**Ventricular Septal Defect in Infancy: A Combined Vectorgraphic and Echocardiographic Study**

**THOMAS RIGGS, M.D., SUDHIR MEHTA, M.D., STEPHEN HIRSCHFELD, M.D., GORDON BORKAT, M.D., AND JEROME LIEBMAN, M.D.**

Summary

Echocardiograms (echo) and vectorcardiograms (VCG) from 40 infants with ventricular septal defects (VSD) were compared with cardiac catheterization data to assess noninvasively the hemodynamics of VSD. The specific aim was to use VCG parameters of right ventricular hypertrophy and echo parameters which reflect pulmonary artery pressure to identify all patients with a nonrestrictive VSD. The configuration of the QRS vector in the horizontal plane was more reliable than individual voltages in assessing right ventricular systolic pressure. Among patients older than 2 months with a clockwise or anterior two-main-vector horizontal loop, 73% (eight of 11) had a nonrestrictive VSD. However, a counterclockwise or posterior two-main-vector loop was also frequently found (43%, six of 14) in infants with a nonrestrictive VSD. The most useful echo parameter was the ratio of right ventricular pre-ejection period-to-right ventricular ejection time (RPEP/RVET), which closely ($r = 0.74$) reflected the pulmonary artery diastolic pressure. An elevated RPEP/RVET to greater than 0.30 was always associated with a nonrestrictive VSD, although many patients (36%, five of 14) with a nonrestrictive VSD had a normal ratio. By combining both echo and VCG parameters, a nonrestrictive VSD was correctly identified in all patients, while a restrictive VSD was correctly identified in 81% (21 of 26).

Isolated Ventricular Septal Defect (VSD) is one of the most common forms of congenital heart disease. The complex hemodynamic changes associated with a VSD are a function of both its size and the resistance and compliance of the pulmonary and systemic circulations. Right ventricular (RV) systolic pressure is primarily determined by the size of the VSD, but resistance and compliance of the pulmonary vascular bed are also factors. Similarly, the extent of left-to-right shunting depends on the size of the VSD and the relative resistance of the pulmonary and systemic circulations. Most infants with large defects develop congestive heart failure (CHF) in the first months of life and may require sequential cardiac catheterization to assess the size of the VSD, the amount of left-to-right shunting and the pulmonary vascular resistance (PVR) as a prerequisite to corrective surgery.

The hemodynamics of infants with a VSD are often assessed by echocardiography and vectorcardiography. The echocardiogram (echo) has been used to estimate the pulmonary-to-systemic flow ratio (Qp/Qs) and the pulmonary artery diastolic pressure (PADP). The vectorcardiogram (VCG) has been suggested as a means of assessing RV pressure and PVR, but few reports have correlated the degree of right ventricular hypertrophy (RVH) with hemodynamic data in infants with VSD or the degree of left ventricular hypertrophy (LVH) with Qp/Qs.

In this study we report the specificity and sensitivity of the echo and VCG in assessing the hemodynamics of VSD in infancy. This combined noninvasive approach, using both echocardiography and vectorcardiography, has not been previously evaluated.

**Methods and Subjects**

The study group was composed of 40 patients (18 males and 22 females) less than 1 year of age (range 1–12 months) in whom an isolated VSD was confirmed by cardiac catheterization. Each infant underwent cardiac catheterization based on the development of CHF and/or clinical evidence of significant pulmonary hypertension. Those with CHF were treated with digitalis and diuretics, as needed. Four patients had significant left-to-right shunts at atrial level, defined as a persistent step-up of at least 5% $O_2$ saturation in the right atrium. The diagnosis of a patent foramen ovale, rather than a true atrial septal defect, was made in three of these four infants, since they had a difference in mean arterial pressures of at least 3 mm Hg, implying a restrictive defect. The infant with a true atrial defect had no difference in mean atrial pressures.

Premedication for the catheterization was either droperidol (0.1 mg/kg) or meperidine hydrochloride and hydroxyzine hydrochloride (1–1.5 mg/kg each) for all infants older than 2 months. Younger patients were not premedicated. Light anesthesia, when necessary, was maintained with either intravenous ketamine hydrochloride (1 mg/kg) or intravenous diazepam (0.1 mg/kg).

The pressure recordings were obtained with either standard Swan-Ganz, Lehman or NIH catheters. The

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From the Department of Pediatrics, Case Western Reserve University, School of Medicine, and Rainbow Babies and Children's Hospital, Cleveland, Ohio.

