Hemodynamics and Annuloplasty in Isolated Mitral Regurgitation in Children

By Rabi Sulayman, M.D., Rajamma Mathew, M.R.C.P. (Edin.), Otto G. Thilenius, M.D., Ph.D., Robert Replogle, M.D., and Rene A. Arcilla, M.D.

SUMMARY
Isolated mitral insufficiency in children is quantitated angiographically by comparing the stroke volumes of the right ventricle and left ventricle. The disease results in greater enlargement of the left atrium than of the left ventricle and is accompanied by a significant increase in left atrial “distensibility.” Right and left heart pressures may be normal or may be increased; they tend to be elevated in the group with regurgitant fractions of over 50%. Annuloplasty results in marked clinical and hemodynamic improvement and may even be corrective.

DESPITE THE SIGNIFICANT DECLINE in incidence during the past two decades, rheumatic heart disease remains a therapeutic problem. This is particularly true in the pediatric age where severe mitral or aortic insufficiency may be present with minimal or no symptoms. The decision to replace a valve to prevent reduction of left ventricular function from the valvar disease has to be balanced against the acknowledged undesirable consequences of valve replacement by a prosthetic device, particularly the need for long-term anticoagulation and what it entails for the active child.

Mitral annuloplasty has been successfully done for the correction of mitral regurgitation,1-4 and its postoperative complications have been shown to be lower than those following prosthetic valve replacement.5,6 Some investigators have advocated it as the treatment of choice for the severe mitral regurgitation encountered in children.1,2

This paper presents the hemodynamic data observed in 25 children with isolated mitral regurgitation, and in six of 11 patients successfully treated by annuloplasty.

Materials and Methods
Twenty-five children, age 2½ to 18 years, with isolated mitral regurgitation underwent hemodynamic investigations. Shunting defects were ruled out by hydrogen inhalation studies, blood gas analyses, and by electrocardiography. Associated mitral stenosis was not present clinically, and was ruled out by simultaneously recorded left ventricular and pulmonary capillary wedge pressure tracings. None had evidence for aortic or tricuspid insufficiency. Eleven underwent mitral annuloplasty; of these, six had repeat cardiac catheterization one to five years after surgery. Positive history for rheumatic fever was obtained in 17 children; in the remaining eight, a rheumatic etiology was assumed and was, with one exception, consistent with the operative findings. In one case, the mitral regurgitation was due to ruptured anterior chordae tendineae.

Surgery was done under cardiopulmonary bypass using a mid- sternotomy approach. The heart was cooled topically with chilled Ringer’s lactate solution, and the ascending aorta crossclamped to provide ischemic coronary arrest. The left atrium was opened on the right, close to the intra-atrial groove; more recently, the “superior” approach has been used since it provides a more symmetric view of the entire mitral apparatus. In one case where ruptured anterior chordae tendineae was noted and the valve leaflets appeared normal, the area of the leaflet served by the ruptured chordae tendineae was plicated using the technique of Gerbode.10

Using 2-0 suture tied over Teflon felt pledgets, stitches were placed in the medial and lateral commissures and brought posteriorly to reduce the annular size as described by Wooler.11 The posterior margin of the repair was measured, and the residual diameter was not less than 2.5 cm. Four primary sutures were placed in the annulus, with a set of two in each commissure, and these were tied over Teflon felt. A second row of sutures were placed in the atrial wall bringing it over the initial suture line to protect the latter; the lateral row was carried to the base of the left atrial appendage. These were also tied over Teflon felt. At the termination of the procedure, two fingers could be introduced across the mitral valve without difficulty. The competence of the repair was initially tested by introducing saline through

From the Cardiology Sections, Department of Pediatrics and Department of Surgery, University of Chicago Pritzker School of Medicine, and Department of Pediatrics, Michael Reese Hospital and Medical Center, Chicago, Illinois.

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Address for reprints: Rene A. Arcilla, M.D., Department of Pediatrics, Wyler Children’s Hospital, University of Chicago, 950 E. 59th Street, Chicago, Illinois 60637.

