Left Heart Volume and Mass Quantification in Children with Left Ventricular Pressure Overload

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SUMMARY
This investigation was designed to quantify left ventricular and left atrial volume, volume change, systolic output, and ventricular mass in 31 patients with isolated left ventricular pressure overload secondary to aortic stenosis (AS, n = 14) or coarctation of the aorta (n = 17). These parameters were compared with normal standards and with data from a group of nine patients with a combined pressure and volume overload due to aortic stenosis plus aortic or mitral insufficiency (AS + AI or MI). Volumes were calculated from biplane cineangiocardiograms exposed at 60 frames/sec. Left ventricular end-diastolic volume (LVEDV) was significantly lower than normal in patients with AS (57 ± 11 cc/m²), but was normal (73 ± 12 cc/m²) in patients with coarctation. An increase in the ejection fraction (LVEF) was found in both groups averaging 0.73 ± 0.12 in AS patients and 0.69 ± 0.09 in coarctation patients. Patients with AS + aortic or mitral insufficiency (AI or MI) showed elevated LVEDV (103 ± 29 cc/m²), but had a normal ejection fraction. The LV mass was significantly increased in all groups: normal, 82 ± 10 g/m²; AS, 126 ± 41 g/m²; coarctation, 130 ± 44 g/m²; and AS + AI or MI, 168 ± 42 g/m². The left ventricular systolic index and left atrial maximal volume were both normal in patients with pure pressure overload but were significantly increased in patients with combined pressure and volume overload. The low LVEDV in patients with AS as well as the normal volume in patients with coarctation occurred in the presence of elevated LV end-diastolic pressure and indicates a decrease in LV diastolic distensibility in patients responding to an isolated LV pressure overload by significant muscular hypertrophy without dilatation.

Additional Indexing Words:
Aortic stenosis Aortic insufficiency
Cineangiocardiograms Systolic output
Mitral insufficiency

Myocardial adaptation to a sustained pressure overload has been considered

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primarily in terms of an increase in muscle mass without a comparable increase in chamber volume. Such an increase in left ventricular wall thickness or muscle mass with left ventricular pressure overload has been documented in man,1-7 and efforts have been made to correlate the degree of hypertrophy with various hemodynamic parameters.1, 8

Recent investigations in the pediatric age group have delineated the normal standards for left heart volumes and mass9 and have shown considerable increase in these variables
Table 1

Vital Statistics and Cardiac Catheterization Data (Mean ± 1 SD)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Normal (n = 20)</th>
<th>Aortic stenosis (n = 14)</th>
<th>AS vs. normal</th>
<th>AS vs. coarctation</th>
<th>Coarctation (n = 17)</th>
<th>Coarctation vs. normal</th>
<th>AS + AI or MI (n = 9)</th>
<th>AS + AI or MI vs. normal</th>
<th>AS + AI or MI vs. AS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>8.0 ± 3.8</td>
<td>7.8 ± 4.4</td>
<td>NS</td>
<td>NS</td>
<td>6.5 ± 3.2</td>
<td>NS</td>
<td>10.0 ± 3.6</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Wt. (kg)</td>
<td>28.8 ± 15.0</td>
<td>25.1 ± 12.6</td>
<td>NS</td>
<td>NS</td>
<td>24.1 ± 12.6</td>
<td>NS</td>
<td>33.2 ± 15.4</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>BSA (m²)</td>
<td>1.00 ± 0.34</td>
<td>0.90 ± 0.34</td>
<td>NS</td>
<td>NS</td>
<td>0.89 ± 0.21</td>
<td>NS</td>
<td>1.12 ± 0.34</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Heart rate</td>
<td>90 ± 16</td>
<td>110 ± 20</td>
<td>NS</td>
<td>NS</td>
<td>103 ± 15</td>
<td>NS</td>
<td>106 ± 23</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Peak LVP (mm Hg)</td>
<td>99 ± 18</td>
<td>170 ± 39</td>
<td>P &lt; 0.001</td>
<td>P &lt; 0.01</td>
<td>135 ± 25</td>
<td>P &lt; 0.001</td>
<td>151 ± 30</td>
<td>P &lt; 0.001</td>
<td>NS</td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>10 ± 3</td>
<td>17 ± 5</td>
<td>P &lt; 0.001</td>
<td>NS</td>
<td>15 ± 6</td>
<td>P &lt; 0.02</td>
<td>19 ± 8</td>
<td>P &lt; 0.01</td>
<td>NS</td>
</tr>
<tr>
<td>Mean LVP during systole (mm Hg)</td>
<td>65 ± 10</td>
<td>97 ± 14</td>
<td>P &lt; 0.001</td>
<td>P &lt; 0.02</td>
<td>80 ± 17</td>
<td>P &lt; 0.05</td>
<td>87 ± 25</td>
<td>P &lt; 0.05</td>
<td>NS</td>
</tr>
</tbody>
</table>