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Address for reprints: Thomas Riggins, M.D., Department of Pediatrics, Case Western Reserve University, School of Medicine, and Rainbow Babies and Children's Hospital, 2101 Adelbert Road, Cleveland, Ohio 44106.

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catheters were connected to Bell and Howell strain gauges and the pressures were recorded on an Electronics for Medicine optical recorder. Oxygen saturations and hemoglobin concentrations were measured with a cooximeter (Instrumentation Lab, Inc). All measurements were made before angiography.

The echocardiograms were obtained with either a Hoffrel or Unirad ultrasonoscope. The recording of right and left ventricular ejection times (RVET and LVET, respectively) and right ventricular pre-ejection period (RPEP) has been described. The end-systolic and end-diastolic internal dimensions of the left ventricle (LVES and LVED) and the end-diastolic internal RV size (RVED) were each measured to the nearest millimeter as described by Meyer, with end-diastole defined by the Q wave of the ECG. The end-systolic dimensions of the left atrium (LA) and aorta (Ao) were also measured.

Frank VCGs were obtained using an Instrument for Cardiac Research (ICR) Instant Vectorcardiograph, with the patient in the supine position. From the recorded VCG, the following voltage amplitudes were measured: 1) maximum Z anterior (ZA), 2) maximum Z posterior (ZP), 3) maximum X to the left (XL), 4) maximum terminal X to the right (XTR), and 5) maximum Y inferior (YI). The maximal right and left spatial voltages (RMSV and LMSV) were computed manually.

We also noted the direction of the QRS loop in the horizontal plane. The diagnosis of RVH was made when either the ZA or XTR exceeded 2 standard deviations above the age-specific value, or when the horizontal loop was clockwise (CW) in an infant older than two months. The diagnosis of LVH was made when either the ZP or XL was more than 2 standard deviations above the age-specific value.

Standard ECGs, obtained within 24 hours of the Frank VCG, were available from all infants. In most cases (35 of 40) these ECGs were recorded using a simultaneous six-lead ECG machine (Minograf-61, Siemens, Inc). The horizontal QRS loop was then reconstructed from the six-channel ECG, by recording the anteroposterior and left-to-right amplitudes at 0.01-second intervals from leads V4 and V5.

The echo and VCG studies were always done within 24 hours of cardiac catheterization. Each patient's height, weight, echo, VCG and hemodynamic data

### Table 1. Summary of Hemodynamic Data

<table>
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<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>sd</td>
<td>Mean</td>
<td>sd</td>
</tr>
<tr>
<td>RVP</td>
<td>82</td>
<td>(12)</td>
<td>85</td>
<td>(17)</td>
</tr>
<tr>
<td>LVP</td>
<td>95</td>
<td>(15)</td>
<td>99</td>
<td>(14)</td>
</tr>
<tr>
<td>RV/LV</td>
<td>0.87</td>
<td>(0.10)</td>
<td>0.86</td>
<td>(0.10)</td>
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<tr>
<td>Qp/Qs</td>
<td>3.9</td>
<td>(1.0)</td>
<td>4.1</td>
<td>(1.1)</td>
</tr>
<tr>
<td>PAPM</td>
<td>45</td>
<td>(11)</td>
<td>41</td>
<td>(12)</td>
</tr>
<tr>
<td>PAPD</td>
<td>28</td>
<td>(11)</td>
<td>21</td>
<td>(5)</td>
</tr>
</tbody>
</table>

Abbreviations: LVP = peak left ventricular systolic pressure; PAPD = pulmonary artery diastolic pressure; PAPM = pulmonary artery mean pressure; Qp/Qs = pulmonary-to-systemic flow ratio; RV/LV = ratio of peak right and left systolic pressures; RVP = peak right ventricular systolic pressure.

### Table 2. Summary of Echocardiographic Data

<table>
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<td>1.13</td>
<td>(0.09)</td>
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<tr>
<td>LA/m²</td>
<td>6.12</td>
<td>(1.00)</td>
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<tr>
<td>LVED</td>
<td>2.48</td>
<td>(0.37)</td>
<td>2.10</td>
<td>(0.48)</td>
</tr>
<tr>
<td>LVEDR</td>
<td>122%</td>
<td>(14%)</td>
<td>97%</td>
<td>(14%)</td>
</tr>
<tr>
<td>RVED</td>
<td>1.4</td>
<td>(0.3)</td>
<td>1.8</td>
<td>(0.4)</td>
</tr>
<tr>
<td>RPEP/RVET</td>
<td>0.32</td>
<td>(0.07)</td>
<td>0.29</td>
<td>(0.06)</td>
</tr>
<tr>
<td>HR</td>
<td>139</td>
<td>(15)</td>
<td>125</td>
<td>(21)</td>
</tr>
<tr>
<td>Vef</td>
<td>1.98</td>
<td>(0.39)</td>
<td>1.61</td>
<td>(0.30)</td>
</tr>
<tr>
<td>%SID</td>
<td>38%</td>
<td>(7%)</td>
<td>34%</td>
<td>(6%)</td>
</tr>
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</table>

