Effect of Site of Shunt on Left Heart-Volume Characteristics in Children with Ventricular Septal Defect and Patent Ductus Arteriosus

By M. M. Jarmakani, M.D., Thomas P. Graham, Jr., M.D., Ramon V. Canent, Jr., M.D., Madison S. Spach, M.D., and M. Paul Capp, M.D.

SUMMARY
Quantitative cineangiocardio graphic technics have been utilized to determine left ventricular (LV) and left atrial (LA) volumes and LV muscle mass in 58 patients with isolated ventricular septal defect, 14 of whom were infants less than 2 years old, and in 25 patients with isolated patent ductus arteriosus, 13 of whom were infants. Patients were divided according to the degree of left-to-right shunt into small (<35%), moderate (35 to 50%), and large (>50%) shunt groups. Data obtained in both the VSD and the PDA groups were compared with normal values. Left ventricular end-diastolic volume and mass and LA maximal volumes in the patients of both groups who had shunts of 35% or more were greater than normal and showed a linear increase with increasing left-to-right shunt. The LV ejection fraction was decreased from normal in infants with either VSD or PDA and a shunt of more than 50%. This variable was normal in older children with either VSD or PDA.

Patients with an aortic or a ventricular defect and equivalent shunts did not differ significantly in terms of LV end-diastolic volume, LV mass, LV ejected fraction, or LA maximal volume, normalized for body surface area. Patients with a patent ductus demonstrated the following differences when compared with patients with ventricular septal defect: (1) elevated LV end-diastolic pressure, (2) elevated LV end-diastolic stress, and (3) elevated value for LV mass (/m² BSA) per degree of shunt in children over 2 years of age with a patent ductus (P < 0.05). These results indicate that left ventricular distensibility is decreased in patients with aortic left-to-right shunts compared to that in patients with ventricular left-to-right shunts of equivalent magnitude.

Additional Indexing Words: Left ventricular function Ventricular volume Ventricular mass Atrial volume

The hemodynamic alterations in patients with an isolated ventricular septal defect or a patent ductus arteriosus are determined primarily by two independent factors: (1) the dynamic size of the defect and (2) the ratio of pulmonary-to-systemic vascular resistance. These two variables determine the resistance to left ventricular ejection through the defect, and thus, the degree of left-to-right shunt and pulmonary blood flow. Children with these defects present with clinical evidence of varying degrees of left cardiac dilatation and hypertrophy in association with an increase in pulmonary flow. The magnitude of these changes in the characteristics of left heart volume resulting from ventricular septal defect (VSD) compared to patent ductus arteriosus (PDA) has not been investigated in children.
This study was conducted (1) to describe quantitatively the characteristic features of left ventricular and left atrial volumes of infants and children with either isolated ventricular septal defect or patent ductus, (2) to evaluate how such characteristics differ from normal, and (3) to determine whether there are distinguishable variations in the left heart response to a left-to-right shunt at the ventricular versus aortic level.

Methods

Patient Groups

Eighty-three patients with an isolated VSD or PDA were studied. Included in the group were 27 infants (less than 2 years old) and 56 older children. All of the infants had large left-to-right shunts (more than 50% of pulmonary blood flow) and were on digitalis therapy for a recent or past episode of congestive heart failure. Fourteen patients had isolated VSD, and 13 had isolated PDA. The older children were divided according to the degree of left-to-right shunt into three groups with mild shunts (less than 35%), moderate shunts (35 to 50%), and large shunts (more than 50% of pulmonary blood flow). This age group included 44 children with ventricular septal defect and 12 patients with patent ductus.

Excluded from the study were patients with (1) a systolic pressure gradient from right ventricle to pulmonary artery greater than 30 mm Hg, (2) pulmonary vascular resistance (PVR) more than 6.0 mm Hg/L/min/m² (RP/RS greater than 0.50), or (3) aortic or mitral insufficiency as demonstrated by aortic and left ventricular cineangiography. Left-to-right shunts were measured by the Fick method 5 to 10 min before the first biplane cineangiogram which was used for volume calculation.

Data Acquisitions

The methods have been described previously in detail. In summary, all data were obtained during routine cardiac catheterization. Patients older than 6 months of age received light nitrous oxide general anesthesia. Those less than 6 months old were studied after premedication only.* All studies were done with the patient in the supine position. Right and left heart pressures were recorded shortly before the first cineangiogram, and during cineangiography. Left and right ventricular pressures were recorded by using either a catheter tip transducer+ or an NIH catheter.

