lead systems in which truly equal and orthogonal representations can be expect-

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**Table 3**

Direction (in Degrees) of Selected QRS Vectors in 40 Patients with Aortic Stenosis (Horizontal Plane)

<table>
<thead>
<tr>
<th></th>
<th>0.03-second vector</th>
<th>0.035-second vector</th>
<th>Maximum QRS vector</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean</td>
<td>S.D.</td>
<td>mean</td>
</tr>
<tr>
<td>Normal subjects</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N = 45</td>
<td>10</td>
<td>12.0</td>
<td>359</td>
</tr>
<tr>
<td>Aortic stenosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N = 40 (all patients)</td>
<td>348</td>
<td>43.0</td>
<td>341</td>
</tr>
<tr>
<td>Aortic stenosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N = 29 (patients with</td>
<td>350</td>
<td>—</td>
<td>341</td>
</tr>
<tr>
<td>gradient &gt; 40 mm)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Significance of difference</td>
<td>t = 4.02</td>
<td>p &lt; 0.01</td>
<td>t = 3.12</td>
</tr>
</tbody>
</table>
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Recently published data in normal subjects confirm these expectations, since they show a highly constant standard deviation of ± 25° for each vector analyzed, whereas this statistical guide in the normal subjects studied with the cubic system, varies from 83° (for the 0.02-second vector), to 7.5° (for the maximum QRS). Despite these obvious disadvantages of vectorcardiographic data derived by the cube system, the observations of a narrow standard deviation, for the direction of the 0.025, 0.03, 0.035 second, and maximum QRS vectors, prompted a comparison with the same timed QRS vectors in the group of patients with aortic stenosis.

It may be seen from table 3 that in the horizontal plane the 0.03-second vector is directed at 348° (S.D. 43.0°) in patients with aortic stenosis and is considerably different from the control value at 10° (S.D. 12.0°) (p < 0.01). The same applies for the 0.035-second vector (at 341°, S.D. 22.3°) and its control (359°, S.D. 20°) (p < 0.01), and to an even greater extent for the direction of the maximum QRS vector as it projects on the horizontal plane (339°, S.D. 19.6°) compared to a control value of 11° (S.D. 7.5°) (p < 0.001) (Fig. 1). It may also be seen in table 3 that the mean values, for these three parameters, within the group of significant aortic stenosis (gradient greater than 40 mm. Hg) are closely similar to the group as a whole.

Thus, in this study, left ventricular hypertrophy was considered to be present by vectorcardiographic criteria, when the direction of these three selected QRS vectors was within the range indicated on table 3.

When the direction of the maximum QRS vector was related to left ventricular peak pressure, one of the determinants of left ventricular work, a positive correlation was found (r = 0.67; p < 0.001). In normal subjects the left ventricular peak systolic pressure was estimated on the basis of the normal systolic blood pressure for age. Since cuff pressures in the brachial artery often exceed left ventricular peak pressure, this assumption may overestimate the latter and

thus result in underestimation of the degree of correlation. The relationship is graphically shown in figure 2, where a continuous and progressive trend toward posterior deflection of this vector with increasing left ventricular pressure is seen. The patients with mild degrees of aortic stenosis were found to range between the normal subjects and those with significant aortic stenosis.

The magnitude of the QRS forces was meas-
Figure 2
Relationship of direction of maximum horizontal QRS vector (in degrees) and peak systolic pressure in left ventricle (in mm. Hg) in 45 controls and 40 patients with aortic stenosis.

Figure 3
Distribution of magnitude of maximum QRS vector (in mV.) in the horizontal plane in 45 normal children and 40 patients with aortic stenosis. The mean value for the group with mild aortic stenosis is 1.04 mV. and falls between the mean value of the normal subjects and the over-all group with aortic stenosis.

Figure 4
Relationship of magnitude of maximum horizontal plane QRS vector (in mV.) and peak systolic gradient (in mm. Hg) across the aortic valve in 45 controls and 40 patients with aortic stenosis. A gradient of 1 mm. Hg has been assumed to exist in the normal.

addition, that those patients with gradients of less than 40 mm. Hg show a mean value of 1.04 mV., which falls between the distribution curves of both major groups. Thus, a positive relationship between voltage of the maximum QRS vector and the aortic valve gradient is found. This is shown better in figure 4, where a significant correlation was calculated, for all patients studied (the control group and the group with aortic stenosis), between voltage of the maximum QRS vector in the horizontal plane and aortic gradient (r = 0.79, p < 0.001). When the left ventricular peak systolic pressure was used, an equally significant correlation with the magnitude of the maximum QRS vector was
found \( r = 0.77, \ p < 0.001 \) (fig. 5). In both figures 2 and 5 it would have been preferable to express the values along the ordinate as units of left ventricular work; however, the lack of data on cardiac output in the normals, and in some of the patients with aortic stenosis, precluded this calculation.

