Alterations in Left Ventricular Geometry, Wall Stress, and Ejection Performance After Correction of Congenital Aortic Stenosis

Gerald W. Dorn II, MD, Richard Donner, MD, Michael E. Assey, MD, James F. Spann Jr., MD, Henry B. Wiles, MD, and Blase A. Carabello, MD

Children with congenital aortic stenosis have "excessive" left ventricular hypertrophy with reduced resting systolic wall stress that allows for supernormal ejection performance. If aortic stenosis is uncorrected, this pattern persists until adulthood. The effect of removing the aortic pressure gradient on left ventricular hypertrophy and wall stress in children with congenital aortic stenosis is unknown. To test the hypothesis that removal of the stimulus for hypertrophy by aortic valve replacement or repair would normalize left ventricular mass and wall stress, we measured left ventricular ejection performance, wall stress, and contractile function in seven patients at cardiac catheterization before and 36±7 months after surgical correction of congenital aortic stenosis. After aortic valve replacement or repair, the aortic valve gradient fell from 87±12 to 7±4 mm Hg, and peak left ventricular pressure fell from 187±14 to 128±8 mm Hg. Left ventricular ejection fraction decreased postoperatively from 86±4% to 74±4% (p<0.001), whereas velocity of circumferential fiber shortening decreased from 2.15±0.15 to 1.6±0.11 (p<0.002). Left ventricular mass remained unchanged preoperatively (121±14 g/m²) and postoperatively (121±16 g/m²), but wall thickness (h) decreased in relation to ventricular radius (r) (h/r=0.55±0.05 preoperatively, 0.36±0.02 postoperatively; p<0.001). Left ventricular end-systolic wall stress increased after surgery from 45±12 to 96±13, whereas mean stress increased from 174±18 to 249±26 dynes×10⁹/cm²². Contractile function assessed by the velocity of circumferential fiber shortening--end-systolic stress relation, did not change after aortic valve replacement or repair. We conclude that after aortic valve replacement or repair in children with congenital aortic stenosis, wall stress increases and ejection performance decreases toward normal. (Circulation 1988;78:1358–1364)

C hronic pressure overload of aortic stenosis produces concentric left ventricular hypertrophy in both children and adults. In adults with acquired aortic stenosis, adequate hypertrophy can normalize afterload as measured by systolic wall stress. However, if hypertrophy is inadequate, systolic wall stress (afterload) increases, and ventricular hypertrophy is inadequate, the resultant increased wall stress often causes reduced ejection performance, which can improve after aortic valve replacement. In contrast to acquired aortic stenosis, recent studies of children with congenital aortic stenosis demonstrated reduced systolic wall stress, permitting increased ejection performance. Reduced resting wall stress was due to greater wall thickness than was necessary to normalize wall stress. Whether the observed increase in wall thickness in children represents "excessive" hypertrophy, a reserve mechanism for compensating increasing left ventricular pressure during exercise, or myocardial hyperplasia is unclear. Whatever the cause, the congenital aortic stenosis pattern of hypertrophy producing low wall stress and increased ejection performance persists into adulthood if the ventricular outflow obstruction is not relieved surgically. However, it is unknown what effect removal of the pressure overload has upon the hypertrophic process and ventricular mechanics of congenital aortic stenosis. We hypothesized that removal of the stimulus...
for hypertrophy by surgical correction of the aortic stenosis would result in the return of left ventricular wall stress and ejection indexes toward normal. To test this hypothesis, we assessed left ventricular ejection performance, wall stress, and contractile function at cardiac catheterization in a retrospective study of children with congenital aortic stenosis before and after aortic valve replacement or repair.