Left ventricular (LV) and left atrial (LA) volumes and LV mass were calculated from the first biplane cineangiograms of the study, which were filmed at least 50 min after anesthesia was initiated. Following the injection of 1.25 ml/kg of body weight of 75% Hypaque M§ into the main pulmonary artery, cines were exposed in the anteroposterior (AP) and lateral (LAT) projections at 60 frames/sec and were recorded on 16 mm film. The output of a special photocell device (to indicate each cine pulse), the electrocardiogram, and the LV pressure were recorded simultaneously on a multichannel oscillograph at a paper speed of 200 mm/sec and on magnetic tape. This provided a means for precise localization of each cine frame within the cardiac cycle. A calibrated grid was filmed at the end of each study to correct for x-ray magnification.

The first two to three beats on the levogram were used for calculating volume. During this period the LV pressure was stable, and peripheral effects of the contrast media had not taken place. The increase in end-diastolic volume due to the volume of contrast medium injected is felt to be small and minimized by using pulmonary artery injections. All studies were performed under similar conditions.

All volumes were calculated according to the area-length method of Dodge and associates, assuming an ellipsoid figure of revolution as reference. Left ventricular mass was calculated according to the method described by Rackley and associates. Left ventricular volume and mass were corrected according to regression equations derived previously in our laboratory. Left atrial and LV volumes were calculated at ventricular end diastole and end systole. LV mass (LVM) was calculated at end diastole. Other details and abbreviations are as follows:

1. LV stroke volume (LVSV) = LV end-diastolic volume (LVEDV) – LV end-systolic volume (LVESV).

2. LV ejection fraction (LVEF) = LVSV/LVEDV.

3. LV systolic index = LVSV × heart rate/body surface area (BSA).

In VSD patients, the LV systolic index is assumed to be equal to the pulmonary blood flow minus any diastolic left-to-right shunt across the defect. In PDA patients, the LV systolic index is equal to the pulmonary blood flow.

4. LV minute work/BSA (LV min W/m²) = LVSV × (LV mean systole pressure – LV end-diastolic pressure [LVEDP]) × heart rate/BSA.

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*Meperidine, promethazine, and chlorpromazine.
+SF-1, Statham Products, Inc.

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Sodium and meglumine diatrozoates, Winthrop Laboratory, New York, New York.

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5. LV end-diastolic stress* (LVEDS), at the equator of the ventricle (hoop stress), as described by Sandler and Dodge7:

\[
\text{Hoop stress} = \frac{Pr}{h} \left[ 1 - \frac{r^3}{\frac{LL^2}{4} (2r + h)} \right]
\]

when \( P \) is the left ventricular end-diastolic pressure, \( r \) is the diastolic LV internal radius at the equator, \( h \) is the LV lateral wall thickness at end-diastole, \( LL \) is the longest length of the left ventricle.

6. LA maximal volume (LA max) = LA volume at ventricular end systole.

7. LVEDV/LVM = Ratio of left ventricular end-diastolic volume to LV mass.

All volume and mass data were corrected for body surface area to normalize the data for patients of different age and size.1 The data acquired in these patients were compared with similar measurement in normal infants and children.1

**Results**

The results for infants (less than 2 years old) with ventricular septal defect and patent ductus arteriosus are presented in table 1, and data in children (more than 2 years old) with either a VSD or a PDA are shown in table 2. The data can be compared with normal values appearing in these tables.

The left ventricular end-diastolic volume (LVEDV) in all patients with either VSD (fig. 1) or PDA and a left-to-right shunt of more than 35% was greater than normal (tables 1 and 2). While the LVEDV/BSA was 1.8 times normal for older children with a large shunt due to either VSD or PDA (table 2), for infants it was 2.5 times normal (table 1). Although the normalized values in the two age groups (LVEDV/BSA) were not different, the infants showed a greater relative increase in volume because of the lower standards for normal infants.1 The LVEDV/BSA showed a linear increase with increasing left-to-right shunt as well as with increasing left ventricular systolic index \((P < 0.001)\) in both VSD (fig. 2) and PDA patients.