Abbreviations: HR = heart rate; LA/Ao = left atrial-to-aortic dimension ratio in end-systole; LA/m² = left atrial end-systolic dimension, indexed by body surface area; LVED = left ventricular end-diastolic dimension; LVEDR = left ventricular end-diastolic dimension, expressed as a ratio of expected normal value; RVED = right ventricular end-diastolic dimension; RPEP/RVET = the ratio of right ventricular pre-ejection period to right ventricular ejection time; %SID = percent shortening of internal left ventricular dimension; Vef = velocity of circumferential fiber shortening.
TABLE 3. Summary of Significance Levels of Noninvasive Parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group A-1</th>
<th>Group A-2</th>
<th>Group B</th>
<th>Group C</th>
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<tbody>
<tr>
<td>ECHO</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>LA/Ao</td>
<td>†</td>
<td>NS</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>LA/m²</td>
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<td>NS</td>
<td>†</td>
<td>†</td>
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<tr>
<td>LVED</td>
<td>†</td>
<td>NS</td>
<td>†</td>
<td>†</td>
</tr>
<tr>
<td>RVET</td>
<td>†</td>
<td>†</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>RPEP/RVET</td>
<td>†</td>
<td>†</td>
<td>NS</td>
<td>NS</td>
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<td>%SID</td>
<td>NS</td>
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<td>NS</td>
<td>NS</td>
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<tr>
<td>Vef</td>
<td>†</td>
<td>†</td>
<td>†</td>
<td>†</td>
</tr>
<tr>
<td>VCG</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ZA</td>
<td>*</td>
<td>NS</td>
<td>*</td>
<td>†</td>
</tr>
<tr>
<td>ZP</td>
<td>NS</td>
<td>†</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>XL</td>
<td>†</td>
<td>NS</td>
<td>*</td>
<td>†</td>
</tr>
<tr>
<td>XTR</td>
<td>*</td>
<td>†</td>
<td>†</td>
<td>†</td>
</tr>
<tr>
<td>YI</td>
<td>NS</td>
<td>NS</td>
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<td>NS</td>
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<tr>
<td>RMSV</td>
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</tr>
<tr>
<td>LMSV</td>
<td>†</td>
<td>NS</td>
<td>†</td>
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</table>

* p < 0.001.
† p < 0.01.
‡ p < 0.05.
NS p > 0.05.

(Comparisons are made with normal controls.)

Abbreviations: LA/Ao = left atrial-to-aortic dimension ratio in end-systole; LA/m² = left atrial end-systolic dimension, indexed by body surface area; LMSV = left maximal spatial voltage; LVED = left ventricular end-diastolic minor dimension; RMSV = right maximal spatial voltage; RPEP/RVET = ratio of right ventricular pre-ejection period to right ventricular ejection time; RVED = right ventricular end-diastolic minor dimension; %SID = percent shortening of left ventricular minor dimension; Vef = velocity of circumferential shortening; VCG = vectorcardiograms, using the Frank lead system; XL = maximal leftward voltage; XTR = terminal rightward voltage; YI = maximal inferior voltage; ZA = maximal anterior voltage; ZP = maximal posterior voltage.

were recorded on computer punch cards and then analyzed with a PDP-11 computer (Digital Equipment Corporation). The following parameters were computed: RPEP/RVET, left atrial-to-aortic dimension in end-systole (LA/Ao), left atrial end-systolic dimension (LA/m²), percent shortening of internal left ventricular dimension (LV) (%SID) and velocity of circumferential shortening (Vcf). Normal values for pediatric patients, based on weight and/or age, have been described for LVED,10 RVED,11 %SID,11 Vcf,11 LA/Ao,8 LA/m² and RPEP/RVET,12,13 and served as controls.

The value of each parameter was expressed as mean ± sd, while some parameters were also expressed as standard deviations above or below the normal mean. (e.g., z = (x - m) / s, where x = actual value, m = normal population mean, s = normal population's standard deviation and z = standard deviation units, or critical ratio).20 The statistical analyses included simple and multiple linear regression (BMD program 02R),21 the two-tailed t test and Fisher's exact test. Unless otherwise stated, the level of significance was specified as p < 0.05.

