Interventricular septal configuration as a predictor of right ventricular systolic hypertension in children: a cross-sectional echocardiographic study

Mary Etta King, M.D., Harold Braun, M.D.,* Allan Goldblatt, M.D., Richard Libethson, M.D., and Arthur E. Weyman, M.D.

ABSTRACT Abnormal interventricular septal position and motion have been noted in patients with right ventricular pressure overload. The quantitative relationship between this alteration in septal configuration and the severity of right ventricular systolic hypertension has not been previously reported. We used cross-sectional echocardiography to assess the radius of septal curvature at end-diastole, mid-systole, and end-systole in 20 normal children and 29 children (ages 2 weeks to 20 years) undergoing cardiac catheterization for a variety of congenital cardiac disorders. The measured septal radius of curvature (r) was normalized by the ideal radius (ri) for the left ventricular cavity area and then expressed as normalized septal curvature [1/(ri/r)]. A slight leftward shift and flattening of the interventricular septum occurred in the course of normal systolic contraction (mean ± SEM normalized curvature at end-diastole 0.92 ± 0.03 and at end-systole 0.85 ± 0.02; p < .05). Marked exaggeration of this configurational change occurred in patients with right ventricular systolic hypertension (right ventricular systolic pressure greater than 50% systemic pressure), with progressive loss of curvature from end-diastole (0.45 ± 0.05) to end-systole (0.19 ± 0.06). Normalized septal curvature correlated well with relative right ventricular systolic pressure at all three sampling periods, with the best correlation at end-systole (r = .86). End-systolic flattening of the interventricular septum thus proved to be a sensitive marker for right ventricular systolic hypertension.

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ABNORMALITIES of interventricular septal motion and configuration have been described in patients with right ventricular volume and pressure overloads.1-8 When right ventricular systolic hypertension is present, the interventricular septum shifts toward the left ventricle and becomes flattened.8-13 However, the quantitative relationship of this shift in septal position to the severity of the right ventricular systolic pressure overload has not been studied, nor has the role of septal position as a marker of right ventricular hypertension been examined.

Right ventricular hypertension is a common problem in children with acquired or congenital heart disease. Elevation of right ventricular pressure in children may result from right ventricular outflow obstruction, left ventricular inflow obstruction, or pulmonary vascular obstructive disease, and the noninvasive diagnosis of such abnormalities may be difficult, especially in a critically ill or uncooperative child. Determination and monitoring of right ventricular pressure in these patients often requires repeated cardiac catheterization with its attendant risks, costs, and complications. Thus a simple, reliable, noninvasive marker of right ventricular hypertension would be of considerable practical value. We therefore examined the relationship between right ventricular systolic pressure and septal configuration in a diverse group of children with acquired and congenital heart disease.

Materials and methods

Subjects. To determine the influence of right ventricular systolic pressure on interventricular septal position, short-axis cross-sectional echocardiographic studies of the interventricular septum were examined at end-diastole, mid-systole, and end-systole in 20 normal children (group 1) and 29 patients with catheterization-proven congenital heart disease (group 2). The 20 normal children were examined as part of a larger study of normal cardiac dimensions and were considered to be free of cardiac abnormality by history, physical examination, and complete echocardiographic study. There were eight male and 12...
female subjects (ages 2 weeks to 19 years, mean 4.8 years).

The children with congenital heart disease (group 2) were selected from a review of the records of all children who underwent two-dimensional echocardiography and cardiac catheterization at the Massachusetts General Hospital between July 1980 and July 1981. Patients with single or common ventricles or hypoplasia of either ventricular chamber were excluded, leaving a study population of 29 patients (ages 1 day to 20 years, mean 4.7 years). There were 17 male and 12 female patients. The principal cardiac diagnoses are listed in Table 1.

Because our initial experience suggested that septal position was related to relative rather than to absolute right ventricular systolic pressure, the patients with congenital heart disease were further subdivided by their relative right ventricular systolic pressures. Group 2A (low pressure) had right ventricular systolic pressures of less than 50% of their systemic pressures (n = 12). This group included seven patients with a left ventricular volume overload, three with a right ventricular volume overload, and two with no pressure or volume overload. Peak right ventricular systolic pressures ranged from 20 to 40 mm Hg (30 ± 6 mm Hg, mean ± SD). Two patients in this group had complete right bundle branch block and one had incomplete right bundle branch block. Group 2B (high pressure) had right ventricular systolic pressures that were greater than 50% of their systemic pressures (n = 17). There were 13 patients with pure right ventricular pressure overload, three with right ventricular pressure and volume overload, and one with relative right ventricular pressure overload. Peak right ventricular systolic pressures ranged from 35 to 110 mm Hg (77 ± 7 mm Hg, mean ± SD). Three patients had electrocardiographic interventricular conduction delay in the form of incomplete right bundle branch block. No patients had clinical evidence of right heart failure, and right ventricular end-diastolic pressures ranged from 2 to 10 mm Hg (mean 5.5).