Abbreviations: LVP = left ventricular pressure; LVEDP = left ventricular end-diastolic pressure; NS = not significant (P > 0.05); AS = aortic stenosis; AI = aortic insufficiency; MI = mitral insufficiency.

Methods

Patient Groups

Combined Pressure and Volume Overload: Aortic Stenosis Plus Aortic or Mitral Insufficiency

Nine additional patients were studied with combined pressure and volume overload. Five patients had aortic stenosis plus aortic insufficiency, three had aortic stenosis plus mitral insufficiency, and one had aortic stenosis plus mitral valve regurgitation. Patients with AI were included if their LV filling pressures were within normal limits.

Instrumental Pressure Overload: Aortic Stenosis and Coarctation

Four patients were excluded from this group because of prior fetal aortic stenosis. These patients had aortic stenosis but no evidence of subaortic obstruction. The peak LV pressure was calculated to be 159 ± 29 mm Hg.

Volume Overload: Aortic Stenosis

Nine patients were included in this group. Five patients had aortic stenosis plus aortic insufficiency, and four patients had aortic stenosis plus mitral insufficiency. The peak LV pressure was calculated to be 147 ± 30 mm Hg.

Volume Overload: Mitral Insufficiency

Three patients were included in this group. Two patients had mitral insufficiency plus mitral stenosis, and one had mitral insufficiency plus mitral stenosis. The peak LV pressure was calculated to be 150 ± 25 mm Hg.

Volume Overload: Aortic Insufficiency

Three patients were included in this group. Two patients had aortic insufficiency plus aortic stenosis, and one had aortic insufficiency plus mitral insufficiency. The peak LV pressure was calculated to be 140 ± 30 mm Hg.

Aortic Insufficiency

Three patients were included in this group. Two patients had aortic insufficiency plus aortic stenosis, and one had aortic insufficiency plus mitral insufficiency. The peak LV pressure was calculated to be 135 ± 25 mm Hg.

Aortic Stenosis

Four patients were included in this group. Three patients had aortic stenosis plus aortic insufficiency, and one had aortic stenosis plus mitral insufficiency. The peak LV pressure was calculated to be 120 ± 30 mm Hg.

Aortic Stenosis

Five patients were included in this group. Four patients had aortic stenosis plus aortic insufficiency, and one had aortic stenosis plus mitral insufficiency. The peak LV pressure was calculated to be 115 ± 25 mm Hg.

Aortic Stenosis

Six patients were included in this group. Five patients had aortic stenosis plus aortic insufficiency, and one had aortic stenosis plus mitral insufficiency. The peak LV pressure was calculated to be 110 ± 25 mm Hg.

Cardiac Catheterization

Ventricular volume, pressure, and conductance data were presented in Table 1. Patient flow included in Table 1 was not required for determination. The peak LV pressure was calculated to be 159 ± 29 mm Hg. The peak LV pressure was calculated to be 135 ± 25 mm Hg in the patients with AI who required aortic root replacement. The peak LV pressure was calculated to be 120 ± 30 mm Hg in the patients with AI who required aortic root replacement.
pressure in this group averaged 151 ± 30 mm Hg (table 1).

Patients in the three groups did not differ significantly in terms of age, weight, body surface area, or heart rate during cineangiography (table 1).