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a left ventricular sump placed through the apex of the left ventricle. Valve competence was not tested while the heart was beating due to the risk of air embolism. The left atrium was closed with continuous suture, and air was aspirated from the left atrium, left ventricle and aorta before the aortic clamp was removed. The pressures in the right ventricle, left ventricle, and left atrium were then measured with the heart in sinus rhythm. A left atrial catheter was also left in place for pressure monitoring in the immediate post-operative period.

Right and left heart catheterization was performed with the subjects under sedation using parenteral Demerol and Vistaril, 1.5 to 1.8 mg/kg of each, to a maximum of 50 mg. In addition to the usual pressure and cardiac output (Fick) determinations, right and left ventricular as well as left atrial volumes were calculated from biplane cineangiograms using the parallelepiped method (unpublished observations, and reference 12) for the ventricles, and the area-length method for the left atrium.18 Of the 25 patients, 21 had angiograms of good contrast or free of arrhythmia to enable end-diastolic and end-systolic volume calculations of both ventricles. The contrast medium used was Renografin 76, 1.0 to 1.5 ml/kg, injected into the right atrium to avoid or minimize ectopic beats which otherwise frequently occur during high-pressure injection into the right ventricle. Border delineation of the left heart was possible from the leftophase of the atrial angiograms in 18 cases; in three others, the left heart analysis was derived from left ventricular angiograms. The left atrial volume was calculated from 19 patients. The heart rates at the time of right heart and left heart opacification were essentially identical; in no instance did they differ by more than 10 beats/min.

The cineangiographic unit consisted of biplane, dual mode (5.9 inch) Phillips image intensifiers with fixed tube-to-film distances of 81 cm in the frontal projection and 89 cm in the lateral projection. A Datacor system recorded the electrocardiogram and a pressure pulse tracing in the cinefilm. Filming rates were 64 frames per second using 35 mm cameras. The initial ventricular volume estimates, \( V_0 \), were corrected by new regression equations specific for the right ventricle (\( V_0 = 0.65 \times V_a + 0.01 \)) or for the left ventricle (\( V_0 = 0.77 \times V_a + 1.17 \)). These correcting equations were derived from a recent study using new heart cast models (Arcilla et al., unpublished observations). The initial left atrial volume estimates were likewise corrected by a regression equation specific for this chamber.

The volume data were expressed in cm\(^3\)/m\(^2\) of body surface area, and also in percent of the predicted normal using age, height, weight, and heart rate as variables. These predictive equations were derived from the cardiac volume data of 39 children, age 2.3 to 19.4 years, without demonstrable hemodynamic abnormality (see Appendix). Stroke volume and ejection fraction of both ventricles were calculated. Regurgitant volume was determined by subtracting the right ventricular stroke volume from that of the left ventricle. The severity of regurgitation was expressed by the regurgitant fraction, obtained as the ratio of the regurgitant volume to the total left ventricular stroke volume.

Relative ventricular "distensibility" was expressed by the ratio of the ventricular end-diastolic volume to the corresponding end-diastolic pressure; left atrial "distensibility" was represented by the ratio of the left atrial maximal volume to the peak "V" wave pressure as obtained from pulmonary arterial wedge tracings. These were corrected for body surface area and were expressed as cm\(^3\)/m\(^2\)/mm Hg.

The Student's \( t \)-test was used to evaluate the data; a \( P \) value of less than 0.05 was considered statistically significant.

**Results**

All patients had the typical pansystolic murmur of mitral insufficiency. None had clinical or laboratory evidence for rheumatic activity at the time of the hemodynamic studies or cardiac surgery. Nine children belonged to the NYHA functional class I, seven to class II, seven to class III and two to class IV. Cardiomegaly was observed in the chest roentgenograms of all but nine children. None showed calcification in the mitral valve area during cinefluorography. Electrocardiographic or vectorcardiographic evidence for left atrial and/or left ventricular hypertrophy was noted in all but one patient. A summary of the critical pressure and cardiac output data are given in table 1.

