Assessment of Left Ventricular Function in Secundum Atrial Septal Defect by Computer Analysis of the M-mode Echocardiogram


SUMMARY Left ventricular function in 53 patients with secundum atrial septal defect was assessed by computer-assisted analysis of the left ventricular echocardiogram and by cardiac catheterization. The patients were divided into two groups, those younger and those older than 60 years, to investigate the effect of aging on left ventricular function. Cavity size was significantly smaller than normal (p < 0.01) and septal motion was abnormal in 86%, but values for cardiac index, left ventricular end-diastolic pressure, velocity of circumferential fiber shortening, left ventricular filling rate, and duration of rapid filling were normal in both groups. Regional dynamics assessed in terms of peak rates of systolic thickening and diastolic thinning of the septum and posterior wall were also normal in both groups. We concluded that, although left ventricular minor dimensions are small, and septal motion is reversed in the majority of patients with atrial septal defect, left ventricular function is normal, and it does not appear to deteriorate with increased age, pulmonary hypertension, or the presence of right ventricular failure. The abnormal septal motion appears to be compensated for by enhanced septal and posterior wall percentage thickening.

ATRIAL SEPTAL DEFEAT (ASD) is one of the most common forms of congenital heart disease in adults, and the morbidity and mortality resulting from it have usually been related to congestive heart failure. Controversy exists as to whether heart failure is due to right, left, or biventricular decompensation. Since left ventricular failure was first reported clinically by Dexter, many authors believe that left ventricular disease plays a significant role in the heart failure that limits life expectancy and accounts for the increased operative risk in elderly patients. Recent angiographic studies have indicated that left ventricular dysfunction may be present even in children with secundum ASD, so Mathew et al. suggested that the current practice of delaying surgical closure may not be the best policy. Others have suggested that left ventricular function is normal and that the structural and functional impairment from chronic right ventricular volume overload is a generalized myocardial response with eventual involvement and failure of both ventricles. Since resolution of the pathophysiologic mechanism of cardiac failure could have great impact on the timing of surgical correction, we investigated this subject. The purpose of this study was to determine 1) whether left ventricular dysfunction occurs in patients with ASD and, if so, with what magnitude and frequency, 2) the significance of reversed septal movement on left ventricular function, and 3) whether left ventricular function in these patients deteriorates with age and therefore accounts for the increased operative risk in the elderly. We investigated left ventricular function of patients with ASD by computer-assisted analysis of the left ventricular echo gram to assess septal and posterior wall dynamics in terms of peak rates of systolic thickening and diastolic thinning and their relationship to cavity filling and emptying. In addition, we analyzed left ventricular hemodynamics in all patients who underwent cardiac catheterization.

Patients

Left ventricular echocardiograms were obtained in 53 patients, in whom the diagnosis of secundum ASD had been unequivocally established by cardiac catheterization in 41 (77%) and by operation in 50 (94%). To investigate the effects of aging on left ventricular function, we divided patients with ASD into two groups. Group 1 consisted of 42 patients younger than 60 years old; 25 were female and 17 were male. Their ages ranged from 10–59 years with a mean age of 33 years (body surface area 1.69 ± 0.23 m²). Group 2 included 11 patients older than 60 years; seven were female and four were male. Their ages ranged from 60–83 years, with a mean age of 67 years (body surface area 1.71 ± 0.30 m²). No patient in either group had any additional congenital heart lesion or any known acquired condition that might have affected myocardial thickness or contractility, that is,
hypertension or coronary artery disease. Six patients in group 2 had clinical right heart failure, as evidenced by severe dependent edema, hepatomegaly, and an elevated jugular venous pressure. No patient, however, had any clinical or roentgenographic evidence of pulmonary edema that suggested concomitant left ventricular failure. All six patients were taking digoxin and diuretics regularly. No patient in group 1 had right or left ventricular failure.

Forty-one patients (77%) underwent cardiac catheterization for estimation of shunt size, pulmonary artery pressure, cardiac index, left ventricular end-diastolic pressure and pulmonary vascular resistance. Left ventricular angiograms were performed in 13 patients, and all were normal. Only two patients (in group 2) underwent coronary arteriography for chest pain, and in both the coronary arteries were normal.

Analyses of left ventricular function in the patients with ASD were compared with the data obtained from 20 normal patients (body surface area 1.77 \pm 0.27 m\(^2\)).