**Subjects and Methods**

**Patients**

Seven children with pure congenital aortic stenosis underwent cardiac catheterization before and more than 6 months after aortic valve replacement (three patients) or repair (four patients). Patients were studied if they met the following criteria for both preoperative and postoperative catheterizations: 1) there was no significant aortic insufficiency as determined by aortic root angiography or Doppler echocardiography; 2) left ventricular dimensions and wall thickness could be accurately determined; 3) there were two consecutive sinus beats during ventriculography; 4) properly damped left ventricular and aortic pressure tracings were available; 5) the patient was not receiving medications known to affect cardiac function at the time of study; and 6) the peak preoperative transvalvular gradient was 50 mm Hg or greater and had to be reduced by at least 80% postoperatively. Gradient was used instead of aortic valve area as the criterion for assessing the severity of aortic stenosis because no specific valve area is agreed upon as the “critical area” in childhood aortic stenosis. Six patients underwent cardiac catheterization and surgery at the Medical University of South Carolina, Charleston, South Carolina, and one patient was studied at St. Christopher’s Hospital for Children, Philadelphia, Pennsylvania. Two patients were recruited from patients previously studied by the authors.12

**Catheterization Procedure and Measurement of Ventricular Performance**

Catheterization was performed through the femoral vessels after premedication with meperidine and promethazine. All patients underwent aortic root angiography during both preoperative and postoperative catheterizations, and in no patient was there more than trace aortic insufficiency. The left ventricle was entered by the retrograde approach. Pressures were measured with properly damped fluid-filled 6F or 7F 100-cm catheters. The peak-to-peak transvalvular gradient was measured by superimposing the left ventricular pressure tracing upon the aortic pressure tracing obtained at the time of left heart pullback. Cineangiography was performed in the right anterior oblique (five patients) and anteroposterior projection (two patients) in both the preoperative and postoperative catheterizations at 60 frames/sec. Left ventricular volumes were calculated by the area-length method,15 and volumes were then indexed to body surface area. The end-diastolic volume was taken as the largest volume, and the end-systolic volume was taken at the time of aortic valve closure, which corresponded in each instance with the smallest volume measured.

A frame-by-frame analysis of ventricular volume and wall stress was performed for the complete cardiac cycle. Premature and postpremature beats were excluded from analysis. Wall thickness was measured at end diastole and was calculated for ensuing frames by the method of Hugenholtz et al.16 This method is based on the assumption that cardiac mass remains constant throughout the cardiac cycle, and it allows the calculation of wall thickening from the measured amount of ventricular dimensional shortening or lengthening. In our laboratory, we have shown excellent correlation between angiographic and echocardiographic measurements of left ventricular wall thickness in this patient population.12 In this study, five patients had echocardiograms that could be correlated with catheterization data. The correlation coefficient was 0.86 (p<0.03), and in no patient did the angiographic estimate of wall thickness differ from the echocardiographic estimate by more than 1 mm. Left ventricular mass was estimated by the method of Kennedy et al.17 Circumferential wall stress was calculated with the formula of Mirsky18 and expressed as dynes×10⁻⁴/cm²:

\[
\text{Stress} = \frac{P \cdot b}{h} \left(1 - \frac{b}{2h} \frac{b^2}{2a^2}\right) \cdot 1,332 \text{ dynes/cm}^2
\]

where b is semiminor axis (D+2h)/2; a is semimajor axis (L+2h)/2; h is wall thickness; and P is pressure. The pressure matched for a given frame was derived from a digitized left ventricular pressure tracing with a similar RR interval as the angiographic beat. Pressure tracings and cineangiographic frames were aligned by matching the cineangiographic frame in which aortic valve opening occurred (visualized in every instance) with the aortic pressure upstroke and superimposing that point on the left ventricular pressure tracing. Sampling from the pressure tracing was made at 0.0167-second intervals to correspond with the angiographic framing rate of 60 frames/sec. End-systolic stress was measured at aortic valve closure and mean systolic stress was calculated by determining the area under the curve by the trapezoid rule19 and dividing by the time interval.

**Estimation of Contractile Function**

Contractile function before and after surgery was estimated with the velocity of circumferential fiber shortening (Vcf) and end-systolic stress relation.20–22 Vcf was calculated as (EDD−ESD)/EDD×et, where EDD is left ventricular end-diastolic dimension; ESD is end-systolic dimension; and et is ejection time measured from the upstroke of aortic pressure
tracing to the dicrotic notch. This relation corrects \( V_{	ext{cf}} \) (a relatively preload-independent measure of ejection performance) for afterload, yielding a useful measure of contractile function. The \( V_{	ext{cf}} \)-stress relation was examined in 10 normal subjects with whom the patients with aortic stenosis were compared.

