Echocardiographic Evaluation of Fixed Left Ventricular Outlet Obstruction in Children

Pre and Postoperative Assessment of Ventricular Systolic Pressures

GREGORY L. JOHNSON, M.D., RICHARD A. MEYER, M.D., DAVID C. SCHWARTZ, M.D., JOAN KORFHAGEN, R.D.M.S., AND SAMUEL KAPLAN, M.D.

SUMMARY Recently, several investigators have utilized the echocardiographically determined magnitude of relative left ventricular posterior wall hypertrophy as a reflection of normalized systolic wall stress to estimate left ventricular systolic pressure noninvasively. In this study, relative wall thickness determined echographically was compared to peak systolic pressure measured at catheterization in 20 children without obstruction to left ventricular outflow and with normal left ventricular function. From these data a relationship, pressure = 225 × left ventricular systolic wall thickness/left ventricular end-systolic internal dimension, was derived. The relationship was then applied to 57 children with fixed aortic stenosis. Left ventricular pressure estimated echographically compared well with that demonstrated at cardiac catheterization (r = 0.89). Twenty-one patients had further echographic studies following surgical relief of outlet obstruction. Estimated left ventricular pressure fell to normal values within two months following surgery in over half the patients with good surgical relief of obstruction, and was normal at subsequent studies up to 22 months postoperatively in all but one patient with good surgical relief. In patients in whom outlet obstruction was not adequately relieved at surgery, echographically estimated left ventricular pressure remained persistently elevated.

AORTIC STENOSIS IN CHILDHOOD is characterized by concentric left ventricular myocardial hypertrophy as a consequence of the increased afterload imposed by outlet obstruction. It has been suggested that such hypertrophy develops in order to normalize systolic stress,1,2 and that such wall stress can be approximated simply as

\[
\text{stress} = \frac{\text{pressure} \times \text{cavity radius}}{\text{wall thickness}}
\]

Recently, Brodie et al.3 demonstrated that left ventricular systolic meridional wall stress can be calculated with a high degree of accuracy from left ventricular systolic wall thickness and internal dimension determined by standard M-mode echocardiography, and that wall stress calculated in this way is, in fact, normalized in patients with pressure overload of the left ventricle. With wall stress normalized in patients with discrete fixed aortic stenosis, it should then become possible to relate left ventricular peak systolic pressure in these patients to relative wall thickness, as defined by the echographic ratio of left ventricular systolic wall thickness to left ventricular systolic cavity dimension.

In this study, left ventricular peak systolic pressure measured at catheterization was related to echographically determined relative left ventricular systolic wall thickness in 20 children with normal left ventricular function and no obstruction to left ventricular outflow. A consistent relationship between relative wall thickness and left ventricular peak systolic pressure was demonstrated, and this relationship was then used to estimate left ventricular pressure in a large group of children with varying degrees of fixed aortic stenosis. The echographically estimated pressure was then compared to pressures measured at cardiac catheterization. The response of peak systolic pressure to surgical relief of outlet obstruction was also examined echographically.

Materials and Methods

The control population consisted of 20 children, aged two months–20 years (median 7 years), in whom echocardiograms were obtained at the time of hospital admission for cardiac catheterization (table 1). There were 12 males and eight females. At catheterization, 16 patients had isolated pulmonic stenosis, and four were found to have normal hearts. Left ventricular pressure tracings were obtained routinely on all 20 patients. None had signs of myocardial decompensation, either clinically or at catheterization.

Thirty-one patients with congenital cardiac lesions potentially resulting in volume overload of the left ventricle were evaluated similarly in order to determine if the relationship between relative wall thickness and left ventricular peak systolic pressure defined in the normal group held true in these patients. Twenty-eight of these patients had isolated ventricular septal defects and three had patent ductus arteriosus. All 31 patients were clinically well compensated at time of catheterization.

The study population consisted of 57 patients (age two years to 22 years, median 11 years) with catheterization-proven aortic stenosis. There were 41 males and 16 females. Site of obstruction was valvar in 50, supravalvar in one, and subvalvar with a discrete membrane demonstrated angiographically in six. Left ventricular-aortic peak systolic pressure gradients ranged from 5 to 180 mm Hg; left ventricular peak systolic pressure ranged from 110 to 258 mm Hg. All study patients underwent aortography; 29 patients had no associated aortic regurgitation demonstrable, 17 patients had very mild aortic regurgitation, five patients had aortic regurgitation graded I/IV by aortography, and six patients

From the Department of Pediatrics, College of Medicine, University of Cincinnati, Children's Hospital, Cincinnati, Ohio.