**Methods**

Echocardiograms were obtained with an Ekoline 20 ultrasonoscope using a 1.25-cm-diameter, 2.25-MHz transducer with a repetition frequency of 1000 cycles/sec. Recordings were made on a Cambridge Scientific Instruments multichannel or Honeywell 1856 strip chart recorder at paper speeds of 50 or 100 mm/sec, with simultaneous ECGs. Studies were made with patients recumbent in the left semilateral position. Echocardiograms were obtained of the right and left sides of the septum and of the endocardium and epicardium of the posterior left ventricular wall at the level of the chordae tendineae. Measurements were made only when these echoes were clear and continuous throughout the cardiac cycle.

Echocardiograms were digitized as previously described with the use of a Science Accessories Corporation Graf pen and digitizing table, and they were processed by a CDC 3500 computing system in the MASTER multitask mode. Data points were generated for both of the endocardial surfaces of the septum and for the endocardial and epicardial surfaces of the posterior left ventricular wall at 6-msec intervals, so that strings of coordinates were obtained for the four surface boundaries. The echoes were calibrated with points defining a time interval of 2000 msec, 4-cm depth, and two successive Q waves on the ECG encompassing the cardiac cycle to be analyzed. Plots were made of continuous computed left ventricular cavity dimension (D) and septal (VS) and posterior wall (PW) thicknesses. From these data, cavity, septal, and posterior wall dynamics were computed and displayed on a Tektronix scan converter, from which permanent records were obtained photographically and from an on-line printer. From this information, the dynamics of the left ventricular cavity, septum and posterior wall were obtained as follows.

**Parameters of Left Ventricular Cavity Dynamics**

**Percent Shortening of Cavity Minor-axis Dimension**

This was obtained by subtracting end-systolic dimension (ESD) from end-diastolic dimension (EDD) and expressing this difference as a percentage of end-diastolic dimension:

\[
\frac{\text{EDD} - \text{ESD}}{\text{EDD}} \times 100.
\]

No attempt was made to derive left ventricular volumes from these minor-axis dimensions because these calculations are based on cube function formulas that assume that the left ventricle is a prolate ellipsoid, a criterion that is not met when the left ventricular shape and geometry are changed by abnormal septal motion. For the same reason, ejection fraction was not determined.

**Peak Rate of Increase of Left Ventricular Dimension During Diastole**

This was obtained as dD/dt (diastole), where D is left ventricular dimension in centimeters and t is time in seconds. This measurement will henceforth be referred to as the "filling rate."

**Normalized Peak Rate of Left Ventricular Dimensional Shortening in Systole**

\[
\left( \frac{1}{D} \cdot \frac{\text{dD}}{\text{dt}} \right)_{\text{(systole)}}
\]

This was derived by dividing the peak rate of dimensional shortening, dD/dt (systole), by instantaneous left ventricular dimension, 1/D. This ratio is also taken to represent peak velocity of circumferential fiber shortening.

**Duration of Rapid Filling Phase**

This represents the time from minimum left ventricular dimension to the time when the left ventricular filling rate decreased to 20% of its maximum value. This point corresponds in normal subjects to the end of the rapid diastolic filling phase and is seen as the discontinuity in the plot of continuous left ventricular dimension (fig. 1). Minimum left ventricular dimension was defined as the point at which left ventricular filling rate changed from negative to positive.

**Parameters of Septal Dynamics**

**Systolic Septal Thickening (Percent)**

This was obtained by subtracting minimum diastolic thickness (VSD) from maximum systolic thickness (VSS) and expressing the difference as a percentage of minimum diastolic septal thickness:

\[
\frac{\text{VSS} - \text{VSD}}{\text{VSD}} \times 100
\]
Normalized Peak Rate of Systolic Septal Thickening

\[
\left( \frac{1}{\text{VS}} \cdot \frac{\text{dVS}}{\text{dt}} \right) \text{ (systole)}
\]

This was obtained by dividing the peak rate of systolic septal thickening, \( \frac{\text{dVS}}{\text{dt}} \) (systole), by the instantaneous septal thickness at the same point in time.

Normalized Peak Rate of Diastolic Thinning

\[
\left( \frac{1}{\text{VS}} \cdot \frac{\text{dVS}}{\text{dt}} \right) \text{ (diastole)}
\]

This was obtained by dividing the peak rate of change of septal thickness during diastole, \( \frac{\text{dVS}}{\text{dt}} \) (diastole), by the instantaneous septal thickness at the same point in time.

Time Interval From Onset of QRS Complex to Peak Septal Thickness in Relation to Timing of Peak Posterior Wall Thickness

This time interval was obtained by subtracting the time from onset of QRS to peak posterior wall thickness from the time from onset of QRS to peak septal thickness.