**Normal Subjects**

Ten patients (mean age, 12.2±1 years) who underwent cardiac catheterization before electrophysiological testing for cardiac arrhythmias composed the group of normal subjects. These patients were subsequently found to be free of any cardiac structural or physiological abnormalities other than supraventricular arrhythmia. None was taking drugs known to affect cardiac function. All had left ventriculograms and pressure tracings that met the same criteria as for the patients with aortic stenosis. The normal \( V_{	ext{cf}} \)-stress relation and prediction bands were derived from these 10 subjects. Contractile function of the patients with aortic stenosis was then compared with this normal relation.

**Surgery**

Surgery at the Medical University of South Carolina was performed by the same surgeon. In no instance was there any intraoperative complication or any postoperative evidence by electrocardiogram or cardiac enzymes that myocardial damage occurred. All patients received cold hyperkalemic cardioplegia intraoperatively. Three patients underwent valve replacement with a St. Jude’s prosthesis. Four patients underwent aortic valve repair.

**Statistical Analysis**

All values are reported as mean±SEM. Differences between preoperative and postoperative values were compared with paired Student’s \( t \) test. The \( V_{	ext{cf}} \)-stress relation was plotted with an exponential regression. The 95% prediction bands were then derived for this relation.

**Results**

The age of our patients was 11.1±2.1 years at the time of preoperative catheterization and 14.3±1.9 years at postoperative catheterization. The postoperative catheterizations were performed 36±7 months after aortic valve replacement or repair. Heart rate during hemodynamic measurements was 110±11 preoperatively and 88±12 beats/min postoperatively (\( p=\text{NS} \)). Pressure, volume, and calculated hemodynamic data are shown in Table 1. The preoperative peak-to-peak aortic valve gradient was 87±12 and decreased to 7±4 mm Hg, and mean aortic valve gradient was 69±11 and decreased to 4±2 mm Hg after aortic valve replacement or repair (\( p<0.001 \)). Peak left ventricular systolic pressure decreased postoperatively from 187±14 to 128±8 mm Hg (\( p=0.011 \)). Left ventricular end-diastolic pressure was 12±2 preoperatively compared with 14±2 mm Hg (\( p=\text{NS} \)) after aortic valve replacement or repair. Figure 1 shows that the ejection fraction decreased significantly after aortic valve replacement or repair (86±4% preoperatively, 74±4% postoperatively; \( p<0.001 \)). \( V_{	ext{cf}} \) also decreased postoperatively from 2.15±0.15 to

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**Table 1.** Pressure, Volume, and Derived Data for Seven Patients With Congenital Aortic Stenosis Before and After Aortic Valve Replacement or Repair

<table>
<thead>
<tr>
<th>Patient</th>
<th>Time</th>
<th>Age (yr)</th>
<th>LVSP (mm Hg)</th>
<th>LVEDP (mm Hg)</th>
<th>EDV1 (cc/ml)</th>
<th>ESV1 (cc/ml)</th>
<th>EF</th>
<th>( V_{	ext{cf}} )</th>
<th>LVM1 (g/m²)</th>
<th>AVG (mm Hg)</th>
<th>h/r</th>
<th>ESS (kdynes/cm²)</th>
<th>EDS</th>
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<tbody>
<tr>
<td>1</td>
<td>Preop</td>
<td>116</td>
<td>220</td>
<td>12</td>
<td>95</td>
<td>19</td>
<td>80</td>
<td>2.13</td>
<td>96</td>
<td>130</td>
<td>0.54</td>
<td>79</td>
<td>27</td>
</tr>
<tr>
<td></td>
<td>Postop</td>
<td>113</td>
<td>6</td>
<td>57</td>
<td>20</td>
<td>66</td>
<td>1.73</td>
<td>78</td>
<td>28</td>
<td>0.38</td>
<td>94</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Preop</td>
<td>17</td>
<td>150</td>
<td>11</td>
<td>78</td>
<td>21</td>
<td>73</td>
<td>1.61</td>
<td>118</td>
<td>50</td>
<td>0.46</td>
<td>76</td>
<td>30</td>
</tr>
<tr>
<td></td>
<td>Postop</td>
<td>21</td>
<td>120</td>
<td>17</td>
<td>86</td>
<td>34</td>
<td>61</td>
<td>1.21</td>
<td>122</td>
<td>8</td>
<td>0.38</td>
<td>126</td>
<td>56</td>
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<tr>
<td>3</td>
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<td>8</td>
<td>158</td>
<td>8</td>
<td>94</td>
<td>23</td>
<td>76</td>
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<td>91</td>
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<td>232</td>
<td>10</td>
<td>90</td>
<td>4</td>
<td>94</td>
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<td>0.52</td>
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<tr>
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<td>168</td>
<td>19</td>
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<td>32</td>
<td>79</td>
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<td>1</td>
<td>98</td>
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<td>68</td>
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<td>0.55</td>
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<td>24</td>
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<tr>
<td></td>
<td>Postop</td>
<td>13</td>
<td>120</td>
<td>18</td>
<td>122</td>
<td>23</td>
<td>81</td>
<td>1.63</td>
<td>155</td>
<td>0</td>
<td>0.32</td>
<td>75</td>
<td>53</td>
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<td>7</td>
<td>Preop</td>
<td>4</td>
<td>180</td>
<td>12</td>
<td>42</td>
<td>6</td>
<td>87</td>
<td>2.21</td>
<td>182</td>
<td>110</td>
<td>0.80</td>
<td>40</td>
<td>19</td>
</tr>
<tr>
<td></td>
<td>Postop</td>
<td>7</td>
<td>125</td>
<td>8</td>
<td>58</td>
<td>13</td>
<td>78</td>
<td>1.70</td>
<td>114</td>
<td>12</td>
<td>0.46</td>
<td>71</td>
<td>21</td>
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</tbody>
</table>