Results

Hemodynamic Data (table 1)

The patients were divided into three groups by RV/LV pressure ratios. Those with a nonrestrictive VSD (RV pressure at least 75% of systemic pressure) were designated group A. Those with RV pressures less than 50% of systemic level were designated group C, and those with RV pressures between 50–75% of systemic level were designated group B. Within group A were 10 patients with isolated VSD (group A-1) and four patients with VSD and significant left-to-right shunting at atrial level (group A-2). There were 11 patients in group B and 15 in group C.

Echo Data (tables 2, 3 and 4)

LA/Ao and LA/m²

Although the mean LA/Ao was significantly elevated in all groups except A-2, the correlation between Qp/Qs and LA/Ao was only fair, and did not allow accurate prediction of Qp/Qs (figs. 1 and 2). In group A-2 patients, the smaller than expected LA/Ao could be explained by atrial left-to-right shunts (fig. 3). Similarly, the mean LA/m² was elevated in all...
groups except A-2, but the correlation with Qp/Qs remained only fair. The difference between actual and predicted Qp/Qs appeared equally wide at high and relatively moderate values of Qp/Qs. For Qp/Qs < 3 the mean difference was 1.03, while for Qp/Qs > 4, the average difference was 1.09.

LVED

The mean LVED was increased significantly in all groups except A-2. The only infants with LVED in the normal range (within 2 standard deviations of their predicted LVED) were infants having a Qp/Qs < 2.5 or patients belonging to group A-2. However, the correlation between Qp/Qs and LVED enlargement was poor.

RVED

The RVED was always increased to greater than 2 standard deviations above the normal value in patients with atrial shunts (group A-2). Three of these four infants had abnormal interventricular septal motion. The RVED was usually normal (28 of 36) in the remaining groups, although an enlarged RVED was more often encountered in group A-1 (four of 10) than in groups B (three of 11) or C (one of 15).

**Figure 1.** The relationship of the pulmonary-to-systemic flow ratio (Qp/Qs) and the end-systolic left atrial-to-aortic dimension ratio (LA/Ao) is demonstrated. Although the LA/Ao increases with increasing Qp/Qs, the variability around the regression line is large. Four infants had ventricular septal defect (VSD) and additional significant left-to-right shunts at atrial level (squares) and had almost normal LA/Ao. Excluding these infants from the analysis, the correlation remained only fair (r = 0.36).

**Figure 2.** Echocardiogram from an infant with an isolated ventricular septal defect. The end-systolic left atrial dimension (LA) and end-systolic and end-diastolic dimensions of the left ventricle (LV) are enlarged, while the end-diastolic dimension of the right ventricle (RV) is normal. The enlarged LA and LV predict the presence, but not the extent, of the left-to-right shunt. The ventricular dimensions show some respiratory variation in this example and part of the tricuspid valve is visualized on the right side of the interventricular septum. Ao = end-systolic aortic dimension.
VSD IN INFANCY/Riggs et al.

FIGURE 3. Echocardiogram from an infant with a ventricular septal defect and atrial left-to-right shunt. The endsystolic left atrial-to-aortic dimension ratio is almost normal and the right ventricle (RV) is enlarged. In another view, the interventricular septum moved paradoxically. LV = left ventricle; Ao = aorta; LA = left atrium.

%SID and Vcf

The mean %SID was not elevated in any group, and it did not correlate well with Qp/Qs. The mean Vcf was elevated in all groups except A-2, but correlated poorly with Qp/Qs.

RPEP/RVET

A RPEP/RVET > 0.30 always indicated a large VSD (group A-1 or A-2), but several infants in these groups (five of 14) had a ratio ≤ 0.30. The correlation between PADP and the RPEP/RVET was very good, particularly when a second-degree term of RPEP/RVET was included (figs. 4 and 5). The RPEP/RVET correlated less well with RV pressure and mean pulmonary artery (PA) pressure, and each see (18 and 11 mm Hg, respectively) was too large to allow accurate prediction of these pressures.

VCG Data (tables 3, 4, and 5)

RVH

Although 38 of 40 had RVH, individual parameters were not helpful in quantitating RV hypertension.

ZA

The mean ZA was elevated in all groups except A-2. The group with the greatest mean ZA was B, the group with intermediate RV hypertension. The correlation of ZA with RV pressure or RV/LV pressure ratio was not significant.

XTR

The mean XTR was elevated in all groups, and tended to increase with increasing RV hypertension. However, the correlation of XTR with RV pressure was poor.