Parameters of Posterior Wall Dynamics

Percentage Systolic Thickening of Posterior Wall

This was obtained by subtracting minimum diastolic thickness (PWd) from maximum systolic thickness (PWs) and expressing the difference as a percentage of minimum diastolic thickness.

\[
\left( \frac{\text{PWs} - \text{PWd}}{\text{PWd}} \right) \times 100.
\]

Normalized Peak Rate of Systolic Posterior Wall Thickening

\[
\left( \frac{1}{\text{PW}} \cdot \frac{\text{dPW}}{\text{dt}} \right) \text{ (systole)}
\]

This was obtained by dividing the peak rate of systolic posterior wall thickening, \( \frac{\text{dPW}}{\text{dt}} \) (systole), by the instantaneous posterior wall thickness at the same point in time.
Normalized Peak Rate of Diastolic Posterior Wall Thinning

\[
\left( \frac{1}{PW} \cdot \frac{dPW}{dt} \right) \text{(diastole)}
\]

This was obtained by dividing the peak rate of change of posterior wall thickness during diastole, \( \frac{dPW}{dt} \) (diastole), by the instantaneous posterior wall thickness at the same point in time.

Validity

The validity of echocardiographic measurements of left ventricular minor axis and its rate of change was established by comparison with angiographic data from previous studies in normal patients\(^{10-12}\) and also in patients with abnormal septal motion.\(^{13}\)

Hemodynamics

In the 41 patients who were catheterized, measurements were made of left ventricular end-diastolic pressure, cardiac index, pulmonary artery pressure, and the ratio of pulmonary-to-systemic blood flow \((Qp/Qs)\).

The numerical data in tables 1–3 and in the Results section are arithmetic means ± SD. Statistical tests for difference were by the \( t \) test.

Results

Normal Subjects

Global Left Ventricular Function

Mean values for left ventricular end-diastolic and end-systolic dimensions and percentage shortening of the left ventricular minor axis are shown in table 1. Peak left ventricular filling rate (\(D/dt\)) in diastole was 14.5 ± 2.3 cm/sec duration of the rapid diastolic filling phase was 160 ± 50 msec, and peak velocity of circumferential fiber shortening, \(\frac{1}{D} \cdot D/dt\) (systole), was 2.66 ± 0.36 sec\(^{-1}\) (table 1).

Regional Left Ventricular Function

Maximum systolic and minimum diastolic septal and posterior wall thicknesses and their respective maximum percent thickening are shown in table 2. Normalized peak rates of septal and posterior wall systolic thickening were 3.9 ± 0.8 sec\(^{-1}\) and 4.1 ± 1.4 sec\(^{-1}\), and peak rates of diastolic thickening were 3.4 ± 0.9 sec\(^{-1}\) and 8.7 ± 4.3 sec\(^{-1}\), respectively. Peak septal thickness occurred 15 ± 13 msec before peak posterior wall thickness, and the latter occurred within 1 ± 6 msec of minimum left ventricular cavity dimension.

Patients With ASD

Global Left Ventricular Function

Mean values for end-systolic and end-diastolic left ventricular cavity dimension in patients in groups 1 and 2 were significantly \( p < 0.01 \) smaller than normal, but percent shortening of cavity minor diameter in both groups was normal (table 1). Peak left ventricular filling rate, duration of rapid diastolic filling, and peak velocity of circumferential fiber shortening in groups 1 and 2 were normal (table 1), revealing that global function was the same in patients older or younger than 60 years and was not different from that of the normal subjects.

Regional Left Ventricular Function

Septal motion was reversed in 86% of patients with ASD. Maximum systolic and minimum diastolic septal and posterior wall thicknesses in groups 1 and 2 were within the normal range. Percentage systolic thickening for septum and posterior wall was calculated from the maximum systolic and minimum diastolic thicknesses rather than from end-systolic and end-diastolic dimensions, and these values were significantly greater than normal \( p < 0.01 \). However, use of these criteria of estimation explains why percent thickening of septum and posterior wall was greater than those previously reported\(^{14}\) (table 2).

Normalized peak rates of septal and posterior wall systolic thickening in group 1 were 5.1 ± 2.1 sec\(^{-1}\) and 5.1 ± 1.7 sec\(^{-1}\) and in group 2, 5.1 ± 1.7 sec\(^{-1}\) and 5.3 ± 1.3 sec\(^{-1}\). Peak rates of septal and posterior wall diastolic thickening were 4.6 ± 2.1 sec\(^{-1}\) and 7.4 ± 2.5 sec\(^{-1}\) in group 1 and 4.7 ± 1.3 sec\(^{-1}\) and 7.7 ± 2.6 sec\(^{-1}\) in group 2 (table 2), showing that septal and posterior wall dynamics in patients with ASD were within the