Left atrial maximal volume (\( L_{A_{max}} \)), was increased in all patients; left ventricular diastolic volume (LVEDV) was likewise increased in all but two patients (fig. 1). The latter had LVEDV of 83.5 cm\(^3\)/m\(^2\) and 73.6 cm\(^3\)/m\(^2\), which are still within the upper two standard deviation limits of normal (63.56 cm\(^3\)/m\(^2\) ± 11.13 sd). The \( L_{A_{max}} \) ranged from 61 to 234 cm\(^3\)/m\(^2\) (mean, 113.5); the LVEDV ranged from 74 to 223 cm\(^3\)/m\(^2\) (mean, 127.2). When expressed as percent of the predicted normal, the LVEDV ranged from 110% to 310% (mean, 196%) whereas \( L_{A_{max}} \) ranged from 136% to 660% (mean, 308%). Left ventricular ejection fractions (EF) ranged from 0.58 to 0.86 (mean, 0.72). These are comparable to those of normal children (mean, 0.71 ± 0.01 se). The right ventricular end-diastolic volumes (RVEDV) ranged from 55 to 112.2 cm\(^3\)/m\(^2\) (mean, 76.2), and the stroke volumes (SV) from 22.6 to 61.2 ml/m\(^2\) (mean, 42.6). These did not differ from the predicted normal for these subjects (mean, 71.7 cm\(^3\)/m\(^2\) and 43.5 ml/m\(^2\), respectively). Right ventricular EF ranged from 0.39 to 0.75; the mean was 0.56 ± 0.01 se, which is com-

**Table 1**

<table>
<thead>
<tr>
<th>Hemodynamic parameter</th>
<th>Range</th>
<th>Average</th>
</tr>
</thead>
<tbody>
<tr>
<td>PA SP (mm Hg)</td>
<td>18–90</td>
<td>45</td>
</tr>
<tr>
<td>PA MP (mm Hg)</td>
<td>11–75</td>
<td>35</td>
</tr>
<tr>
<td>PWP (mm Hg)</td>
<td>6–25</td>
<td>14</td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>8–24</td>
<td>14</td>
</tr>
<tr>
<td>CI (L/min/m(^2))</td>
<td>2.7–6.4</td>
<td>4.2</td>
</tr>
<tr>
<td>PVR (dynes·sec·cm(^{-5})/m(^2))</td>
<td>65–994</td>
<td>258</td>
</tr>
<tr>
<td>SVR (dynes·sec·cm(^{-5})/m(^2))</td>
<td>804–2320</td>
<td>1608</td>
</tr>
</tbody>
</table>

Abbreviations: PA SP = pulmonary artery systolic pressure; PA MP = pulmonary artery mean pressure; PWP = pulmonary wedge pressure; LVEDP = left ventricular end-diastolic pressure; CI = cardiac index; PVR = pulmonary vascular resistance; SVR = systemic vascular resistance.

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The regurgitant volumes calculated by our method were compared to those obtained by subtracting the angio-derived left ventricular stroke volume from the Fick-derived stroke output. This is shown in figure 2 which reveals excellent correlation in 18 subjects who were in good "steady" state during the diagnostic run. The regurgitant fractions (RF) derived from the 21 subjects varied from 13% to 83%; in 15 children (71%), RF ranged from 30% to 70%. In 12 patients, RF was greater than 50% and this was empirically considered to indicate severe mitral regurgitation. Those with RF of less than 50% (two cases) were considered to have mild regurgitation, and those with 30% to 50%, moderate regurgitation. Positive correlation was demonstrated between RF and the cardiothoracic ratio obtained from the frontal chest roentgenograms (fig. 3). Of nine patients with cardiothoracic ratios of less than 50% (normal), only two had RF higher than 50%; of 11 patients with cardiothoracic ratios of over 50%, two had RF of less than 50%.