FIGURE 4. Pulmonic valve echocardiograms from two infants demonstrate the measurement of right ventricular systolic time intervals. The right ventricular preejection period (PEP) is measured from the Q wave of the ECG to the opening of the pulmonic valve, while right ventricular ejection time (VET) is measured from opening to closing of the pulmonic valve. Ratio = ratio of right ventricular PEP to right ventricular VET.
RMSV

The mean RMSV was elevated in groups A-1 and B and normal in groups A-2 and C. The correlation of RMSV with RV pressure was insignificant.

Horizontal Configuration

Thirteen of 40 infants had either a CW or two-main-vector and anterior VCG loop in the horizontal plane (fig. 6), implying predominant RVH. We observed this pattern more often in group A (eight of 14) than in group B (three of 10) or group C (two of 15). The difference in proportions between groups A and C is significant ($p < 0.01$), but the difference between groups A and B or B and C were not significant. Although a CW or anterior two-main-vector-horizontal VCG loop was usually associated with a large or intermediate-size VSD, two infants less than 2 months old from group C had CW loops.

LVH

Twenty-eight of 40 infants had LVH, although the degree of LVH was not predictive of the extent of left-to-right shunting.

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**Table 5. Summary of Vectorcardiographic Data**

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<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>sd</td>
<td>Mean</td>
<td>sd</td>
</tr>
<tr>
<td>ZA</td>
<td>1.68 (0.77)</td>
<td>0.90 (0.61)</td>
<td>2.18 (1.0)</td>
<td>1.48 (0.44)</td>
</tr>
<tr>
<td>ZP</td>
<td>1.10 (0.71)</td>
<td>0.30 (0.33)</td>
<td>1.07 (0.64)</td>
<td>1.05 (0.77)</td>
</tr>
<tr>
<td>XL</td>
<td>1.80 (1.26)</td>
<td>1.53 (0.70)</td>
<td>2.36 (1.66)</td>
<td>2.12 (0.76)</td>
</tr>
<tr>
<td>XTR</td>
<td>1.67 (1.12)</td>
<td>1.04 (0.52)</td>
<td>1.23 (0.74)</td>
<td>0.88 (0.59)</td>
</tr>
<tr>
<td>YI</td>
<td>1.25 (0.66)</td>
<td>0.45 (0.13)</td>
<td>1.27 (0.46)</td>
<td>1.26 (0.78)</td>
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<tr>
<td>RMSV</td>
<td>2.01 (0.73)</td>
<td>1.17 (0.59)</td>
<td>1.78 (0.50)</td>
<td>1.33 (0.68)</td>
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<tr>
<td>LMSV</td>
<td>2.37 (1.16)</td>
<td>1.59 (0.73)</td>
<td>2.84 (0.44)</td>
<td>2.67 (0.60)</td>
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<tr>
<td>ZA*</td>
<td>3.31 (2.36)</td>
<td>0.73 (1.86)</td>
<td>4.30 (3.07)</td>
<td>2.15 (1.36)</td>
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<tr>
<td>ZP*</td>
<td>-0.04 (1.92)</td>
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<td>-0.11 (1.99)</td>
<td>-0.14 (2.02)</td>
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<tr>
<td>XL*</td>
<td>1.46 (3.79)</td>
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<td>2.60 (3.81)</td>
<td>1.93 (1.98)</td>
</tr>
<tr>
<td>XTR*</td>
<td>2.85 (3.21)</td>
<td>1.51 (1.52)</td>
<td>2.21 (2.56)</td>
<td>1.03 (1.26)</td>
</tr>
<tr>
<td>YI*</td>
<td>0.17 (1.33)</td>
<td>-0.37 (0.44)</td>
<td>0.12 (0.90)</td>
<td>0.11 (1.44)</td>
</tr>
</tbody>
</table>

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*Voltages expressed in standard deviation units above or below the expected normal value.

Abbreviations: LA/Ao = left atrial-to-aortic dimension ratio in end-systole; LA/m² = left atrial end-systolic dimension, indexed by body surface area; LMSV = left maximal spatial voltage; LVEDR = left ventricular end-diastolic dimension, expressed as a ratio of the expected normal value; PAPM = pulmonary artery diastolic pressure; PADP = pulmonary artery diastolic pressure; PAPM = pulmonary artery mean pressure; Qp/Qs = pulmonary-to-systemic blood flow ratio; RMSV = right maximal spatial voltage; RPEP/RVET = ratio of right ventricular pre-ejection period to right ventricular ejection time; RV = right ventricular peak systolic pressure; RV/LV = ratio of right-to-left ventricular systolic pressures; %SID = percent shortening of left ventricular minor dimension; Vef = velocity of circumferential shortening; XL = maximal leftward voltage; XTR = maximal terminal rightward voltage; YI = maximal inferior voltage; ZA = maximal anterior voltage; ZP = maximal posterior voltage.
XL

The mean XL was increased in all groups except A-2. Groups A-1 and B had comparable values for Qp/Qs, but differed significantly in mean XL. In addition, group C, which had the lowest mean Qp/Qs, had a larger mean XL than group A-1. The correlation of XL with Qp/Qs was poor.