**Sagittal and Frontal Planes**

Analysis similar to that outlined for the horizontal plane was performed. Again, a wide spread of the initial QRS forces was encountered. Relatively narrow ranges were found for the sagittal plane maximum vector and for the 0.03- and 0.035-second vectors. No significant difference from the normal could, however, be calculated. The frontal plane maximum axis and the 0.03 vector were entirely within normal limits. The 0.035-second vector showed a very wide spread.

In summary, aortic stenosis causes changes in the spatial QRS loop best represented in its projection on the horizontal plane. These changes consist of an increase in magnitude, as well as in posterior deflection of these forces. These abnormalities are best seen at 0.03 second, 0.035 second after the beginning of the QRS loop, and at the time of the maximum excursion of the loop in the horizontal plane. They relate in a positive fashion to left ventricular pressure work and thus permit an assessment of the severity of the aortic valve lesion. Rautaharju and Blackburn\(^{32}\) found a similar significant relation between increased electrocardiographic amplitudes and spatial vector changes and elevated systemic blood pressure in 468 adults. In an earlier study from this laboratory\(^{33}\) a similar correlation was found between voltage measured in the R wave in lead V\(_1\) and the right ventricular systolic pressure in patients with pulmonic stenosis and intact ventricular septum. These results indicated that right ven-

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tricular pressures could be estimated by the magnitude of depolarization forces over the right precordium.

Comparison of Vectorcardiogram and Electrocardiogram

In 28 of the 29 patients with significant aortic stenosis, a diagnostic abnormality was present in the vectorcardiogram, whereas the electrocardiogram definitely led to the correct diagnosis of only 18 (62 per cent) of the 29. Table 4 indicates the relative incidence of QRS and T-wave abnormalities.

Particular interest centered on 11 of the 29 patients with gradients of over 40 mm. Hg, in whom normal or nearly normal electrocardiograms were found (table 5, fig. 6). Although in five of these 11, the R wave in leads V₅ and V₆ was greater than the 90th percentile, in no case did the voltage of the R wave in these leads exceed the maximum normal values observed.¹⁴ Furthermore, in none of these patients were ST- or T-wave changes present, thus precluding the definite diagnosis of left ventricular hypertrophy. Ten of these 11 patients demonstrated vectorcardiograms diagnostic of left ventricular hypertrophy as defined earlier; nine by QRS and T-loop changes, one by T-loop changes alone. The remaining patient (G.H., table 5) showed a gradient of 50 mm. Hg with a cardiac output, as determined by the dye-dilution technic, of 3.14 L./min./M.². From these data, an aortic valve area of 0.50 em.² was calculated. Thus, in one of the 29 patients with significant aortic stenosis, the vectorcardiogram as well as the electrocardiogram failed to indicate the true severity of the lesion. In all others, however, the vectorcardiogram led to an appro-

Table 4

Comparison of Vectorcardiogram and Electrocardiogram for Evidence of Left Ventricular Hypertrophy in 40 Patients with Aortic Stenosis

<table>
<thead>
<tr>
<th>% Positive evidence for left ventricular hypertrophy</th>
<th>29 patients with aortic valve gradient of 40 mm. Hg and over</th>
<th>11 patients with aortic valve gradient less than 40 mm. Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>By vectorcardiogram</td>
<td>24/29</td>
<td>9/11</td>
</tr>
<tr>
<td>By QRS-loop and T-loop changes only</td>
<td>28 (97%)</td>
<td>9 (82%)</td>
</tr>
<tr>
<td>By T-loop changes only</td>
<td>4/29</td>
<td>0/11</td>
</tr>
<tr>
<td>By electrocardiogram</td>
<td>17/29</td>
<td>0/11</td>
</tr>
<tr>
<td>By QRS complex and S-T changes alone</td>
<td>18 (62%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>By T-wave changes only</td>
<td>1/29</td>
<td>0/11</td>
</tr>
</tbody>
</table>