There were no significant differences between the LVEF of the patients with severe mitral regurgitation (mean, 0.69 ± 0.015 SE) and those with mild-moderate regurgitation (mean, 0.71 ± 0.015 SE). Table 2 provides a summary of the hemodynamic findings in the two groups. Those with RF of more than 50% showed significantly higher pulmonary artery pressure, pulmonary capillary pressure, left ventricular end-diastolic pressure, and pulmonary vascular resistance than those with less than 50% RF. Cardiac index, obtained by the Fick method, was also lower in the former group. However, the angiographically-derived right ventricular stroke volumes in the former, although also smaller, did not differ significantly from those with RF of less than 50%. The LVEDV and LSVV were also larger in the former group, but the LAMAX did not differ significantly from those with less than 50% RF. The RVEDV were comparable in the two groups.

Analysis of the pressure-volume relations of the left ventricle and of the left atrium, expressed by relative "distensibility," revealed striking increase of left atrial distensibility (mean, 6.87 cm²/m²/mm Hg ± 0.67 SE vs normal of 2.74 ± 0.18, P < 0.001). There was also a trend for left ventricular distensibility to increase (mean, 9.57 cm²/m²/mm Hg vs normal of 7.18 ± 0.47

**Figure 1**
Comparative left ventricular end-diastolic and left atrial maximal volumes expressed in cm²/m² of body surface area. The predicted normal values for these subjects are shown by the open dots.

**Figure 2**
Comparison of regurgitant volumes calculated by the standard method, using Fick output determination (ordinate), and determined by our angiographic method (abscissa).

**Figure 3**
Correlation between regurgitant fraction and cardiothoracic ratio. The regression slope is shown by the solid line.
Table 2
Comparative Hemodynamic Data in Mitral Regurgitation

<table>
<thead>
<tr>
<th>Parameters</th>
<th>ReF &lt; 50%</th>
<th>ReF &gt; 50%</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pressure and Output Data (mean ± se)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PA mean*</td>
<td>17.1 ± 1.0</td>
<td>33.1 ± 5.8</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>PC mean*</td>
<td>10.8 ± 1.0</td>
<td>16.3 ± 1.7</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LVEDP*</td>
<td>11.8 ± 0.9</td>
<td>16.6 ± 1.5</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>PVR†</td>
<td>111 ± 11.7</td>
<td>391 ± 80.4</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>SVR†</td>
<td>1442 ± 212</td>
<td>1740 ± 127</td>
<td>NS</td>
</tr>
<tr>
<td>CI‡</td>
<td>4.9 ± 0.54</td>
<td>3.9 ± 0.23</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Cardiac Volume Data (mean, Cc predicted normal)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LAXmax</td>
<td>97.2 ± 292 Cc</td>
<td>128.1 ± 331 Cc</td>
<td>NS</td>
</tr>
<tr>
<td>LVEDV§</td>
<td>102.1 ± 150 Cc</td>
<td>150.2 ± 237 Cc</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>RVEDV§</td>
<td>76.3 ± 107 Cc</td>
<td>76.2 ± 115 Cc</td>
<td>NS</td>
</tr>
<tr>
<td>LVSV*</td>
<td>76 ± 143 Cc</td>
<td>103.1 ± 208 Cc</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>RVSV*</td>
<td>46.1 ± 102 Cc</td>
<td>38.5 ± 90 Cc</td>
<td></td>
</tr>
</tbody>
</table>

*mm Hg
βdyuness see cm³/m²
βL min/m²
βml/m²

Abbreviations: ReF = regurgitant fraction; PA = pulmonary artery; PC = pulmonary capillary; LAmax = left atrial maximal volume; LVEDV = left ventricular end-diastolic volume; RV = right ventricle; LVSV = left ventricular systolic volume; for others, see Table 1.

se, P < 0.01) although this was less apparent (fig. 4). True distensibility was not calculated because fluid-filled catheters were used for pressure recording.