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Dr. Johnson's present address is Department of Pediatrics, University of Kentucky Medical Center, Lexington, Kentucky 40506.

Address for reprints: Richard A. Meyer, M.D., Division of Cardiology, Children's Hospital, Cincinnati, Ohio 45229.

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had grade II/IV aortic regurgitation demonstrated angiographically. In no patient was aortic insufficiency felt to be the dominant lesion.

All patients were premedicated with droperidol or pento-barbital prior to cardiac catheterization, and children between two and five years of age received ketamine anesthesia. Peak left ventricular systolic pressures were obtained with a retrograde arterial catheter in the left ventricular cavity, and pressure curves obtained were measured to the nearest one mm Hg. Forty-six patients had cardiac output determinations by the Fick method performed at catheterization. In 21 of these patients oxygen consumption was measured by use of a continuous flow-through system. In the other 25 patients output determinations were performed using an assumed oxygen consumption on the basis of age and heart rate.

Twenty-one patients in the study group came to operation during the study period. Two patients (44 and 47) required insertion of an aortic valve prosthesis, one underwent resection of supravalvar obstruction, four underwent resection of discrete subvalvar obstruction, and 14 had simple aortic valvotomy.

Left ventricular-aortic peak systolic pressure gradients were measured intraoperatively following relief of obstruction by a catheter inserted into an apical vent in the left ventricle and then passed into the ascending aorta. When this catheter was withdrawn, pressures across the left ventricular outflow tract were monitored continuously. Measurements were obtained following termination of cardiopulmonary bypass and rewarming, just prior to closure of the chest. Seventeen of the 21 patients had left ventricular-aortic peak systolic pressure gradients of 30 mm Hg or less measured in this manner; four patients had residual gradients greater than 30 mm Hg. For the purposes of this study, these measurements were assumed to give a fair approximation of residual left ventricular-aortic gradient. Due to lack of clinical indications, no study patient has yet undergone late postoperative cardiac catheterization. Two patients (40 and 49) had clinical signs of significant residual aortic regurgitation following surgery. The remaining 19 patients had clinical signs of minimal or no residual aortic valve insufficiency.

Echocardiograms were performed within 24 hours of cardiac catheterization in all patients in both control and study populations. The echocardiograms were obtained as previously described.7 Left ventricular end-systolic dimensions (D_s) were determined along a perpendicular line at the point of closest approximation of the left ventricular endocardium to the left septal surface (fig. 1). Left ventricular systolic wall thickness (T_w) was measured as the distance between the inner surface of the endocardium and the outer surface of the epicardium at the point of maximum anterior movement of the endocardium. Measurements were obtained at a point where portions of both anterior and posterior mitral leaflets were visible. Great care was taken to avoid confusing left ventricular endocardium with echoes emanating from the chordae. Five separate cardiac cycles were measured and averaged to obtain the final dimensions.

Echocardiograms were obtained in the immediate postoperative period (7–20 days, median 9 days) in 13 of the 17 patients with residual left ventricular-aortic gradients less than 30 mm Hg, and between 20 days and two months postoperatively in the remaining four. Fifteen were studied at subsequent intervals up to 22 months postoperatively. Postoperative echocardiograms were obtained during the immediate postoperative period on all four patients with residual gradients greater than 30 mm Hg, and one of these patients had follow-up studies up to 20 months postoperatively.

The ratio (C) of left ventricular peak systolic pressure (P) measured at catheterization to relative systolic wall thickness (T/D) by echocardiogram in the 20 patients comprising the control group was calculated utilizing the formula

$$C = \frac{P}{T/D}.$$  

The ratio was calculated separately for each patient and a mean value for all 20 patients was then derived.

A mean value of C was similarly derived for the 31 patients with cardiac lesions potentially resulting in volume overload of the left ventricle. The value obtained was then compared with that of the control population utilizing Student's t-test.

### Table 1: Control Group Data

<table>
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<th>Patient</th>
<th>Age (yrs-mos)</th>
<th>Diagnosis</th>
<th>P (mm Hg)</th>
<th>Ts (cm)</th>
<th>Da (cm)</th>
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<th>C</th>
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Abbreviations: P = left ventricular peak systolic pressure at catheterization; Ts = left ventricular systolic wall thickness by echo; Da = left ventricular end-systolic internal dimension by echo; C = ratio of left ventricular peak systolic pressure at catheterization to relative wall thickness by echo; PS = pulmonic stenosis; VSD = ventricular septal defect; NL = normal; FDA = patent ductus arteriosus.
Peak left ventricular systolic pressure (Pe) was echographically estimated in the patients with aortic stenosis using the formula

\[ Pe = C \times (T/D), \]

with \( C \), a constant, being the mean value derived from the control population.