ZP

The mean ZP was normal in all groups except A-2, and correlated poorly with Qp/Qs.

YI

The mean YI was normal in all groups and correlated insignificantly with Qp/Qs.

LMSV

The mean LMSV was elevated in all groups except A-2, and was most increased in those patients with the intermediate VSD size (group B). The correlation of Qp/Qs with LMSV was poor.

Horizontal Configuration

Twenty-seven of 40 infants had predominant LVH either by a counterclockwise (CCW) or two-main-vector horizontal VCG loop which crossed through or was posterior to the origin (fig. 6). This pattern was most frequent in group C (13 of 15 patients) and group B (eight of 11). It was seen less often in group A (six of 14). These proportions were significantly different for groups A vs C; other combinations were not significantly different. Among the infants with a CCW or two-main-vector and posterior loop neither ZA, XTR nor RMSV were useful in differentiating groups A, B and C. Infants in groups A-1 and A-2 with predominant LVH had a mean Qp/Qs that was insignificantly higher than those with predominant RVH (4.1 vs 3.8).

Multiple Correlations and Comparisons (tables 4, 6 and 7)

The prediction of PADP, mean PA and RV pressures by RPEP/RVET were not significantly improved when ZA and XTR were also considered. The correlation of Qp/Qs with LA/Ao, LVED (expressed with respect to the normal) and XL was slightly better than either parameter alone, but the total correlation was not predictive.

A CW or anterior two-main-vector horizontal vector loop was observed in most (eight of 14) patients with a nonrestrictive VSD and in a few (five of 26) patients with a restrictive VSD. Thus, in the prediction of a nonrestrictive VSD, a CW or anterior two-
main-vector loop had a 10% false positive rate and a 43% false negative rate. A RPEP/RVET > 0.30 was calculated in nine of 14 patients with a nonrestrictive VSD and was never calculated in patients with a restrictive VSD. Thus, an elevated RPEP/RVET could be predictive of a large VSD with a 0% false positive rate and 36% false negative rate. When these two criteria were combined so that a nonrestrictive VSD was predicted by either an elevated RPEP/RVET or an anterior two-main-vector or CW vector loop, the false negative rate decreased to 0% and the false positive rate remained 19%. This allocation of the patients was highly significant (table 7).

VCG and ECG

The construction of the horizontal VCG loop from the standard ECG recording was accurately done in all patients with a CW or CCW pattern. However, in those patients with two main vectors (12 of 40), the site of crossing of the horizontal loop with respect to the origin was frequently different from the Frank VCG.

Discussion

The natural history of VSD in childhood is usually benign, although when the defect is large, so that pulmonary hypertension is severe, the clinical course may be malignant. Although cardiac catheterization in infants is relatively safe, it does have some risk. Therefore, a reliable, noninvasive exam that clearly identifies infants with large VSDs would be helpful in patient management. Patients with a large VSD require cardiac catheterization and perhaps corrective surgery in infancy. Infants with a restrictive VSD whose CHF was controlled might be studied at an older age when cardiac catheterization could be done more safely.

This study differs from previous echo studies in that our patients were younger, had larger defects with greater left-to-right shunts, and many had CHF. We found LA/Ao and LA/m² have about equal, but only fair, value in predicting Qp/Qs in our patients. Bloom and Lewis found a linear relationship between LA/Ao and Qp/Qs, but many of their patients had a Qp/Qs < 2. We examined the upper portion of the LA/Ao vs Qp/Qs regression line, where all patients had a large Qp/Qs and elevated LA/Ao. When we analyzed the data from Bloom’s study for patients with Qp/Qs greater than 1.9, we found a correlation of 0.26, consistent with our value.