**Volume Data Acquisition**

All volume data were obtained during diagnostic cardiac catheterization with the patients supine and under light nitrous oxide and halothane (≤ 0.5%) anesthesia. Volumes were calculated from the first biplane cineangiogram of the catheterization procedure and were performed at least 50 minutes following the onset of the study. This initial cine was exposed at 60 frames/sec in the AP and lateral projections following the injection of 1.0 to 1.25 ml/kg of 75% Hypaque-M into the main pulmonary artery. The electrocardiogram, left ventricular pressure, and LV dp/dt were monitored and recorded simultaneously during cineangiography. Volumes were calculated during the initial two to three beats of the levogram phase before any peripheral effects of the contrast medium were apparent. Patients with ectopic beats were excluded. The AP and lateral tubes were pulsed asynchronously with a photocell device recording each exposure on a multichannel oscillograph at a paper speed of 200 mm/sec. All cine data were recorded on 16-mm film. A more detailed description of this system has been reported previously.9

After each study, a grid with 625 squares (1 cm by 1 cm) was placed parallel to the AP and then to the lateral tube at the position the heart occupied and was filmed. This film was then used to correct for x-ray magnification and to evaluate distortion. There was no measurable distortion of the grid in the inner % of the 16-mm projected image, and thus only a linear correction factor for magnification was used as long as the ventricular image occupied the inner % of the frame.

**Volume and Mass Calculation**

Left ventricular (LV) and left atrial (LA) volumes were calculated by the area-length method of Dodge and associates,11 and LV mass was calculated by the method of Rackley and coworkers.12 Left ventricular volumes were calculated at end diastole and end systole for two or three consecutive beats and then averaged. Left ventricular stroke volume (LVSV) was obtained as the difference between end-diastolic and end-systolic volumes, and LV systolic output was derived as LVSV times heart rate. Left ventricular mass was calculated only at end diastole. The mass and volume values were then corrected by previously derived regression equations from our laboratory.9 Left ventricular minute work was calculated as the product of the mean LV pressure during systole minus LV end-diastolic pressure and the LV systolic output; this variable was expressed in kg-m/min. Left ventricular circumferential or hoop stress was calculated at end diastole using the formula of Sandler and Dodge for an ellipsoid of revolution. Left atrial maximal volume (LAmax) was calculated in 34 patients whose atrial margins could be defined. The atrial appendage was excluded from the drawings and subsequent calculations. All volume and mass data were normalized for body surface area to facilitate comparison of patients of different age and size.

**Validation of Volume Calculations**

During the course of the investigation, the stability of left ventricular hemodynamics was studied both before and during contrast visualization of the left heart. Eighteen patients (seven with AS and 11 with coarctation) had left ventricular pressure (LVP) monitored with a high fidelity catheter tip transducer* both preceding and during the time when left heart volumes were determined. The first derivative of the LVP (LV dp/dt) measured with an electronic differentiator† and the electrocardiogram were monitored simultaneously with the LVP. The characteristics of the differentiator have been described previously.14 Left ventricular end-diastolic pressure, peak LVP, maximal LV dp/dt, and the R-R interval were averaged for five beats preceding the pulmonary artery injection of contrast medium and compared with the average values for these factors for the two or three beats during the levogram phase when volume calculations were performed. The results are shown in figure 1. There was no significant change during the levogram phase in the peak LV pressure, maximal LV dp/dt, or R-R interval. There was a small but significant increase in the LV end-diastolic pressure from an average of 15.8 to 17.4 mm Hg.

The stability and reproducibility of the volume determinations during the levogram also were assessed. The LV end-diastolic and end-systolic volumes calculated from the first beat of the levogram phase were compared with the volumes calculated from the second beat. There was no statistically significant difference in these determinations:

As a final evaluation of the cineangiographic measurement of left ventricular volume, the left ventricular output derived from the calculation of

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*Statham Products Inc. SF-1.
†Electronic Gear, Inc., Valley Stream, N. Y., Model No. 5602.
end-diastolic and end-systolic volumes was compared with Fick or indicator-dilution cardiac outputs performed immediately preceding the cine. This analysis was performed on 21 patients who had no left-to-right shunts or left heart valvular insufficiency. Seven patients in the present study were included in this evaluation. The results are shown in figure 2. There was an excellent correlation of the two methods with a correlation coefficient of 0.962. The regression equation relating the two variables had a P value of < 0.001 with a slightly higher cine output as compared with the Fick or dye output. Finally, the standard error of the estimating equation was only 0.278 cc/min.

Results

The results for individual patient groups are summarized in table 2. Normal values are included for comparison.