**Results**

Data from the control group are shown in table 1. Values for \( C \) were similar, the mean being 225 ± 4 (SEM) mm Hg. There was no correlation between patient age and value for \( C \) (\( r = 0.36 \)). The mean value compares favorably with results obtained utilizing systolic pressure obtained by cuff blood pressure determination in the arm, rather than measured peak left ventricular pressure, described by Bennett et al.\(^6\) (\( C = 30 \text{kPa} \approx 225 \text{ mm Hg} \)) in 30 patients aged 8 to 51 years, and by Glanz et al.\(^7\) (\( C = 225 \pm 7 \text{ mm Hg} \)) in 16 normal children.

Mean value for \( C \) in the group of patients with left volume overload was 219 ± 5. There was no significant difference between this value and that obtained in the control group (\( T = 1.07 \)). However, in order to minimize the possibility of error being introduced into the evaluation of the study group, echographically estimated pressure in these patients was calculated using the value for \( C \) derived from the control group alone, that is, \( C = 225 \).

Data from the 57 patients with aortic stenosis are given in table 2. There was a strong linear correlation demonstrated between the peak left ventricular pressure estimated echographically and that demonstrated at cardiac catheterization (\( r = 0.89 \)) (fig. 2). Exclusion of patients with aortic insufficiency graded I or II/IV angiographically did not significantly change the correlation (46 patients, \( r = 0.87 \)). In addition, there was no correlation between heart rate and left ventricular pressure estimated echographically (\( r = 0.09 \)).

In nearly all the 46 patients in whom cardiac output data were calculated, there appeared to be normalization of both cardiac index and stroke volume index. No correlation was therefore attempted between output data and either invasive or noninvasive pressure determinations.

All postoperative echocardiograms were performed at least seven days postoperatively, when the patients were in a relatively stable clinical condition. Because left ventricular peak systolic pressures measured intraoperatively were frequently elevated due to transfusion and pharmacologic intervention, the left ventricular-aortic peak systolic pressure gradient obtained at that time was felt to be a more representative indicator of the degree of residual left ventricu-
ular outlet obstruction. For this reason, postoperative data were analyzed by comparing the residual intraoperative pressure gradient with the difference between echographically estimated left ventricular peak systolic pressure and simultaneous cuff blood pressure, in effect a non-invasive estimation of left ventricular-aortic systolic pressure gradient.

Results of postoperative studies are presented in table 3. All 17 patients with residual left ventricular-aortic gradients < 30 mm Hg measured intraoperatively had one or more echocardiograms performed within two months following operation. Nine of the 17 had echographically estimated left ventricular peak systolic pressures within the normal range (arbitrarily defined as Pe ≤ 15 mm Hg greater than simultaneous cuff blood pressure) 8–55 days (median 11 days) postoperatively. Six of these nine had one or more follow-up echocardiograms up to 22 months postoperatively; none of these studies demonstrated abnormally elevated Pe. Three of the nine have not yet had late postoperative follow-up.

Of the eight patients with residual intraoperative gradients < 30 mm Hg who demonstrated echographic evidence of increased left ventricular peak systolic pressure in the early postoperative period, four demonstrated normal findings at 7, 7, 8, and 15 months postoperatively, respectively, three have not yet undergone late postoperative evaluation, and one (patient 47) has shown further fall in estimated left ventricular pressure but is still slightly abnormal 14 months following aortic valve replacement.

All four patients with residual intraoperative left ventricular-aortic pressure gradients > 30 mm Hg demonstrated elevated peak systolic left ventricular pressures echographically in the first two months postoperatively. One of these four has had further echographic studies at 11 and 20 months postoperatively which demonstrate persistent elevation of echographically estimated pressure.

One of the two patients with clinically significant residual aortic regurgitation (patient 40) demonstrated an abnormally dilated left ventricular systolic dimension, and hence a low value for relative wall thickness, 26 days postoperatively. The value was normal, however, 10 months following surgery and it is possible that the early post-
operative finding may have been due to subclinical heart failure. There was no other significant difference attributable to volume overload in the postoperative echographic findings of the two patients with significant residual aortic regurgitation as compared to the other 19 patients.