A simple linear relationship between LA/Ao and Qp/Qs implies that the increased flow passing through the LA is proportional to a single echo dimension. Our study suggests that this relationship weakens with a large Qp/Qs. Yabek et al. found that the relationship between echo LA dimension and angiographically determined left atrial volumes changed when LA volume overload occurred. The relationship of LA volume to flow is further complicated by variable LV end-diastolic pressure and LA compliance. Both LA/Ao and LA/m² correctly identified most of our patients with significant left-to-right shunts, but they were not useful in accurately predicting Qp/Qs. In addition, the presence of an atrial shunt allows LA decompression and may reduce LA/Ao and LA/m² toward normal.

Errors in calculating Qp/Qs by the Fick method increase as the extent of left-to-right shunting increases. However, the variability about a linear model (fig. 1) appeared constant in the range of Qp/Qs studied, suggesting that errors in calculating the Qp/Qs did not significantly influence the results.

The increased LVED in most patients was secondary to left ventricular volume overload, while patients with atrial shunting had relatively less ventricular shunting and a smaller LVED. The poor correlation between LVED (expressed with respect to the normal LVED) and Qp/Qs may be secondary to the changing shape of the left ventricle in left ventricular volume overload, or the occurrence of diastolic shunting, or the influence of tachycardia, rather than enhanced stroke volume, to increase LV output.

The increased RVED and abnormal septal motion in infants with atrial shunts was consistent with right ventricular volume overload. This finding, coupled

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**Table 6. Summary of Vectorcardiographic and Echocardiographic Data**

<table>
<thead>
<tr>
<th></th>
<th>Group A (N = 14)</th>
<th>Group B (N = 11)</th>
<th>Group C (N = 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ECHO</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RVSTI &gt; 0.30</td>
<td>9 (64%)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>RVSTI ≤ 0.30</td>
<td>5 (36%)</td>
<td>11 (100%)</td>
<td>15 (100%)</td>
</tr>
<tr>
<td><strong>VCG</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CW or Anterior</td>
<td>8 (57%)</td>
<td>3 (27%)</td>
<td>2* (13%)</td>
</tr>
<tr>
<td>CCW or Posterior</td>
<td>6 (43%)</td>
<td>8 (73%)</td>
<td>13 (87%)</td>
</tr>
</tbody>
</table>

*Both infants were less than 2 months of age.

Abbreviations: CCW = counterclockwise; CW = clockwise; Echo = echocardiographic; RVSTI = ratio of right ventricular pre-ejection period to right ventricular ejection time; VCG = vectorcardiographic.

**Table 7. Summary of False Positive and False Negative Results**

<table>
<thead>
<tr>
<th></th>
<th>Group A</th>
<th>Groups B &amp; C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Either VCG or Echo positive*</td>
<td>14/14 (100%)</td>
<td>5/26 (19%)</td>
</tr>
<tr>
<td>Both VCG and Echo negative†</td>
<td>0/14 (0%)</td>
<td>21/26 (81%)</td>
</tr>
</tbody>
</table>

p <0.0001 by Fisher’s exact test.

*A positive test means that either the horizontal VCG loop is clockwise or two-main-vector and anterior to the origin at its crossover point or that the ratio of right ventricular pre-ejection period to right ventricular ejection time is ≥ 0.30.

†A negative test means that the ratio of right ventricular pre-ejection period to right ventricular ejection time is < 0.30 and that the horizontal VCG loop is counterclockwise or has two main vectors and the crossing point is through or posterior to the origin.

Abbreviations: Echo = echocardiogram; VCG = Frank vectorcardiogram.
with the smaller than expected LA/Ao, suggests that an additional atrial left-to-right shunt may be suspected in an infant with a VSD. The result that some infants with an isolated VSD had a large RVED is analogous to angiographic RV volume studies.27

Echocardiographic parameters of contractility, %SID and Vcf, were either normal or enhanced, despite clinical CHF. The normal %SID in association with a dilated LVED would imply an enhanced stroke volume. Tachycardia, rather than increased %SID, was responsible for the increased Vcf. The increased Vcf was consistent with a previous study,28 but could not be used in predicting the amount of left-to-right shunting in VSD. In the small group of infants with atrial shunts, the %SID and Vcf are difficult to interpret because of the abnormal interventricular septal motion.