Pressure Overload

The left ventricular end-diastolic volume (LVEDV) was significantly decreased from normal by an average value of 20% in patients with isolated aortic stenosis. In contrast, patients with coarctation had normal LVEDV (fig. 3; table 2).

Pressure Overload

The left ventricular end-diastolic volume (LVEDV) was significantly decreased from normal by an average value of 20% in patients with isolated aortic stenosis. In contrast, patients with coarctation had normal LVEDV (fig. 3; table 2).

The left ventricular ejection fraction (LVEF) was significantly increased in both AS and coarctation patients and averaged 0.73 and 0.69, respectively, versus a normal value of 0.63 (fig. 4; table 2). In the coarctation group the LVEF showed a significant negative correlation with BSA with the ejection fraction decreasing with increasing size. There was no significant correlation of the LVEF with BSA in the AS patients.
LEFT VENTRICULAR PRESSURE OVERLOAD

Figure 2
Comparison of cinecardiographic measurement of left ventricular output with Fick or dye determinations of cardiac output in 21 patients without left-to-right shunts or left heart valvular insufficiency.

The left ventricular systolic index (LVS1) was not different from normal in either AS or coarctation patients, but the left ventricular minute work (LV min work) was significantly increased in both groups averaging 60% above normal in AS patients and 43% above normal in coarctation patients (table 2).

Both the left ventricular mass (LVM) and the normalized LV wall thickness were significantly increased in both AS and coarctation patients (fig. 5; table 2). Although the average peak LV pressure was higher in the AS group, there was no significant difference in mean normalized LV mass or wall thickness between the AS and coarctation groups.

Figure 3
Left ventricular end-diastolic volume as a function of body surface area. (A) Aortic stenosis. (B) Coarctation. Solid lines indicate the regression line for normals and the broken lines are ± standard error of the estimate (SEE).

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The mean left ventricular systolic pressure showed a significant correlation with LV mass in both the AS and coarctation patients (fig. 6). There was no significant correlation between LV mass and LV peak systolic pressure, peak gradient, mean gradient, or valve index in the patients with AS.

The left ventricular end-diastolic pressure (LVEDP) was significantly increased in both AS and coarctation patients (table 1). There was no significant correlation between LV

Figure 4
Left ventricular ejection fraction as a function of body surface area. (A) Aortic stenosis. (B) Coarctation. Solid lines indicate the mean, and the broken lines, ±1 standard deviation for normals. For the coarctation group there was a significant relationship between ejection fraction and BSA: LVEF = 0.28 (BSA) + 0.94; SEE = 0.07; P < 0.001; r = 0.672.

Figure 5
Left ventricular mass as a function of body surface area. (A) Aortic stenosis. (B) Coarctation. Solid lines indicate the regression lines for normals, and the broken lines are ± SEE.
end-diastolic pressure and end-diastolic volume. Figure 7 illustrates the mean values for end-diastolic pressure and volume for normals, AS, and coarctation patients.

The left ventricular end-diastolic stress (LVEDS) calculated as circumferential force per unit cross sectional area of LV wall was not different from normal in either AS or coarctation patients.

The left atrial maximal volume (LA_max) was not different from normal in either the AS or coarctation patients.

**Combined Pressure and Volume Overload**

In contrast to the pure pressure overload group, the left ventricular end-diastolic volume was significantly increased in patients with AS plus aortic or mitral insufficiency (table 2).

In addition the left ventricular ejection fraction was normal in this group as compared with an elevated ejection fraction in patients with isolated AS or coarctation.

The left ventricular systolic index was significantly increased in this group with the degree of increase in this variable reflecting the severity of the valvular regurgitation.

The left ventricular mass in patients with a pressure and volume overload was significantly increased above normal as well as significantly higher than that found in isolated AS (table 2). The LV wall thickness, however, was not different from normal in patients with AS + AI or MI.

The left ventricular end-diastolic stress in this patient group was not significantly different from normal.

The left atrial maximal volume was increased in patients with AS plus mitral insufficiency, but was within the normal range for patients with AS plus aortic insufficiency. Due to the high values of LA_max for the AS and MI patients, the mean value for the entire group was increased above normal.