**Discussion**

In this study, we have confirmed that, in the presence of normal left ventricular function, a consistent relationship can be demonstrated between left ventricular peak systolic pressure and relative left ventricular wall thickness measured echocardiographically. This relationship can be expressed as

\[
\text{Pressure (mm Hg)} = 225 \times \frac{\text{systolic wall thickness}}{\text{systolic internal diameter}}.
\]

The correlation can be utilized in children with varying degrees of severity of fixed left ventricular outflow obstruction to approximate left ventricular systolic pressure, and does not appear to be significantly affected by at least mild to moderate degrees of left ventricular volume overload, including associated aortic insufficiency. Despite the excellent correlation demonstrated between echographically estimated left ventricular peak systolic pressure and that found at catheterization, it can be appreciated readily that there is some scatter in the data and, particularly at high left ventricular pressures, the range for Pe is quite wide (fig. 2). Although there were no cases in which a patient with severe obstruction demonstrated echographic findings suggesting only mildly elevated left ventricular peak systolic pressure, the error inherent in the regression analysis must be considered when adapting the method for clinical use.

Additionally, we serially evaluated the response of relative left ventricular systolic wall thickness to surgical relief of fixed left ventricular outflow obstruction. Many factors,
such as analgesics, blood loss and relative myocardial hypoxia during perfusion may affect left ventricular function in the immediate postoperative period. For this reason, all of our “early” postoperative studies were performed after the period in which these factors would ordinarily be felt to influence the echographic findings. Nonetheless we cannot exclude the possibility that such factors may, in fact, have had some influence on findings in individual cases.

Of 17 children with apparently adequate relief of obstruction, nine demonstrated normal values for echographically estimated left ventricular pressure, determined from relative systolic wall thickness, within 60 days following operation. All of these patients who have had long-term echographic follow-up have continued to demonstrate normal findings throughout the postoperative course up to 22 months following surgery. Of the eight patients who demonstrated abnormally high values for relative wall thickness in the immediate postoperative period, five have had further echographic follow-up. Four of these five patients demonstrated normal echographic estimations of left ventricular pressure at studies between 7 and 15 months following surgery. No interval evaluation between the immediate postoperative studies and the subsequent normal studies was performed in these patients, and it is impossible to determine exactly when systolic wall stress was normalized following relief of increased ventricular afterload. Two of these four have had further echographic follow-up and have demonstrated persistently normal relative wall thickness values up to 19 months postoperatively. One patient with apparently adequate relief of outlet obstruction by insertion of an aortic valve prosthesis has demonstrated persistent mild elevation of left ventricular pressure, estimated echographically, up to 14 months following surgery. It is possible that, although no residual left ventricular-aortic gradient was measured at time of operation in this patient, the aortic valve prosthesis may be mildly obstructive.

In contradistinction to patients with good surgical relief of outlet obstruction, none of the four patients with significant residual left ventricular-aortic systolic pressure gradients demonstrated normal values for relative wall thickness in the first two months following surgery. The one patient with long-term echographic follow-up has continued to demonstrate high left ventricular systolic pressure, estimated echographically, 20 months following surgery. We feel that, in view of the data observed in the patients with adequate surgical relief of obstruction, systolic wall stress has normalized by this time and the echographically estimated pressure accurately reflects residual obstruction. Late postoperative cardiac catheterization will, of course, eventually be necessary to confirm this impression.

Aortic stenosis in childhood can follow a progressive course in the absence of clinical changes. \(^6\) \(^9\) \(^11\) A simple method of serially measuring left ventricular outlet obstruction would be useful in the clinical management and timing of cardiac catheterization in these children. Recently, we published a study of various echocardiographic indexes of left ventricular function in 45 children with fixed aortic stenosis,\(^12\) including some patients in the present study. Of the indexes studied, only the percent shortening fraction of the left ventricle correlated well with severity of left ventricular outlet obstruction. A major determinant of percent shortening fraction in patients with normal myocardial function is the degree of systolic thickening of the posterior left ventricular wall. With systolic wall stress normalized by left ventricular myocardial hypertrophy, relative systolic wall thickness becomes an expression of left ventricular peak systolic pressure. To the extent that the systolic thickening of the posterior wall contributes to percent shortening fraction, shortening fraction will increase with increasing pressure. Shortening fraction, however, is influenced by a number of parameters of myocardial function and hence does not provide the same good correlation with severity of disease provided by relative wall thickness.

The echocardiographic evaluation of relative left ventricular systolic wall thickness in children with fixed aortic stenosis without signs of myocardial decompensation would seem, from the results of the present study, to assess reasonably the severity of obstruction. As such, it should prove a useful addition to clinical, electrocardiographic, and vectorcardiographic evaluation and graded exercise testing in the management of these children. The results of surgical relief of obstruction also can be assessed, and the long-term postoperative management, when clinical and echocardiographic evaluation may be even less reliable, may also be enhanced by serial echocardiographic study.

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