**Echocardiographic and catheterization methods.** Cross-sectional echocardiograms were performed with an Advanced Technology Laboratories 300 series mechanical sector scanner with either a 3.5 or a 5 MHz transducer. Septal curvature was analyzed in the parasternal short-axis plane of the left ventricle above the papillary muscles where the distal portion of the anterior and posterior mitral valve leaflets could still be clearly discerned. All studies were reviewed in real time and appropriate segments were transferred to a video disc for frame-by-frame analysis. Representative stop-frame images at end-diastole, mid-systole, and end-systole were photographed and enlarged for quantitative analysis. End-diastole was defined as the frame preceding mitral valve closure, and end-systole as the frame preceding mitral valve opening. The mid-systolic frame was determined by counting the total number of frames from end-diastole to end-systole and dividing by 2. Figure 1 illustrates the typical normal short-axis configuration of the septum at each of these sampling periods.

To quantify septal configuration, the radius of curvature of the septal segment was measured as previously described by Brinker et al. (figure 2). Initially, the endocardial surface of the septal segment was traced. Two chords crossing separate parts of the septal arc were then drawn, and perpendicular lines bisecting the chords were constructed so that their point of intersection defined the center of the circle described by the septal arc. A straight line from the center of the circle to any point on the arc is the radius of curvature (r) for the septal segment. When the septal curvature was reversed, becoming convex toward the left ventricle, the radius of curvature was determined from the right ventricular septal surface and assigned a negative value.

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**TABLE 1**

<table>
<thead>
<tr>
<th>Cardiac diagnoses</th>
<th>n</th>
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<tbody>
<tr>
<td>Ventricular septal defect</td>
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</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>3</td>
</tr>
<tr>
<td>Pulmonic stenosis</td>
<td>3</td>
</tr>
<tr>
<td>Atrial septal defect</td>
<td>3</td>
</tr>
<tr>
<td>Patent ductus arteriosus</td>
<td>3</td>
</tr>
<tr>
<td>Congenital mitral stenosis</td>
<td>3</td>
</tr>
<tr>
<td>Primary pulmonary hypertension</td>
<td>2</td>
</tr>
<tr>
<td>Endocardial cushion defect</td>
<td>2</td>
</tr>
<tr>
<td>No significant heart disease</td>
<td>2</td>
</tr>
<tr>
<td>Transposition of the great arteries</td>
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</tr>
<tr>
<td>Mitral regurgitation</td>
<td>1</td>
</tr>
<tr>
<td>Anomalous origin, left coronary artery</td>
<td>1</td>
</tr>
</tbody>
</table>

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**FIGURE 1.** Parasternal short-axis stop-frame images of the left ventricle in a normal child at end-diastole, mid-systole, and end-systole. The typical rounded configuration of the interventricular septum is demonstrated throughout the cardiac cycle. LV = left ventricle; RV = right ventricle.
The radius of curvature was normalized for patient size in two ways. First, the left ventricular cavity area was measured for each still-frame image and the measured radius of curvature was divided by the square root of the area \((r/V^\frac{1}{2})\). Second, an idealized radius \((r_i)\) was computed for the measured cavity area, assuming that area to be perfectly circular (figure 3). The actual radius of curvature was then expressed as a function of the ideal radius of curvature \((r/r_i)\). Because these two methods differ only by the constant \(V\pi\), the data are expressed only for the normalized radii \(r/r_i\). To plot the normalized radii as continuous variables that pass through zero rather than infinity, the reciprocal function, or normalized curvature \([1/(r/r_i)]\), was used. To test interobserver variability, all echocardiographic measurements were repeated independently (both in terms of frame selection and actual measurement) by a second observer.

Cardiac catheterization was performed by standard hemodynamic and angiographic techniques, and pressures were measured with a fluid-filled catheter system. Peak systolic right ventricular pressure was compared with peak left ventricular or aortic systolic pressure.