LVSP, left ventricular systolic pressure; LVEDP, left ventricular end-diastolic pressure; EDV1, end-diastolic volume index; ESV1, end-systolic volume index; EF, left ventricular ejection fraction; \( V_{	ext{cf}} \), velocity of circumferential fiber shortening; LVM1, left ventricular mass index; AVG, peak-to-peak aortic transvalvular gradient; h/r, ratio of end-diastolic left ventricular thickness to radius; ESS, end-systolic stress (kdynes/cm²); EDS, end-diastolic wall stress; preop, postop, before and after aortic valve replacement or repair, respectively.
1.6±0.11 (p=0.002). The end-systolic volume index doubled after aortic valve replacement or repair (11±4 preoperatively, 23±4 cc/m² postoperatively; p<0.02), and end-diastolic volume index increased 21% postoperatively (76±8 preoperatively, 92±13 cc/m² postoperatively), but this change did not achieve statistical significance (Figure 2). Left ventricular mass index was 121±14 g/m² preoperatively and was unchanged postoperatively (121±16 g/m²). Left ventricular mass was also indexed to the subject’s weight and again did not significantly change after aortic valve replacement (5.1±1.1 preoperatively, 4.2±0.4 g/kg postoperatively). The ratio of end-diastolic wall thickness to ventricular radius (h/r) decreased after aortic valve replacement or repair (0.55±0.05 preoperatively, 0.36±0.02 postoperatively; p=0.001). Left ventricular end-systolic stress and mean-systolic stress increased after aortic valve replacement or repair (45±12, 174±18 preoperatively; 96±13, 249±26 dynes×10⁵/cm² postoperatively; p<0.01 each) (Figure 3). Overall, systolic circumferential wall stress and systolic volumes increased throughout the cardiac cycle as illustrated by the stress-volume loops (Figure 4). Left ventricular end-diastolic wall stress was 27±4 preoperatively and 39±7 dynes×10⁵/cm² postoperatively (Table 1), but this difference was not statistically significant (p=0.1).

Contractile function was assessed with the V_{cf}-stress relation.²⁰⁻²² Our patients are plotted preoperatively and postoperatively against the 95% prediction bands for normal subjects (Figure 5). Figure 5 demonstrates a parallel shift within the confidence bands, indicating a fall in V_{cf} postoperatively commensurate with the increase in afterload (wall stress). The V_{cf}-stress relation did not suggest a postoperative fall in contractile function.