Of the 11 patients who underwent annuloplasty, three were in NYHA functional class II, six in class III, and two in class IV. All but one had radiologic evidence for cardiomegaly. All had electrocardiographic evidence for left atrial enlargement and/or left ventricular hypertrophy. The pulmonary artery, pulmonary capillary, and left ventricular end-diastolic pressures were elevated in seven patients. All had RF of more than 50%. Rheumatic mitral disease was confirmed surgically in ten. In one child, a girl of 11 years with mitral insufficiency murmur for at least four years, the regurgitation was due to ruptured anterior mitral chordae tendineae; the valve itself appeared normal. All the other patients had a dilated mitral anulus with either little or moderate amount of fibrosis, thickening but no calcification of the mitral leaflets, and some adhesions of the chordae tendineae.

There were no operative or late deaths. Figure 5 summarizes the clinical data before, and one to five years after surgery. All showed striking clinical improvement except for one boy, age five years at the time of surgery, who developed carditis postoperatively but improved subsequently. The functional status reverted to NYHA class I in all but two children. The chest X-rays revealed significant reduction in heart size in all but one, and the electrocardiograms showed similar improvement in left ventricular hypertrophy. In five patients, including that of the child with ruptured chordae tendineae, the murmur of

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

**Figure 4**  
Pressure-volume relations of left ventricle (in end-diastole) and of left atrium (in end-systole) in 18 subjects. The mean and ± 2 se range, observed in normal children, are shown by the solid lines and hatched area, respectively.

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

**Figure 5**  
Summary of clinical findings following mitral annuloplasty (N = 11 cases). Auscultatory findings were graded using the usual 0–6 intensity scale. Class 4 had grade 4 or more pansystolic murmur with or without short apical diastolic rumble; class 3 had grade 3 intensity pansystolic murmur and no associated apical rumble; class 1 had borderline or only faint-high pitched apical systolic murmur; and class 0 had no murmur or only a functional ejection systolic murmur.
mitral regurgitation disappeared. Postoperative hemodynamic investigations in six patients showed significant reduction in right and left heart pressures but no change in cardiac index and systemic vascular resistance (table 3). Corresponding changes in the cardiac chambers are summarized in figure 6. The marked decrease in RF was accompanied by reduction in the left atrial and left ventricular volumes. The mean LA$_{\text{max}}$ (66.2 cm$^3$/m$^2$) remained higher than normal ($P < 0.025$) but the mean LVEDV (87.4 cm$^3$/m$^2$) did not differ significantly from the predicted normal. The drop in LVEF from a mean of 0.75 preoperatively to 0.68 postoperatively was not statistically significant.

The above changes are illustrated in the angiocardograms in figure 7. The patient, a nine-year-old boy, was in functional class IV preoperatively, with severe pulmonary hypertension and markedly elevated left heart pressures. The LVEDV was 223 cm$^3$/m$^2$ (310% of normal), LA$_{\text{max}}$ was 111 cm$^3$/m$^2$ (219% of normal), and RF was 83%. Following surgery, the clinical findings became normal. Right and left heart pressures, obtained five years after surgery, were normal; angiocardiology failed to demonstrate mitral insufficiency. The LVEDV decreased to 80 cm$^3$/m$^2$ (137% of predicted normal), and LA$_{\text{max}}$ to 43 cm$^3$/m$^2$ (135% of predicted normal).

**Discussion**

Under normal conditions, or in the absence of shunting defects or valvar regurgitation, the stroke output of both ventricles are identical. Methods currently in use for quantitating mitral regurgitation$^{14, 18}$ compare the angiographically derived left ventricular output with the systemic (forward) output determined by the Fick method or by the dye dilution technique. Our method compares the stroke outputs of both ventricles measured virtually at the same time, and by the same method.

The highly comparable regurgitant volumes estimated by our method and by the standard method were not unexpected since our subjects had no associated defects. In the presence of additional valvar disease, e.g., tricuspid regurgitation, the mitral regurgitant volume calculated by our method would be smaller than that by the standard method; the difference, theoretically speaking, would represent the
tricuspid regurgitant volume. With associated aortic insufficiency, the regurgitant volume estimated by either method would represent that of both valves combined.