Statistical methods. Linear regression was computed by the least squares method, and the Student’s t test was used to determine statistical significance. Interobserver and intraobserver variability was evaluated with a one-way analysis of variance and Spearman’s rank correlation.

Results

The configuration of the interventricular septum in each of the normal children (group 1) was concave toward the left ventricle at each of the sampling points (i.e., end-diastole, midsystole, and end-systole; figure 1). Although the left ventricle appeared qualitatively round in short-axis imaging, it was not perfectly circular because the mean normalized curvature at all phases of the cardiac cycle was less than unity (table 2). There was also a slight but statistically significant \((p < .05)\) flattening of the septum from end-diastole \([1/(r/r_i) = 0.92 \pm 0.3]\) to end-systole \([1/(r/r_i) = 0.85 \pm 0.02]\).

Patients in group 2A with right ventricular pressures of less than 50% of their systemic pressures showed no significant difference in their septal curvature at either of the systolic sampling periods when compared with normals (table 2). The mean end-diastolic septal curvature for this group, however, was flatter than that for normals \((0.80 \pm 0.03 vs 0.92 \pm 0.03; p < .02)\). This may reflect the inclusion of patients with right ventricular volume overload in group 2A, which altered the diastolic septal position but did not affect systolic septal configuration.

In contrast, patients with right ventricular systolic pressures that were greater than 50% of their systemic pressures (group 2B) demonstrated end-diastolic septal flattening that progressed throughout systole. This was associated with elongation of the septal radius of curvature and with actual reversal of septal curvature in some instances (figures 4 and 5). The mean normalized curvatures for group 2B are compared with those for group 2A for each sampling period in figure 6. A statistically significant difference \((p < .0001)\) was found between the mean septal radii of curvature for groups 2A and 2B at all three points in the cardiac cycle (table 2). As shown in figure 7, there was some overlap in septal curvature at end-diastole and midsystole between the two groups. At end-systole, however, there was a clear difference between those patients with right ventricular systolic pressures that were greater than 50% of their systemic pressures and those with more normal pressures. The demarcation was noted at an end-systolic normalized radius of curvature of 2.0 (or a reciprocal normalized radius of 0.5). Thus an observed end-systolic radius of curvature that was more than twice the ideal radius of curvature was extremely sensitive as a marker of right ventricular systolic hypertension.

Although a correlation was found between septal radius of curvature and relative right ventricular systolic pressure at all three points during the cardiac cycle, the relationship was clearly strongest at end-
systole. Correlation coefficients for end-diastole and midsystole were .67 and .82, respectively, whereas at end-systole the correlation coefficient was .86 (figure 8).

Interobserver variability was evaluated by comparing the radius of curvature measurements obtained independently by two observers. There was no statistically significant difference between the means of the paired observations (p < .4, one-way analysis of variance). Spearman’s rank correlation coefficient was significant at .01 when paired observations were compared. Intraobserver variability was determined for seven patients and showed no statistically significant differences between paired observations.

Discussion

We observed consistent leftward displacement and flattening of the interventricular septum in children with right ventricular systolic hypertension (right ventricular systolic pressures greater than 50% of systemic). This shift in septal position was present at end-diastole and increased progressively throughout systolic contraction. Furthermore, the absolute change in septal configuration, as reflected by the normalized septal curvature, correlated well with the relative peak systolic pressures of the right and left ventricles.

Before the effects of elevated right ventricular pressure on absolute septal position and curvature could be evaluated, it was necessary to determine the normal curvature of the septum at each of our sampling periods. Prior studies have suggested that the normal left ventricle is nearly circular at end-diastole and that alterations in this “normal” curvature are dependent on changes in the relative pressures in the two ventricles.7,10,11 We therefore anticipated that septal curvature in our control group would remain constant or increase from end-diastole to end-systole, since there should normally be a large left-to-right systolic pressure gradient. Surprisingly, we observed a progressive leftward shift in septal position during systole, with resultant septal flattening and a decrease in curvature. This shift in septal position occurred in direct oppo-
tion to the increasing left-to-right pressure gradient and suggested an active effect of muscular contraction on septal position independent of the pressures of the two ventricles.