The RPEP/RVET is an extremely specific but poorly sensitive test in the identification of infants with a nonrestrictive VSD, since it is most closely associated with PADP, rather than PA or RV systolic pressure. The nonlinear relationship between PADP and RPEP/RVET is very similar to our previous reports, which included patients with a variety of congenital and acquired diseases.10,11 Although the correlation between PA systolic or mean pressure and RPEP/RVET is not strong enough to predict these pressures, estimation of PADP is feasible. Sequential examination of patients may allow timing of cardiac catheterization or corrective surgery to coincide with early evidence of increasing PADP. As a qualitative test in individual patients, a RPEP/RVET ≤ 0.30 is almost always associated with a normal PADP, while a RPEP/RVET > 0.35 almost always implies a PADP > 20 mm Hg, provided that complete right bundle branch block and cardiomyopathy are not present.11 Intermediate values of RPEP/RVET include a few patients with normal PADP and many with moderate elevation of their PADP. Use of the RPEP/RVET ratio depends upon clear visualization of the opening and closing of the pulmonic valve. In our experience, this usually can be accomplished in infants. In a technically satisfactory echo, the error in measurement of RVET is about 5 msec, while the error in RPEP/RVET is about 0.02.13

The Frank VCG is theoretically a more accurate reflection of cardiac depolarization than the standard ECG.17 Proximity effect, a significant problem in interpreting the ECG of an infant with cardiomegaly, is theoretically less in the orthogonal Frank lead system.29 In addition, the recording of the VCG allowed analysis of the configuration of the horizontal loop, which was sometimes difficult to reconstruct accurately from the standard ECG recording.

Most infants with a significant VSD have ECG evidence of both RVH and LVH. While RVH results from RV hypertension and occasional volume overload, LVH is a function of volume overload. The imprecise relationship between hemodynamic and VCG parameters may reflect the fact that each voltage amplitude (e.g., ZA) represents a sum of right and left ventricular forces.

The voltage amplitude parameters of RVH were poorly correlated with RV pressures. Although the ZA is often used clinically to assess RVH, its correlation with RV pressure in this study was very poor. We cannot substantiate Elliott's findings11 of a good correlation (r = 0.65) between RMSV and RV systolic pressure.

In infants older than 2 months, severe RVH is manifested in the horizontal plane by a large XTR and ZA and a CW or two-main-vector orientation, while mild RVH is manifested by a less accentuated XTR and ZA with a CCW orientation. We found the configuration of the horizontal VCG loop was more valuable than individual voltage parameters in assessing RV pressure. All infants older than 2 months with predominant RVH had RV pressures > 50% systemic, and frequently > 75% systemic. However, many infants with large defects had predominant LVH, and thus falsely negative tests. Therefore, a CW or anterior two-main-vector loop is a fairly specific but insensitive indicator for a large VSD. Elliott et al.12 observed that all of their patients older than 2 months who had either CW or two-main-vector loops had RV systolic pressures > 60 mm Hg. However, no mention was made of the site of crossing of the horizontal loop. Perhaps many of them had loops which crossed anteriorly, as in our patients.

In a comparable study, Elliott et al.12 found a mean LMSV (2.57 mV) similar to ours and stated that the correlation of LMSV with Qp/Qs was poor. The LMSV and XL may be counterbalanced by the relative RVH, since the group with the least RV hypertension (C) had higher mean values for these voltages than did the group with the greatest RV hypertension (A-1), despite a lower mean Qp/Qs. Similarly, groups A-1 and B (which had a comparable mean Qp/Qs) showed a mean XL which increased as RV pressure decreased. The LMSV may also vary inversely with the degree of LV chamber enlargement, as in adults.30-32

By multiple regression analysis of several VCG and echo parameters we were not able to predict RV pressure accurately. However, by using a combination of echo and VCG findings, we could retrospectively identify all 14 infants with a nonrestrictive VSD, with a relatively low false positive rate (19%). This combination of RPEP/RVET > 0.30 and/or predominant RVH of the VCG loop therefore increased the sensitivity of noninvasive assessment of VSD size.

The complex hemodynamic changes in infants with a significant VSD are reflected by their abnormal VCG and echo results. Previous studies9,12 have reported separate use of VCG and echo in infants with VSD. However, we found that one apparently cannot use a single test to predict reliably the size of either the VSD or the Qp/Qs in infants. In this report we used the VCG and echo results together to predict hemodynamics, rather than comparing these noninvasive tests with the physical examination or chest roentgenogram. We believe the physical examination is a very useful in assessing the hemodynamics of infants with VSD. The number of infants in group C
would have been much greater had we not made the diagnosis of a small VSD on clinical grounds. The infants in groups A and B were not as easy to differentiate, particularly in the presence of CHF and tachycardia. Our experience also suggests that the roentgenographic chest examination allows a less reliable estimate of the Qp/Qs than the echo LA/Ao ratio. Prospective experience with a combined VCG-echo technique may allow reliable identification of infants who are at greatest risk of developing pulmonary vascular obstructive disease during infancy.

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