**Discussion**

Previously it has been suggested that the left ventricular cavity in patients with a left-sided pressure overload may be normal or smaller than normal. The present investigation, in addition to quantitating the degree of left ventricular hypertrophy, also documents the normal or decreased size of the ventricular cavity as well as the increased ejection fraction in children with a chronic pressure overload. These data demonstrate that a hypertrophied ventricle in childhood can eject a greater than normal percentage of its end-diastolic volume despite large increases in afterload. An important next question is whether or not hypertrophy per se can increase the ejection fraction without an
### Table 2

**Volume Data (Mean ± 1 SD)**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Normal</th>
<th>Aortic stenosis</th>
<th>AS vs. normal</th>
<th>AS vs. coarctation</th>
<th>Coarctation</th>
<th>Coarctation vs. normal</th>
<th>Aortic stenosis + AI or MI</th>
<th>AS + AI or MI vs. normal</th>
<th>AS + AI or MI vs. AS</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV end-diastolic volume (cc/m²)</td>
<td>71 ± 8</td>
<td>57 ± 11</td>
<td><em>P &lt; 0.001</em></td>
<td><em>P &lt; 0.001</em></td>
<td>73 ± 12</td>
<td>NS</td>
<td>105 ± 27</td>
<td><em>P &lt; 0.001</em></td>
<td><em>P &lt; 0.001</em></td>
</tr>
<tr>
<td>LV ejection fraction</td>
<td>0.63 ± 0.05</td>
<td>0.73 ± 0.12</td>
<td><em>P &lt; 0.01</em></td>
<td>NS</td>
<td>0.69 ± 0.09</td>
<td><em>P &lt; 0.05</em></td>
<td>0.66 ± 0.13</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>LV systolic index (L/min/m²)</td>
<td>4.51 ± 0.88</td>
<td>4.58 ± 1.40</td>
<td>NS</td>
<td>NS</td>
<td>5.07 ± 1.04</td>
<td>NS</td>
<td>7.00 ± 2.24</td>
<td><em>P &lt; 0.001</em></td>
<td><em>P &lt; 0.01</em></td>
</tr>
<tr>
<td>LV minute work (kg-min/m²)</td>
<td>5.36 ± 1.57</td>
<td>8.60 ± 3.80</td>
<td><em>P &lt; 0.01</em></td>
<td>NS</td>
<td>7.68 ± 3.65</td>
<td><em>P &lt; 0.05</em></td>
<td>9.72 ± 3.33</td>
<td><em>P &lt; 0.001</em></td>
<td>NS</td>
</tr>
<tr>
<td>LV mass (g/m²)</td>
<td>82 ± 10</td>
<td>126 ± 41</td>
<td><em>P &lt; 0.001</em></td>
<td>NS</td>
<td>130 ± 44</td>
<td><em>P &lt; 0.001</em></td>
<td>168 ± 42</td>
<td><em>P &lt; 0.001</em></td>
<td><em>P &lt; 0.05</em></td>
</tr>
<tr>
<td>LV wall thickness (cm/m²)</td>
<td>0.71 ± 0.29</td>
<td>0.92 ± 0.21</td>
<td><em>P &lt; 0.02</em></td>
<td>NS</td>
<td>0.92 ± 0.35</td>
<td><em>P &lt; 0.02</em></td>
<td>0.71 ± 0.26</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>LV end-diastolic stress (g/cm²)</td>
<td>38 ± 12</td>
<td>36 ± 11</td>
<td>NS</td>
<td>NS</td>
<td>36 ± 12</td>
<td>NS</td>
<td>43 ± 19</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>LA max (cc)</td>
<td>34 ± 10</td>
<td>39 ± 10</td>
<td>NS</td>
<td>NS</td>
<td>42 ± 9</td>
<td>NS</td>
<td>71 ± 25</td>
<td><em>P &lt; 0.001</em></td>
<td><em>P &lt; 0.001</em></td>
</tr>
</tbody>
</table>

Abbreviations: AS = aortic stenosis; Coarct. = coarctation; AI = aortic insufficiency; MI = mitral insufficiency; LA max = left atrial maximal volume; NS = not significant (*P > 0.05*).
increase in the basic inotropic state of the heart. Thus, it may be possible in pressure overload lesions that systemic output can be maintained because of an increase in muscle mass in the presence of normal or even decreased contractility of individual muscle fibers.