The precise effects of contraction of various components of the muscular septal wall on septal configuration are difficult to define, since the orientation of the septal musculature varies by layer, and due to its spiral orientation, contains both longitudinally and circumferentially oriented components. Despite this complex architecture, if we simply assume that the anterior, posterior, and basal insertions of the triangular septum are fixed in space, contraction of either the circumferentially or longitudinally oriented muscular components would tend to straighten the septum. Furthermore, if there were no pressure in either ventricular chamber, isolated contraction of the septum (assuming the points of insertion are fixed) should result in a straight, taut muscular barrier midway between the right and left ventricles.

The normal systolic septal curvature must therefore represent the effect of the transseptal pressure gradient acting in opposition to the natural tendency of the septum to straighten as it contracts, with the absolute degree of curvature at any given time reflecting the relative magnitude of these forces at that instant. Since the systolic pressure on the two sides of the septum varies constantly, the absolute septal position at any temporal sampling point will be the resultant of the forces of musculature contraction tending to pull the septum toward a midline position and the instantaneous pressure gradient tending to displace it to either the right or left. During systole this is in effect a damped system, since systolic contraction should oppose the free effects of either a right-to-left or left-to-right.

FIGURE 5. Parasternal short-axis stop-frame images of the left ventricle from a patient with suprasystolic right ventricular pressures. The interventricular septum is flattened at end-diastole, and at end-systole reverses its curvature to become convex toward the left ventricle. RV = right ventricle; LV = left ventricle.

FIGURE 6. Mean reciprocal normalized radii of curvature at end-diastole, midsystole, and end-systole for group 2A and group 2B patients. Open circles, group 2A patients, with right ventricular systolic pressures of less than 50% of systemic pressures; closed circles, group 2B patients, with right ventricular systolic pressures greater than 50% of systemic pressures. The means of the two patient groups were statistically different at all three phases of the cardiac cycle ($p < .0001$), most markedly at end-systole.

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right pressure gradient. During diastole, however, the septal position should be more freely responsive to the transseptal pressure gradient alone, since it will be independent of the restraining effect generated by active musculature contraction.

The fact that the greatest septal flattening occurred at end-systole undoubtedly reflects the timing of our end-systolic sample. By using the frame preceding mitral valve opening, we included isovolumetric relaxation and thereby sampled during the phase of systole, where muscular shortening is greatest while the transseptal gradient is rapidly decreasing.

In group 2A the end-diastolic septal curvature was significantly different from that observed in normal subjects, which reflects the inclusion of patients with isolated right ventricular volume overload in this group. At the midsystolic and end-systolic sampling points, however, there was no significant difference between patients and controls in absolute septal curvature, suggesting that the hearts of these patients do not react differently from normal hearts during the period of active septal contraction.

In children with right ventricular systolic hypertension (group 2B) we noted a significantly greater shift in septal position and a greater decrease in septal curvature at each of our sampling periods than were present in the control subjects. As in our normal group, the septal shift was progressive from end-diastole to end-systole and the distortion in shape was most profound at the end-systolic sampling point. Previous investigators have also noted abnormal septal position in systole and diastole in response to a right ventricular pressure overload. In a study of experimental acute pulmonary hypertension in dogs, Stool et al. reported that increasing pulmonary artery pressure resulted in increased right ventricular volumes that in turn caused displacement of the septum and disproportionate shortening of the angiographic left ventricular septal-to-lateral axis in systole and diastole. Krayenbeul et al. also noted flattening of the interventricular septum in systole and diastole in an angiographic study of patients with chronic pulmonary hypertension. By M mode echocardiography, Goodman et al. and Pearlman et al. described diastolic and systolic leftward displacement of the interventricular septum in patients with primary pulmonary hypertension caused by enlargement of the right ventricle. Nichol et al., in an early two-dimensional echocardiographic study of pa-
patients with mitral stenosis, commented on systolic straightening of the interventricular septum and noted its association with the degree of elevation in pulmonary artery pressure. Tanaka et al.\textsuperscript{11} suggested that this shift in septal position in patients with right ventricular systolic hypertension was caused by a reversal of the normal pressure gradient across the septum and presented experimental data to support this hypothesis. Since peak right ventricular systolic pressure exceeded systemic levels in only two of our subjects, a simple reversal in the transseptal pressure gradient cannot explain the profound changes noted at mid-systole. Furthermore, since the degree of abnormal septal curvature at this sampling point appeared to form part of a continuum from end-diastole to end-systole, a more complex but nonetheless integrated series of relationships is necessary to explain the pattern and progression of these abnormalities throughout the systolic contraction period.