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Table 5

Comparison of Hemodynamic, Vectorcardiographic, and Electrocardiographic Data on 11 Patients with Severe Aortic Stenosis, in Whom the Electrocardiogram Was Normal

| Patient | Age | Left ventricular peak systolic pressure (mm. Hg) | Aortic valve area cm.² | Direction of 0.03-sec. vector (Degrees) | Direction of 0.035-sec. vector (Degrees) | Magnitude of QRS vector (m.v.) | Character of T loop | Over-all evidence | Height of R Vs (mm.) | Height of R Vs (mm.) | ST segment | T-wave configuration | Over-all evidence |
|---------|-----|-----------------------------------------------|------------------------|-------------------------|--------------------------------------|----------------------------------|---------------------|------------------|------------------|------------------|------------------|-----------------|-------------------|------------------|
| R.P.    | 12  | 110                                           | 109                    | 4.8                     | 0.33                                 | 3.04                            | 2.0                 | A               | +               | 28              | 15               | N               | N                 | —                |
| R.McN.  | 9   | 130                                           | 50                     | 3.14                    | 0.6                                  | 3.34                            | 1.4                 | A               | +               | 24              | 22               | N               | N                 | —                |
| G.H.    | 9   | 130                                           | 164                    | 0.5                     | 0.31                                 | 1.5                             | A                   | +               | 26              | 18               | N               | N                | —                |
| R.B.    | 9   | 130                                           | 165                    | 0.6                     | 0.31                                 | 1.5                             | A                   | +               | 24              | 18               | N               | N                | —                |
| E.B.    | 11  | 140                                           | 130                    | 0.9                     | 0.31                                 | 1.5                             | A                   | +               | 24              | 18               | N               | N                | —                |
| M.P.    | 13  | 210                                           | 120                    | 0.4                     | 0.31                                 | 1.5                             | A                   | +               | 24              | 18               | N               | N                | —                |
| J.P.    | 5   | 150                                           | 175                    | 0.5                     | 0.31                                 | 1.5                             | A                   | +               | 24              | 18               | N               | N                | —                |
| D.S.    | 13  | 150                                           | 150                    | 0.6                     | 0.31                                 | 1.5                             | A                   | +               | 24              | 18               | N               | N                | —                |
| J.R.    | 17  | 150                                           | 150                    | 0.5                     | 0.31                                 | 1.5                             | A                   | +               | 24              | 18               | N               | N                | —                |
| W.R.    | 10  | 150                                           | 150                    | 0.6                     | 0.31                                 | 1.5                             | A                   | +               | 24              | 18               | N               | N                | —                |
| J.S.    | 13  | 150                                           | 150                    | 0.7                     | 0.31                                 | 1.5                             | A                   | +               | 24              | 18               | N               | N                | —                |

*Pressure gradient obtained from technically unsatisfactory withdrawal tracing. On recatheterization, a gradient of 60 mm. Hg was found with a left ventricular peak systolic pressure of 170 mm. Hg.

A, abnormal; N, normal.
could be recognized in every instance, except one, where cardiac catheterization demonstrated a gradient of over 40 mm. Hg. Furthermore, the detection of quantitative differences in magnitude as well as altered directions of horizontal plane vectors, becoming progressively more abnormal as left ventricular pressure rises, allowed, in most instances, good separation of patients with severe aortic stenosis from those with mild stenosis.

**Lead V₆ Exercise Electrocardiogram**

**General Considerations**

As a third device to measure the severity of aortic stenosis, the exercise electrocardiogram was evaluated. Graduated increases in exercise are generally assumed to increase...
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Response of the Electrocardiogram Recorded in the V6 Position to Maximal Work in Patients with Aortic Stenosis

<table>
<thead>
<tr>
<th>Peak systolic aortic valve gradient mm. Hg</th>
<th>Normal ST-T at rest and with exercise (No. patients)</th>
<th>Ischemic changes present at rest and exercise (No. patients)</th>
<th>Ischemic changes developing with exercise (No. patients)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>75-130</td>
<td>1</td>
<td>5</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>40-74</td>
<td>4</td>
<td>7</td>
<td>2</td>
<td>13</td>
</tr>
<tr>
<td>10-39</td>
<td>11</td>
<td>1</td>
<td>0</td>
<td>12</td>
</tr>
<tr>
<td>Total</td>
<td>16</td>
<td>13</td>
<td>4</td>
<td>33</td>
</tr>
</tbody>
</table>

Discussion

The criteria used in the present study are those outlined by Bengtsson, who studied the exercise electrocardiogram in 84 healthy children aged 5 to 14 years. Abnormal responses in lead V6 are considered to be the appearance of T-wave inversion when it was previously upright or biphasic, the appearance of a flat or biphasic T wave when it was previously upright, and ST depression of 2 mm. or greater. In addition, the occurrence of precordial pain with simultaneous change in the contour of the ST segment in one child was considered to be an ischemic response.