The group of patients with regurgitant fractions of over 50% had larger left ventricular end-diastolic and stroke volumes, and higher pulmonary artery, pulmonary capillary and left ventricular end-diastolic pressures than those with regurgitant fractions of less than 50%. In addition, pulmonary vascular resistance was higher in the former. However, no significant difference in left atrial size was observed between the two groups. Although these differences were obviously related to the magnitude of the regurgitation, other factors such as duration of the disease and extent of myocardial damage may have played contributory roles.

Our study has shown that the degree of enlargement of the left atrium (mean, 308% of predicted normal), was much greater than that of the corresponding left ventricle (mean, 196% of predicted normal) despite their identical volume overload. This may be explained by the left atrium being the more distensible chamber. In addition, the extra volume load distends these two chambers dissimilarly. In the case of the left atrium, the regurgitant volume is transported by the left ventricular ejective force whereas this same volume load enters the left ventricle during passive ventricular filling.

True ventricular distensibility is difficult to assess, particularly in the intact heart. The passive pressure-volume filling curve is affected by alterations in heart size and ventricular geometry independently of changes in intrinsic muscle stiffness. The P-V relationship is curvilinear, and the ratio ΔV/ΔP varies, depending on which segment of the curve is being analyzed. Nevertheless, it has been suggested that the passive elastic modulus can be derived from the major exponential portion of the P-V curve. We did not attempt to analyze the ventricular P-V filling curves in our subjects due to the limited frequency response of our fluid-filled catheter-transducer system. For this type of analysis, high fidelity pressure recordings are necessary. The pressure-volume relations of the ventricle in end-diastole was instead used as an expression of relative "distensibility." When analyzed as a group, ventricular distensibility was elevated (mean, 9.57 cm³/mm Hg vs normal of 7.18 cm³/mm Hg, P < 0.01), although the individual values were often still in the normal range. The elevated left ventricular end-diastolic pressure in patients with mitral regurgitation may be a reflection of a normal or minimally altered ventricular distensibility and not necessarily of ventricular dysfunction or failure. On the other hand, normal end-diastolic pressure may also be observed in the presence of gross ventricular failure; this is accounted for by increased ventricular distensibility. Increased left atrial distensibility was a striking finding in our study. Without this left atrial change, it is likely that marked pulmonary hypertension would be more common in this disease.

The prosthetic ball valve has been conventionally used to replace the diseased mitral valve. Although the mortality is not high and clinical improvement is observed in the majority of patients, significant morbidity occurs despite the many improvements in design and the introduction of other prosthetic valves. Some of the more serious long-term complications include thromboembolic problems, infection, and left ventricular dysfunction.

While valve replacement may be necessary in patients with thick, scarred, deformed and heavily calcified valves, annuloplasty appears to be the surgery of choice for the young patients. The mitral valve in the latter subjects is still pliable, not grossly deformed, and not calcified. The flexible valve enables the ventricle to adapt well to the rapidly increasing pressure during systole, especially during isovolumic contraction, and allows for maximal ventricular filling as well. The rigid prosthetic valve with its struts protruding into the left ventricular cavity is not as hemodynamically suitable, and may eventually compromise left ventricular function.

The fate of the mitral valve following successful annuloplasty remains to be seen. By abolishing or minimizing the trauma to the valve from the regurgitation, eventual scarring and calcification may hopefully be arrested. On the other hand, even after complete correction of the regurgitation and the demonstration postoperatively of normal hemodynamics, the possibility of stenosis appearing many years later secondary to the initial (preoperative) mitral valvulitis cannot be ignored. Rheumatic mitral stenosis is a slowly progressive disease, generally becoming clinically manifest after childhood. The enthusiasm from the successful conversion of the diseased heart to a functionally normal one must, therefore, be tempered by this theoretical possibility. On the other hand, it is also quite possible that these hearts may remain normal indefinitely, although it is obvious that recurrent rheumatic attacks could lead to progressive valvar disease. This argues for continued chemoprophylaxis in these patients for an indefinite period of time.