### Table 1. Left Ventricular Global Function in Normal Subjects and Patients With Secundum Atrial Septal Defect

<table>
<thead>
<tr>
<th>Study group</th>
<th>No. pts</th>
<th>BSA (m(^2))</th>
<th>EDD (cm)</th>
<th>ESD (cm)</th>
<th>Fractional shortening of minor LV diameter (%):</th>
<th>dD/dt (diast)</th>
<th>Peak Vcf (sec(^{-1}))</th>
<th>Rapid filling phase (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>20</td>
<td>1.77 ± 0.27</td>
<td>4.6 ± 0.4</td>
<td>2.7 ± 0.3</td>
<td>40.2 ± 1.2</td>
<td>14.5 ± 2.3</td>
<td>2.7 ± 0.4</td>
<td>160 ± 50</td>
</tr>
<tr>
<td>Group 1 ASD</td>
<td>42</td>
<td>1.69 ± 0.23</td>
<td>3.9 ± 0.7*</td>
<td>2.2 ± 0.5</td>
<td>44.0 ± 7.3</td>
<td>13.9 ± 4.2</td>
<td>3.2 ± 0.9</td>
<td>179 ± 43</td>
</tr>
<tr>
<td>Group 2 ASD</td>
<td>11</td>
<td>1.71 ± 0.30</td>
<td>4.0 ± 0.5*</td>
<td>2.4 ± 0.4</td>
<td>40.3 ± 6</td>
<td>15.4 ± 4.7</td>
<td>3.4 ± 0.8</td>
<td>184 ± 38</td>
</tr>
</tbody>
</table>

*\( p < 0.01 \), less than normal.

Abbreviations: ASD = atrial septal defect; BSA = body surface area; ESD = end-systolic dimension; EDD = end-diastolic dimension; LV = left ventricular; Vcf = velocity of circumferential fiber shortening.
TABLE 2. Regional Left Ventricular Function in Normals and in Patients With Secundum Atrial Septal Defect

<table>
<thead>
<tr>
<th>Study group</th>
<th>No. pts</th>
<th>VS thickness (cm)</th>
<th>% VS thickening</th>
<th>PW thickness (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Diastolic</td>
<td>Systolic</td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>20</td>
<td>0.7 ± 0.2</td>
<td>1.2 ± 0.3</td>
<td>0.9 ± 0.2</td>
</tr>
<tr>
<td>Group 1 ASD (&lt; 60 years)</td>
<td>42</td>
<td>0.7 ± 0.2</td>
<td>1.3 ± 0.3</td>
<td>0.7 ± 0.2</td>
</tr>
<tr>
<td>Group 2 ASD (&gt; 60 years)</td>
<td>11</td>
<td>0.7 ± 0.2</td>
<td>1.3 ± 0.3</td>
<td>0.8 ± 0.1</td>
</tr>
</tbody>
</table>

*P < 0.01.

Abbreviations: VS = continuous septal thickness; PW = posterior wall; LV = left ventricular; ASD = atrial septal defect.

normal range and were not influenced by older age. Regardless of the direction of septal motion, normalized peak rates of change of septal dimension in systole and diastole were similar and were in the normal range. Similarly, peak rates of change of posterior wall dimension were higher during relaxation than contraction, as in normals (table 2). In addition, the close time relation between peak posterior wall thickness and minimum left ventricular cavity diameter was preserved in both group 1 and group 2 regardless of septal motion (table 2).

Mean values for left ventricular end-diastolic pressure and cardiac index were normal, mean pulmonary artery pressure was increased, and Qp/Qs was approximately 2.3 in both groups. There was no difference in the hemodynamic parameters between group 1 and group 2 except that pulmonary artery pressure was higher in group 2 (table 2).

Left ventriculograms were performed in 13 patients and were normal in all. Coronary arteriograms were performed in two patients and both were normal.

Discussion

Although patients with ASD may live a normal life span, and survival for 75 years has been well documented, the average age at death has been reported to be 39 to 49 years; many patients have died from congestive heart failure. The cause of the cardiac failure is not clear, but many investigators have indicated that left ventricular dysfunction may play a significant role. Indeed, animal experiments have indicated that chronic right ventricular volume and pressure overload results in altered left ventricular function manifest by reduction in all of the following: peak systolic pressure, peak dP/dt, wall stress, and compliance. However, how these changes relate to myocardial contractility is unknown because of the significant alterations in left ventricular cavity shape and left ventricular pressure-volume relations, both of which are important when assessing ventricular performance.