In table 6 it will be seen that only in four of the 33 patients did exercise provide evidence of myocardial ischemia when none was suspected by the resting electrocardiogram. In one of these patients, though the peak systolic gradient was recorded as 40 mm. Hg, it was thought to be higher because of an artifact on the withdrawal tracing (McN, table 5). In each of these four patients, however, the vectorcardiogram had already led to the diagnosis of significant left ventricular hypertrophy.

Table 6 also indicates that in five patients with significant aortic valve gradients no ST- or T-wave changes occurred during or after the exercise.

In summary, we think that the exercise electrocardiogram can only be expected to reveal ischemic changes, not present on the resting electrocardiogram, if the stenosis is

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Cardiac work. If a discrepancy between coronary blood supply and the increased myocardial mass results, the relative ischemia has been assumed to express itself by changes in cardiac repolarization, i.e., ST- and T-wave changes. These are best seen in the precordial electrocardiogram. In addition, the response of the cardiac rate to an increased work load, has been used for assessment of cardiac reserve.

Methods and Materials

Thirty-three children, whose ages ranged from 8 to 18 years, were exercised to their maximal working capacity (as defined by Bengtsson) on a bicycle ergometer. Most of them were patients selected from the group studied by the vectorcardiogram. The equipment used was the Elema-Schonander D.C. variable-resistance bicycle ergometer. This apparatus works on the principle that it allows the subject to perform work (pedaling) against a resistance that is accurately known. Four electrocardiographic electrodes were connected to the limbs and a fifth one was placed in the lead V6 precordial position. The work load was increased every 2 minutes and the electrocardiogram from V6 was recorded at 2-minute intervals, and, intermittently, for 5 minutes after completion of the work. The exercise was terminated when the subject became exhausted or developed significant ischemic changes in the electrocardiogram, or complained of precordial pain. In normal subjects the test was also terminated when a maximal plateau in acceleration of cardiac rate was attained. This level was infrequently reached in our subjects and hence was not used as an end point. Good cooperation was obtained, the children regarding the test as more of a challenge than a chore.

Results

Table 6 indicates the changes observed in the ST segment and T waves immediately after cessation of maximal work. The cardiac rate reached at this load varied considerably and did not lend itself to further analysis.
of severe degree. It does not allow the con-
clusion, however, that no significant aortic
stenosis is present when the electrocardiogram
remains normal after exercise.

Summary and Conclusions
The standard electrocardiogram, the cube
vectorcardiogram, and lead V₆ exercise elec-
trocardiogram have been evaluated in patients
with congenital aortic stenosis, assessed at
cardiac catheterization.

The most reliable indicators of significant
aortic stenosis in the standard electrocardio-
gram are ST- and T-wave changes in left pre-
cordial leads in the presence of increased
voltage of the R wave. In 26 per cent of 69
cases with peak systolic gradients of more
than 40 mm. Hg, these changes were absent.
It should be emphasized, that all these pa-
tients were children or young adults. Changes
in repolarization are obviously of lesser im-
portance in older patients, since the possibil-
ity of coronary artery disease always exists.

Vectorcardiograms were recorded in 40
patients and compared with those obtained
from 45 normal subjects. A significant differ-
ence was found in the direction of QRS vec-
tors at 0.03, 0.035 second, as well as at the
time of maximum excursion, of the QRS loop
in the horizontal plane in almost all patients
with aortic stenosis, irrespective of severity.
Furthermore, a positive correlation could be
demonstrated between the left ventricular
systolic peak pressure and the direction and
magnitude of the maximum QRS vector in
the horizontal plane. These findings indicate
that increased left ventricular pressure work
may be assessed by measurement of the over-
all electrical activity at the time of maximum
leftward and posterior excursion of the QRS
loop.

In 10 of the 11 cases of severe aortic
stenosis with normal or equivocal electrocar-
diograms, the vectorcardiograms showed de-
finite evidence for significant left ventricular
hypertrophy.

Finally, the exercise electrocardiogram was
found helpful in that in four patients with
normal resting electrocardiograms abnormali-
ties occurred suggesting myocardial ischemia.
A normal exercise electrocardiogram did not
always indicate normal hemodynamics, how-
ever.

It appears, therefore, that in the routine
clinical assessment of aortic stenosis the elec-
trocardiogram remains the most useful screen-
ing procedure. The cube vectorcardiogram,
however, has proved to be of great accuracy
in selecting those patients who, despite a
normal electrocardiogram, have significant
aortic stenosis. Both changes in direction and
magnitude of vectors are factors not readily
available in the routine electrocardiogram,
and can be used in a quantitative sense to
identify patients in whom cardiac catheteri-
ization is imperative. This technic should find
its place beside other routine procedures and
permit a reliable assessment of this frequent
cardiac disorder.

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