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* Circumferential force per unit cross sectional area of wall at the equator of the left ventricle.
The left ventricular ejection fraction (LVEF) in infants with either VSD or PDA and a large shunt was similar and was significantly less than normal (P < 0.02). The heart rate was not significantly different in normals from that of infants with VSD or PDA (table 1). In older children with VSD or PDA, the LVEF was not different from normal (table 2).

The left ventricular systolic index in infants and older children with VSD or PDA and shunt of more than 35% was increased significantly (tables 1 and 2).

The left ventricular minute work was also higher than normal in all patients with left-to-right shunts greater than 35%.

Left ventricular wall thickness “lateral wall” (LV h/BSA) in all patients with either VSD or PDA was significantly higher (P < 0.05) than normal (tables 1 to 3).

The left ventricular wall mass was significantly increased above normal in all patients with either VSD or PDA, (fig. 3; tables 1 and 2). In both infants and older children with VSD or PDA, the LV mass showed a linear increase with increasing left-to-right shunt (P < 0.001). Similarly, the LV mass increased

![Figure 3](http://circ.ahajournals.org/lookup/suppl/doi:10.1161/01.CIR.71.4.373A/-/DC1/fig3)

**Figure 3**

Left ventricular wall mass (LVM) as a function of body surface area (BSA) in 39 patients with a VSD and a moderate or large shunt. LVM = 99.2 (BSA) + 13.9 g, 1 sd = 16.7 g, r = 0.866, P < 0.001. Solid line indicates the regression line in normals and dashed lines indicate the 1 sd.
### Table 2

**Hemodynamic and Volume Data on Children (More Than Years 2 Old): Normal Children and Patients With Ventricular Defects and With Patent Ductus Arteriosus**