Of the 11 patients in our series subjected to annuloplasty five have shown complete disappearance of the mitral insufficiency murmur. Postoperative hemodynamic evaluations in six have demonstrated return of the right and left heart pressures to normal, and marked reduction of the left atrial and left ventricular...
Volumes. As in the preoperative studies, the degree of residual left atrial enlargement was greater than that of the left ventricle. None of these patients had early or late postoperative complications. None of them is on anticoagulant therapy, and nine of the 11 are now in functional class I of the NYHA classification. We have not encountered any instance yet, in any of our pediatric patients, where the anatomy of the mitral valve apparatus turned out, at the time of open-heart surgery, to be unfavorable for annuloplasty with consequent valve replacement as the only other alternative.

Our current indications for annuloplasty are summarized in Table 4. Surgery should preferably be done prior to the development of left ventricular failure. Reduced left ventricular ejection fraction of 0.55 or less is, therefore, not used as one of the criteria although its presence is certainly an additional indication for surgery. Generally speaking, most if not all of the prescribed indications are present in the ideal surgical candidate. However, there are exceptions, such as the occurrence of normal pulmonary arterial or capillary wedge pressures in the face of moderate or severe mitral insufficiency due to increased left heart distensibility, or the occasional lack of correlation between the clinical and hemodynamic findings. These criteria are based on our limited clinical and hemodynamic experiences thus far. It is likely that these may have to be revised as more experience evolves.

Acknowledgment

The authors wish to thank Dr. Burton Grossman and Dr. Donald Cassels, professors of pediatrics, and Dr. Pipit Chiemmongkoltip, Assistant Professor of Pediatrics, University of Chicago, for their permission to include their patients in this study.

References


Table 4

Criteria for Mitral Annuloplasty in Isolated Mitral Regurgitation in Children*  

<table>
<thead>
<tr>
<th>Hemodynamic (major criteria)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Regurgitant fraction of 50% or more</td>
</tr>
<tr>
<td>2. Elevated pulmonary artery pressure</td>
</tr>
<tr>
<td>3. Elevated pulmonary artery wedge pressure</td>
</tr>
<tr>
<td>4. Left ventricular end-diastolic volume of 250% or more of predicted normal</td>
</tr>
</tbody>
</table>

Clinical (minor criteria)

| 1. NYHA Functional Class II or more |
| 2. Cardio-thoracic ratio of over 50% |
| 3. Left ventricular hypertrophy, moderate or severe, in ECG/VCG |

*Surgeries are indicated in presence of: 3 or more major criteria, 2 major plus 2 or more minor criteria.
Appendix

Normal Chamber Volumes and Predictive Equations in Children 2–19 Years

<table>
<thead>
<tr>
<th>Chamber</th>
<th>Volume* (cm³/m²)</th>
<th>Regression equations for prediction of normal volume (cm³)†</th>
<th>r²</th>
<th>95% Limits (±2 SD)* §</th>
</tr>
</thead>
<tbody>
<tr>
<td>RVEDV</td>
<td>75.5 ± 13.1</td>
<td>2.03 (age) + 0.29 (ht) + 1.19 (wt) + 0.009 (HR) - 11.44</td>
<td>0.90</td>
<td>68–132%</td>
</tr>
<tr>
<td>LVEDV</td>
<td>63.6 ± 11.1</td>
<td>4.22 (age) - 0.33 (ht) + 1.0 (wt) - 0.178 (HR) + 62.03</td>
<td>0.84</td>
<td>67–133%</td>
</tr>
<tr>
<td>LA_{max}</td>
<td>36.6 ± 6.8</td>
<td>2.74 (age) - 0.27 (ht) + 0.57 (wt) - 0.054 (HR) + 34.73</td>
<td>0.83</td>
<td>65–135%</td>
</tr>
</tbody>
</table>

*mean volume ± SD.
†age in yrs; height in cm; weight in kg; heart rate in beats/min.
‡r² = multiple correlation coefficient.
§2 SD range of observed/predicted × 100 (%).
For abbreviations see text tables.
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