The finding of normal or decreased LV end-diastolic volumes in the presence of elevated LV end-diastolic pressures in patients with sustained pressure overload indicates a decrease in diastolic distensibility. Stewart and co-workers showed a decreased rate of the Y descent in the left atrial pressure curve in patients with left ventricular obstructive lesions. This finding suggests a decreased rate of LV filling which could be due to a decrease in LV distensibility. A decreased rate of LV filling in the presence of a normal heart rate could account for the small LV end-diastolic volumes in the patients with aortic stenosis in this study.

Those patients in the present study with either aortic or mitral regurgitation showed an increase in left ventricular end-diastolic volume and systolic index with the degree of increase in these two variables reflecting the severity of the regurgitant lesions. The enlarged left atrial maximal volume in patients with mitral insufficiency is in agreement with previous investigations which have shown an excellent correlation between LA$_{max}$ and the degree of mitral regurgitation. The normal LA$_{max}$ in patients with isolated pressure overload and elevated LV end-diastolic pressure suggests a decrease in atrial distensibility which might be related to atrial hypertrophy.

The methodology employed in quantitative cineangiography deserves some emphasis. Theoretical objections to such quantification center around the model employed to represent the heart and the changes in hemodynamics following injection of contrast medium. The ellipsoid of revolution reference figure and the area-length method for left ventricular volume calculations have been validated with postmortem studies by several investigators. This method has not been tested, however, in hearts with severe myocardial hypertrophy in which papillary muscle enlargement may distort the normal left ventricular cavity particularly at end systole. In this regard seven patients in the present investigation had a comparison of Fick or indicator-dilution cardiac outputs with cine outputs (fig. 2). The close agreement between the two methods suggests that in these patients the cine method was providing accurate quantification of systemic flow. Since end-diastolic cavities are not markedly distorted in these patients, it is unlikely that there were significant errors in estimation of this variable. It then follows that end-systolic volume must have been represented accurately by the model used in order for the stroke volume and thus systemic flow to parallel closely the flows estimated by an independent method.

The cardiovascular effects of contrast medium are well known, and changes in hemodynamics invariably occur approximately 20 sec following injection. To ameliorate any changes in resting hemodynamics due to the volume of contrast medium used, we have used pulmonary artery injections with left heart volume calculations performed during the levogram phase. The present study demonstrated the stability of left ventricular hemodynamics during the levogram phase following pulmonary artery cineangiography (fig. 1). Additional evidence for hemodynamic stability during this period was provided by the close agreement between cine and Fick or dye cardiac outputs. The small increase in end-diastolic pressure during cineangiography as well as the slight increase in the calculated left ventricular output as compared with the Fick or dye output is compatible with a small increase in end-diastolic volume and output due to the volume of contrast medium injected and is in agreement with previous experimental findings. Significant changes in peak ventricular pressure and heart rate frequently were observed 3 to 5 sec following the levogram phase in the patients with isolated aortic stenosis or coarctation.

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The data reported herein have potential clinical applications for children beyond infancy with sustained left ventricular pressure overload and no symptoms of cardiac decompensation. In such patients, significant regurgitation at either the mitral or aortic valve should be given strong consideration when enlargement of the left ventricle is evident. Furthermore, enlargement of the left atrium in this setting should suggest significant mitral regurgitation. Of perhaps greater clinical importance, the degree of myocardial hypertrophy as estimated from left ventricular mass calculations can be a useful adjunct in assessing the severity of a chronic pressure overload lesion. In addition, preoperative and postoperative determinations of volume and mass can be utilized effectively in the more critical evaluation of the myocardial response to surgical therapy.

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25 Years Ago

An operation for increasing the flow of blood through the lungs and thereby reducing the cyanosis in patients with congenital malformations of the heart consists in making an anastomosis between a branch of the aorta and one of the pulmonary arteries, in other words, the creation of an artificial ductus arteriosus. Thus far the procedure has been carried out on only three children, each of whom had a severe degree of anoxemia. Clinical evidence of improvement has been striking. . .—BLALOCK A, TAUSSIG H: The surgical treatment of malformations of the heart in which there is pulmonary stenosis or pulmonary atresia. JAMA 128:189, 1945.
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