<table>
<thead>
<tr>
<th></th>
<th>Normals: N = 16</th>
<th>L-R shunt &lt; 35% N = 10</th>
<th>L-R shunt 35-50% N = 16</th>
<th>L-R shunt &gt; 50% N = 18</th>
<th>PDA: N = 12</th>
<th>L-R shunt &lt; 35% N = 5</th>
<th>L-R shunt 35-50% N = 4</th>
<th>L-R shunt &gt; 50% N = 3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (yr)</strong></td>
<td>7.9 ± 3.8</td>
<td>6.4 ± 3.1</td>
<td>7.1 ± 3.0</td>
<td>5.7 ± 2.5</td>
<td>5.8 ± 2.5</td>
<td>4.7 ± 2.2</td>
<td>3.7 ± 1.5</td>
<td></td>
</tr>
<tr>
<td><strong>Weight (kg)</strong></td>
<td>27.3 ± 15.0</td>
<td>21.3 ± 5.6</td>
<td>22.2 ± 9.2</td>
<td>17.4 ± 4.5</td>
<td>18.4 ± 5.1</td>
<td>16.0 ± 5.5</td>
<td>12.9 ± 3.6</td>
<td></td>
</tr>
<tr>
<td><strong>BSA (m²)</strong></td>
<td>0.98 ± 0.43</td>
<td>0.83 ± 0.16</td>
<td>0.85 ± 0.22</td>
<td>0.73 ± 0.17</td>
<td>0.75 ± 0.20</td>
<td>0.67 ± 0.21</td>
<td>0.59 ± 0.17</td>
<td></td>
</tr>
<tr>
<td><strong>Heart rate</strong></td>
<td>101 ± 18</td>
<td>100 ± 13</td>
<td>108 ± 16</td>
<td>107 ± 19</td>
<td>104 ± 18</td>
<td>114 ± 10</td>
<td>120 ± 14</td>
<td></td>
</tr>
<tr>
<td><strong>Peak RV pressure (mm Hg)</strong></td>
<td>27 ± 10</td>
<td>32 ± 8</td>
<td>54 ± 9</td>
<td>55 ± 23</td>
<td>28 ± 9</td>
<td>40 ± 15</td>
<td>51 ± 10</td>
<td></td>
</tr>
<tr>
<td><strong>Peak LV pressure (mm Hg)</strong></td>
<td>103 ± 10</td>
<td>101 ± 15</td>
<td>108 ± 11</td>
<td>106 ± 22</td>
<td>104 ± 13</td>
<td>109 ± 10</td>
<td>118 ± 3</td>
<td></td>
</tr>
<tr>
<td><strong>LVEDP (mm Hg)</strong></td>
<td>10 ± 3</td>
<td>11 ± 4</td>
<td>11 ± 2</td>
<td>13 ± 3</td>
<td>13 ± 4</td>
<td>11 ± 3</td>
<td>19 ± 3</td>
<td></td>
</tr>
<tr>
<td><strong>L-R shunt (%)</strong></td>
<td>0</td>
<td>19 ± 3</td>
<td>43 ± 5</td>
<td>61 ± 8</td>
<td>25 ± 7</td>
<td>39 ± 5</td>
<td>64 ± 12</td>
<td></td>
</tr>
<tr>
<td><strong>MPAP — LVEDP (mm Hg)</strong></td>
<td>6 ± 5</td>
<td>8 ± 5</td>
<td>16 ± 18</td>
<td>19 ± 12</td>
<td>11 ± 10</td>
<td>9 ± 8</td>
<td>37 ± 19</td>
<td></td>
</tr>
<tr>
<td><strong>PVR (mm Hg/L/min/m²)</strong></td>
<td>1.8 ± 1.2</td>
<td>1.6 ± 1.1</td>
<td>2.9 ± 2.9</td>
<td>1.7 ± 1.5</td>
<td>1.8 ± 1.1</td>
<td>1.3 ± 1.1</td>
<td>3.9 ± 1.5</td>
<td></td>
</tr>
<tr>
<td><strong>LVEDV/BSA (cc/m²)</strong></td>
<td>71 ± 8</td>
<td>79 ± 8</td>
<td>100 ± 14</td>
<td>126 ± 22</td>
<td>86 ± 11</td>
<td>104 ± 11</td>
<td>129 ± 30</td>
<td></td>
</tr>
<tr>
<td><strong>LV ejection fraction</strong></td>
<td>0.63 ± 0.05</td>
<td>0.60 ± 0.07</td>
<td>0.59 ± 0.08</td>
<td>0.61 ± 0.05</td>
<td>0.61 ± 0.06</td>
<td>0.71 ± 0.06</td>
<td>0.58 ± 0.03</td>
<td></td>
</tr>
<tr>
<td><strong>LV systolic index (L/min/m²)</strong></td>
<td>4.51 ± 0.88</td>
<td>4.98 ± 1.03</td>
<td>6.26 ± 1.83</td>
<td>8.27 ± 2.36</td>
<td>5.41 ± 1.30</td>
<td>8.30 ± 1.56</td>
<td>8.9 ± 2.6</td>
<td></td>
</tr>
<tr>
<td><strong>LV min W/BSA (kg/min/m²)</strong></td>
<td>4.76 ± 0.46</td>
<td>5.96 ± 1.03</td>
<td>7.12 ± 2.0</td>
<td>9.48 ± 2.08</td>
<td>7.22 ± 1.61</td>
<td>10.41 ± 1.31</td>
<td>11.63 ± 3.11</td>
<td></td>
</tr>
<tr>
<td><strong>LVM/BSA (g/m²)</strong></td>
<td>82 ± 10</td>
<td>98 ± 12</td>
<td>119 ± 26</td>
<td>135 ± 24</td>
<td>95 ± 8</td>
<td>128 ± 17</td>
<td>155 ± 22</td>
<td></td>
</tr>
<tr>
<td><strong>LV h/BSA (mm/m²)</strong></td>
<td>6 ± 1</td>
<td>7 ± 1</td>
<td>8 ± 2</td>
<td>9 ± 2</td>
<td>7 ± 1</td>
<td>9 ± 1</td>
<td>10 ± 1</td>
<td></td>
</tr>
<tr>
<td><strong>LVEDS (g/cm²)</strong></td>
<td>38 ± 12</td>
<td>33 ± 14</td>
<td>42 ± 19</td>
<td>39 ± 12</td>
<td>49 ± 14</td>
<td>34 ± 9</td>
<td>57 ± 15</td>
<td></td>
</tr>
<tr>
<td><strong>LA max (cc/m²)</strong></td>
<td>34 ± 10</td>
<td>45 ± 7</td>
<td>63 ± 11</td>
<td>73 ± 17</td>
<td>40 ± 7</td>
<td>53 ± 10</td>
<td>74 ± 19</td>
<td></td>
</tr>
<tr>
<td><strong>LVEDV/LVM</strong></td>
<td>0.91 ± 0.12</td>
<td>0.81 ± 0.08</td>
<td>0.84 ± 0.19</td>
<td>0.90 ± 0.19</td>
<td>0.90 ± 0.12</td>
<td>0.81 ± 0.08</td>
<td>0.83 ± 0.19</td>
<td></td>
</tr>
</tbody>
</table>