The end-diastolic distortion in septal configuration may be explained by the right ventricular dilatation that has been consistently observed in response to a right ventricular pressure overload and that probably accounts for the right-to-left end-diastolic transseptal pressure gradient noted by Tanaka et al.\textsuperscript{11} The progressive flattening to mid-systole, despite a persistent but diminished left-right pressure gradient, suggests an exaggeration of the normal tendency of the septum to flatten as it contracts. This results from a normal or increased force of muscular septal contraction that tends to flatten the septum, combined with a relative decrease in the gradient between the two chambers and with a lessening of the restraining effect of this gradient on septal flattening. This further explains why the septum tended to become flat as the pressure in the two chambers equalized with reversal in septal curvature when right ventricular pressure was at suprasystemic levels.

The most striking shift in septal curvature in the group with right ventricular systolic hypertension was at end-systole. This was similar in direction but far greater in magnitude than the pattern in our control group. Logically, the leftward septal displacement should be exaggerated in the hypertensive group at end-systole because of a prolongation of both the right ventricular ejection time and the isovolumetric relaxation period. The resultant dissociation between right and left ventricular isovolumetric relaxation periods would decrease or reverse the left-to-right pressure gradient at end-systole and produce a more profound shift in the interventricular septum to the left. This is again consistent with the results of studies of Tanaka et al.,\textsuperscript{11} who demonstrated a right-to-left gradient at end-systole that was associated with dissociation of the isovolumetric periods of the two ventricles.

To fully integrate these observations, it is necessary to explain why absolute septal flattening progresses from end-diastole to end-systole and is greatest at end-systole. For this to occur, the various effects of elevated right ventricular systolic pressure, (i.e., the in-

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure8.png}
\caption{Correlation of end-systolic septal curvature with relative right ventricular systolic pressure. A good correlation is apparent over the wide range of relative right ventricular pressures, with a correlation coefficient of .86. RVP = right ventricular pressure; LVP = left ventricular pressure.}
\end{figure}
crease in right ventricular diastolic volume, the change in relative pressures across the septum during midsystole, and the end-systolic delay in right ventricular isovolumetric relaxation and shift in the left-to-right pressure gradient at end-systole) must all be present in a relatively constant proportion to one another. The consistency of our data and the fact that septal position at each of the sampling periods correlates with peak right ventricular systolic pressure sampled at midsystole suggest that this is the case.

Finally, our method of patient selection deserves some comment. We elected to study patients with a wide variety of congenital disorders, including right and left ventricular volume and pressure overloads. This enabled us to study the effects of the absolute difference in pressures across the interventricular septum independent of the conditions with which these pressure differences were associated. The clear relationship of septal curvature to relative right and left ventricular pressure in this heterogenous patient population therefore suggests that septal position can be used as a marker of an alteration in the pressure relationship between the right and left ventricles regardless of the type of lesion encountered.

There are potential difficulties with the use of the septal radius of curvature as a measure of septal flattening. First, the curvature of the septum is not uniform in some patients and the absolute septal curvature may vary, depending on the region selected for measurement. In this study the predominant curvature of the septal segment was selected and the radius of curvature of that segment was determined. In our interobserver studies, in which cycles and areas of measurements were selected independently, this convention appeared to provide consistency of measurement; there was no significant variation in the curvatures derived by either of the observers. Second, in normal subjects it is possible to make the left septal surface appear flattened by inappropriate apical positioning of the transducer and angulation of the scan plane. Thus proper technique in left ventricular recording is essential if any quantitative measure of septal curvature is to be derived. Finally, a pathologic condition such as idiopathic hypertrophic subaortic stenosis may distort the left septal surface. Although these potential problems are real, careful attention to the technique of left ventricular recording and appropriate recognition of the presence of other pathologic conditions should preclude any confusion.

In summary, these data support prior observations that flattening of the interventricular septum occurs at both end-diastole and end-systole in patients with right ventricular systolic hypertension and suggest that the absolute degree of the septal displacement as measured by the normalized septal curvature can be used as a marker of this abnormal pressure relationship. The determination of the septal radius of curvature has the advantage of being a relatively simple, quantitative measurement derived from the easily obtained cross-sectional echocardiographic plane. In addition, the qualitative observation of systolic interventricular septal flattening can predict, with reasonable assurance, an elevation of right ventricular systolic pressure of greater than 50% of systemic pressure.

References

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