**Discussion**

A major finding of this study of children with congenital aortic stenosis was that left ventricular wall stress increased (returned toward normal) after removal of the obstruction to left ventricular outflow. A second important finding was that ejection performance decreased from supernormal toward normal. Ejection performance is determined by preload, contractile function, and afterload. Because end-diastolic pressure increased significantly postoperatively and end-diastolic volume index tended to increase, reduced preload probably does not contribute to the decline in postoperative ejection performance that we noted. Likewise, the fall in ejection performance after aortic valve replacement or repair probably did not result from a reduction in contractility. All patients received cold hypothermic cardioplegia during surgery, and in none was there evidence of intraoperative myocardial damage. More important, patients remained within or above the V_{cf}-stress relation for normal subjects. Rather, it is likely that ejection performance fell as a direct result of the increase in systolic wall stress (afterload) that we observed. Indeed, the V_{cf}-stress...
relation suggested that the postoperative fall in $V_{cf}$ was commensurate with the observed rise in wall stress. Further, the fall in ejection fraction was due to a numerical increase in end-systolic volume index, the expected result from an increase in end-systolic stress. Our preoperative findings confirm those of other studies that children with congenital aortic stenosis have decreased resting wall stress and enhanced ejection performance.\textsuperscript{3,4,12} After relief of the pressure overload, wall stress and ejection performance returned toward normal values (Table 2).

It is initially surprising that ventricular stress increased despite removal of on average an 80 mm Hg aortic valve gradient. Examination of the components used to calculate wall stress demonstrates how wall stress increased postoperatively despite a fall in left ventricular pressure. Wall stress, approximated by LaPlace's law, is dependent not only on generated pressure but also on ventricular dimension and wall thickness: $\text{Stress} = \frac{p \cdot r}{2h}$ where $p$ is pressure; $r$ is radius; $h$ is wall thickness. In the present study, peak left ventricular pressure fell by approximately 33%, which by itself would have

![Figure 3](image)

**Figure 3.** Plot of left ventricular wall stress at end systole (ESS) and mean systolic wall stress (MSS) compared before (Preop) and after (Postop) aortic valve replacement or repair.

![Figure 4](image)

**Figure 4.** Plot of frame-by-frame values before (preop) and after (postop) aortic valve replacement or repair for wall stress plotted against the left ventricular volume index. Each point represents the mean ±SEM for seven subjects. Note that wall stress and ventricular volume increase in the postoperation group.

![Figure 5](image)

**Figure 5.** Plot of velocity of circumferential fiber shortening ($V_{cf}$) before (●) and after (→) aortic valve replacement or repair versus end-systolic stress for the seven subjects and compared with the $V_{cf}$-stress relation from 10 normal subjects. Each of the subjects demonstrates a parallel shift within the 95% confidence bands for the normal subjects, suggesting no change in postoperative contractile state.
TABLE 2. Comparison of Hemodynamic Data for Children With Congenital Aortic Stenosis, Congenital Aortic Stenosis After Aortic Valve Replacement or Repair, and Normal Children

<table>
<thead>
<tr>
<th>Study</th>
<th>Age (yr)</th>
<th>EF</th>
<th>LVMl (g/m²)</th>
<th>ESS (kdynes/cm²)</th>
<th>h/r</th>
</tr>
</thead>
<tbody>
<tr>
<td>Current study</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Preop congenital AS</td>
<td>11.1±2.1</td>
<td>0.86±0.04</td>
<td>121±14</td>
<td>45±12</td>
<td>0.55±0.05</td>
</tr>
<tr>
<td>Postop congenital AS</td>
<td>14.3±1.9</td>
<td>0.74±0.04</td>
<td>121±16</td>
<td>96±13</td>
<td>0.36±0.02</td>
</tr>
<tr>
<td>Donner et al³</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Congenital AS (n=11)</td>
<td>10±3.6</td>
<td>0.88±0.08</td>
<td>180±58</td>
<td>24±12</td>
<td>...</td>
</tr>
<tr>
<td>Normal (n=10)</td>
<td>8.5±2.9</td>
<td>0.64±0.08</td>
<td>96±9</td>
<td>109±30</td>
<td>...</td>
</tr>
<tr>
<td>Assey et al¹²</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Congenital AS (n=7)</td>
<td>8.4±1.1</td>
<td>0.80±0.03</td>
<td>125±11</td>
<td>63.3±6.7</td>
<td>0.52±0.05</td>
</tr>
<tr>
<td>Normal (n=7)</td>
<td>8.4±1.1</td>
<td>0.69±0.03</td>
<td>68±6</td>
<td>105±11</td>
<td>0.30±0.01</td>
</tr>
</tbody>
</table>