**Abbreviations:** BSA = body surface area; LVEDP = LV end-diastolic pressure; L-R shunt = left-to-right shunt (% of pulmonary blood flow); MPAP = mean PA pressure; PVR = pulmonary vascular resistance; LVEDV = LV end-diastolic volume; LVEF = LV ejection fraction; LV systolic index = LV systolic cardiac output/BSA; LV min W = LV minute work; LVM = LV mass; LV h = LV lateral wall thickness; LVEDS = LV end-diastolic stress; LA max = LA maximal volume.
Table 3

Left Ventricular Volume, Mass, and Wall Thickness Normalized for Shunt

<table>
<thead>
<tr>
<th>Variable</th>
<th>Infants: age &lt;2 yr</th>
<th>Children: age &gt;2 yr</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>VSD: shunt &gt; 50% N = 14</td>
<td>PDA: shunt &gt; 50% N = 13</td>
</tr>
<tr>
<td>(LVEDV/BSA)/shunt (cc/m²/% shunt)</td>
<td>189 ± 49</td>
<td>162 ± 53</td>
</tr>
<tr>
<td>(LVM/BSA)/shunt (g/m²/% shunt)</td>
<td>260 ± 39</td>
<td>243 ± 53</td>
</tr>
<tr>
<td>(LV h/BSA)/shunt (mm²/m²/% shunt)</td>
<td>26 ± 5</td>
<td>22 ± 6</td>
</tr>
</tbody>
</table>

Abbreviations: (LVEDV/BSA)/shunt = LV end-diastolic volume/body surface area/shunt; (LVM/BSA)/shunt = LV mass/body surface area/shunt; (LV h/BSA)/shunt = LV wall thickness/body surface area/shunt.

(P < 0.001) linearly with an increase in the LV systolic index (fig. 4).

To compare the LV volume, LV wall thickness, and LV mass in patients with PDA with those in patients with VSD for equal shunts, LVEDV/BSA, LV h/BSA, and LVM/BSA were divided by the degree of left-to-right shunt. These data (table 3) showed: (1) no difference in LVEDV between patients with PDA and VSD; (2) LV mass and lateral wall thickness were not different in infants with PDA and VSD; however, in older children the LV mass and wall thickness were significantly greater in PDA patients than in VSD patients.

Left ventricular volume mass ratio in infants with VSD and PDA with large shunts was greater than normal (P < 0.01). In older children with either VSD or PDA and a large shunt, this ratio was not different from normal (tables 1 and 2).

The left ventricular end-diastolic pressure (LVEDP) was not different from normal in all VSD and PDA patients with shunts less than 50%. In contrast, the LVEDP was

![Figure 4](http://circ.ahajournals.org/)

**Left atrial maximum volume (LA max) as a function of body surface area (BSA) in 41 VSD patients with moderate or large shunts. LA max = 55.3 (BSA) + 2.6 cc, 1 sd = 13.5 cc, r = 0.755, P < 0.001. Solid line indicates the regression line of normals and dashed lines indicate the 1 sd.**

![Figure 5](http://circ.ahajournals.org/)

Left ventricular wall mass normalized for body surface area (LVM/BSA) as a function of left ventricular systolic index for 39 patients with VSD. The LVM/m² = 6.63 (LV systolic index) + 79.4 g, 1 sd = 22.4 g, r = 0.652, P < 0.001.
significantly increased \((P < 0.001)\) in infants and children (tables 1 and 2) with large shunt patent ductus to greater than 1.5 times normal (tables 1 and 2).

The left ventricular end-diastolic stress in patients with a VSD and large shunt was not significantly different from normal (tables 1 and 2). In patients with a PDA and similar shunts, LV end-diastolic stress was significantly higher than that found in either normals \((P < 0.05)\) or VSD patients \((P < 0.02)\).

Left atrial maximal volume \((LA \text{ max})\) was increased in all VSD and PDA patients with shunts greater than 35% (fig. 5). This variable was not different in the infant age group from that in older children when normalized for BSA (tables 1 and 2). The LA maximal volume showed a significant increase \((P < 0.001)\) with increasing left-to-right shunt and increasing LV systolic index in both VSD and PDA patients.

**Discussion**

In the present study, the left ventricular end-diastolic volume and the left atrial maximal volume were increased in all patients with VSD and PDA and left-to-right shunts greater than 35%. This finding suggests a close relationship of LV end-diastolic volume and LA maximal volume with pulmonary blood flow in patients with either an aortic or ventricular shunt.

Levin and co-workers\(^8\) demonstrated the presence of left-to-right shunting across isolated ventricular septal defects in diastole, within the "isovolumic-contraction" period, and during the aortic ejection period. Mesel\(^9\) also found left-to-right shunting in these phases of the cardiac cycle by using electromagnetic flowmeter measurements in dogs with a created ventricular defect. It has been shown in patients that variable portions of the left-to-right shunt occur immediately after the onset of systole during the so-called isovolumic-contraction period.\(^1\) This shunting is accounted for in the stroke volume calculation in this study. Since diastolic shunting cannot be quantified by cineangiocardiography, we have assumed that the left ventricular systolic index provides an estimate of total pulmonary blood flow in the VSD patients although it does not account for the diastolic left-to-right shunt.

The left ventricular ejection fraction was abnormally low in infants with large shunts due to VSD or PDA. The average LV ejection fraction in these infants was 0.64, a value which is normal when compared to that of normal older children with hearts of similar size. Thus, this value is decreased from normal only in comparison with that of normal infants who have small normalized end-diastolic volumes and high ejection fractions.\(^1\)

The increase in left ventricular muscle mass with increasing magnitude of the left-to-right shunt and LV systolic index indicates that volume overload on the left ventricle stimulates muscular hypertrophy. In infants with VSD or PDA, the LV mass was almost twice normal. These findings emphasize the considerable myocardial hypertrophy which young infants can develop as a compensatory mechanism to a ventricular or aortic left-to-right shunt. Although LV mass was increased, the volume-to-mass ratio was above normal \((P < 0.001)\) in infants with either VSD or PDA; this observation indicates a greater volume than mass response to these lesions. The early onset of heart failure in these infants may be partly related to inability to increase left heart mass rapidly enough to compensate for the large left-to-right shunt present at this age.

In this present study the LV mass per BSA normalized for degree of shunt was significantly increased in older children with PDA and shunts of more than 35% over that of VSD patients (table 3). This increase could be related to differences in pressure-volume loop in the two groups.\(^10\) These differences are: (1) a decrease in the LV volume during the isovolumic-contraction period in patients with VSD and a stable volume during this interval in patients with PDA, and (2) a lower peak LV systolic pressure in patients with VSD than in patients with PDA with large left-to-right shunts. These factors result in greater stroke work in PDA patients than in VSD patients for equal shunts.
This study also emphasizes many similarities in left heart volume parameters in VSD and PDA patients with equivalent left-to-right shunts. The major differences between the two groups of patients were: (1) the higher LV end-diastolic pressure in PDA patients versus VSD patients, (2) elevated LV end-diastolic stress in PDA patients versus VSD patients with large shunts, and (3) the elevated LV mass/BSA and LV wall thickness/BSA normalized for shunt in children with VSD and shunt > 35%. Findings 1 and 2 mentioned above parallel the results of Spann and co-workers who observed a much greater increase in LVEDP in dogs with acutely produced equivalent aortic versus ventricular shunts. The elevated left ventricular end-diastolic pressure in patients with PDA over that of patients with VSD when shunts were equivalent suggests a decrease in LV distensibility in the group with PDA. One factor contributing to this altered distensibility may be the difference in resistance to LV ejection between the two groups. In patients with ventricular shunt, ejection begins during isovolumic contraction and thus the ventricle can begin unloading early in the cardiac cycle and continue to eject the shunted blood into the pulmonary circuit proximal to the aortic valve. In contrast, in PDA patients this unloading does not begin until the opening of the aortic valve with ejection of the total stroke volume into the aorta; by this means a slightly higher than normal systemic pressure is created. Thus the area underneath a tension-time curve is somewhat greater in patients with PDA than in patients with VSD who have similar shunts. Such a discrepancy in total developed-tension could be postulated to play a role in the altered LV distensibility and the increased normalized LV mass/shunt value in patients with an aortic versus ventricular shunt.

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References


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