Values are mean±SEM.
EF, left ventricular ejection fraction; LVMl, left ventricular mass index; ESS, end-systolic stress; h/r, ratio of end-diastolic left ventricular thickness to radius; preop, postop, before and after aortic valve replacement or repair; AS, aortic stenosis.

decreased stress. However, the ratio of ventricular radius to ventricular thickness (r/2h) increased by approximately 70%. Thus, wall stress increased despite the decrease in left ventricular pressure because of the changes that occurred in left ventricular geometry. These changes do not represent simple regression of left ventricular hypertrophy because left ventricular mass index did not change after aortic valve replacement. Rather, the increase in wall stress probably occurred as the left ventricular chamber was remodeled after removal of the pressure overload. The occurrence of remodeling is suggested by a decrease in the ratio of wall thickness to radius (h/r), suggesting a relative decrease in concentric hypertrophy and an increase in eccentric hypertrophy.

Two major questions remain unanswered. Why is congenital aortic stenosis attended by "excessive" concentric hypertrophy? What is the cause of the postoperative remodeling that we observed? Answers to these questions can be speculative only. The "excessive" hypertrophy seen preoperatively may not be excessive at all but rather represents a response to increasing ventricular pressure during exercise. It is known that a stimulus to hypertrophy of short duration may incite cardiac hypertrophy despite longer periods without the stimulus. 23 Thus, periods of exercise may induce enough hypertrophy to normalize wall stress during exercise when left ventricular pressure is predictably very high. If stress is normalized during exercise when the intraventricular pressure is higher than at rest, the amount of concentric hypertrophy present would produce subnormal stress at rest when left ventricular pressure is less than during exercise. Alternatively, cardiac hyperplasia instead of, or together with, hypertrophy may have occurred in our patients. Although hypertrophy is the usual response to pressure or volume overload, hyperplasia can occur when the overload is present from birth. 13,14 If hyperplasia did develop, it may explain why cardiac mass did not decrease postoperatively because hyperplasia unlike hypertrophy would not be expected to regress after removal of the pressure overload.

The cause of the ventricular remodeling after relief of the pressure overload is also only speculative. Immediately after surgery when the pressure overload was removed and before remodeling could have occurred, wall stress at rest must have been extremely low. This extreme systolic unloading likely would lead to regression of concentric hypertrophy eventually increasing systolic stress toward normal. The increased systolic wall stress, in turn, would inhibit ventricular emptying, thus increasing the volume retained in the ventricle at end systole. Increased end-systolic volume could lead to decreased stroke volume and increased diastolic filling pressure. The increase in diastolic filling pressure would increase diastolic stress, which according to Grossman's hypothesis would lead to eccentric hypertrophy, enlargement of the ventricle, and return of stroke volume to normal (Figure 6). 24

Limitations
We recognize that seven patients is a small study group. However, even if a larger series demonstrated that the phenomena we observed was not universal, the phenomena even in a small group would still be, in our opinion, of scientific interest requiring an explanation.
A second limitation is that our pressure recordings were made just before but not simultaneous with the left ventriculograms. Although this may have injected some error into our study, there was no suggestion that a physiological change occurred.
in any patient in the minute or two between pressure recording and ventriculography.

Finally, an average of 36 months elapsed between the preoperative and postoperative studies. One could argue that as the patients grew during this period, an increase in heart size (eccentric hypertrophy) would be expected. We emphasize that all volumes and masses in the study are indexed to body surface area to correct for the influence of growth between studies.

Conclusion

We conclude that after correction of the pressure overload in congenital aortic stenosis, systolic wall stress increases, returning toward normal. Increased wall stress, in turn, reduces ejection performance from supranormal preoperative levels toward normal postoperative levels. The fall in ejection fraction primarily occurs due to an increase in end-systolic volume index as expected from an increase in afterload. These observed changes in stress, ejection performance, and volume possibly occur secondary to ventricular remodeling without actual regression of hypertrophy or hyperplasia or both.

References


KEY WORDS • congenital aortic stenosis • left ventricular hypertrophy • aortic valve replacement • ventricular wall stress
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