The evidence for left ventricular dysfunction in patients with ASD has been suggested clinically and angiographically but nevertheless has been largely circumstantial. Mild mitral regurgitation and systemic hypertension have been reported to be poorly tolerated and the response of the cardiac output to exercise to be reduced both preoperatively and postoperatively. Furthermore, these studies have indicated that cardiac index is low, long-axis shortening and left ventricular distensibility are reduced, and the frequently elevated right or common atrial pressure has been regarded as an increase in left ventricular filling pressure required to maintain systemic output. We still do not know why the left ventricle should fail when the right ventricle does the extra work.

Among the numerous explanations for this apparent left ventricular dysfunction, two physiologic mechanisms have been considered. One is reduced left

TABLE 3. Left Ventricular Hemodynamics

<table>
<thead>
<tr>
<th>Study group</th>
<th>No. pts</th>
<th>LVEDP</th>
<th>Cardiac index</th>
<th>PA pressure (mm Hg)</th>
<th>Qp/Qs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1 ASD</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(&lt; 60 yr)</td>
<td>32</td>
<td>12.3 ± 3.5</td>
<td>3.2 ± 0.9</td>
<td>35.0 ± 9.6</td>
<td>2.4 ± 0.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>11.2 ± 5.0</td>
<td></td>
</tr>
<tr>
<td>Group 2 ASD</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(&gt; 60 yr)</td>
<td>9</td>
<td>14.2 ± 2.8</td>
<td>3.4 ± 0.8</td>
<td>48.7 ± 13.9</td>
<td>2.3 ± 0.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>16.3 ± 4.7</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: LVEDP = left ventricular end-diastolic pressure; PA = pulmonary artery; Qp/Qs = pulmonary-to-systemic flow ratio; ASD = atrial septal defect.
ventricular filling, which has been successively thought to be due to 1) a tight pericardium as a result of increased right ventricular volume, 2) reversed Bernheim effect, and 3) systolic and diastolic left-to-right shunting at the atrial level. The net effect of reduced left ventricular filling would be to reduce myocardial fiber stretch (preload) which, according to Starling's law, would reduce systemic output. The other mechanism is left ventricular hypoplasia due to disuse atrophy, suggested by the small size of the left ventricle, its poor tolerance of stretch (preload) and left ventricular failure postoperatively when the left ventricle handles the entire systemic load for the first time.

Previously, M-mode echocardiography in patients with ASD has been used solely to enumerate the diagnostic features of right ventricular volume overload. Measurements of internal left ventricular dimensions and functional data derived from them have been considered to be unrepresentative in patients with reversed septal motion. Assessment of left ventricular function in these patients has therefore been confined to hemodynamics and angiography. The presence of reversed septal motion with its resultant abnormal cavity geometry is equally evident on left ventriculography, but has not deterred measuring the left ventricular minor axis or calculating left ventricular volume.

In the normal heart, changes in left ventricular cavity volume are brought about mainly by changes in the minor axis, with no more than a 20% reduction in the longitudinal axis. Changes in the long axis of the left ventricle in patients with ASD estimated from biplane angiography in the left anterior oblique projection are normal. The reduction in long-axis shortening observed in the right anterior oblique projection may be more apparent than real because of the changed position of the left ventricle within the chest. The enlarged right ventricle causes posterior rotation of the left ventricle so that the chord between the aortic valve and the apex of the heart is foreshortened and therefore no longer measures the same left ventricular dimension as in a normal subject. Since long-axial changes in left ventricular dimension in patients with ASD are probably normal, abnormal rates of change of cavity volume must result from abnormal changes in minor axis. Since M-mode echocardiography can reliably define the position, and therefore motion, of both surfaces of the septum and of the free wall throughout the heart cycle, we used it to assess cavity, septal, and wall dynamics.

End-systolic and end-diastolic left ventricular cavity dimensions in patients with ASD were smaller than normal, although body surface areas in the normal and ASD populations were not significantly different. Plots of continuous cavity dimension were qualitatively normal and showed a clearly identifiable rapid diastolic filling phase and a normal period of diastasis during which there was little or no change in cavity diameter (fig. 2). In spite of the small ventricular cavity diameter and reversed septal motion in the majority of patients, cavity function, as measured by percent shortening of the minor left ventricular axis, peak velocity of circumferential fiber shortening, peak left ventricular filling rate, and duration of rapid diastolic filling, was normal. The last two parameters have been repeated shown to be sensitive to even minor disturbances of left ventricular behavior. That left ventricular cavity function can remain normal in the presence of severely abnormal or reversed septal motion is shown in patients with idiopathic hypertrophic subaortic stenosis (IHSS) and in patients after mitral valve surgery. Although peak velocity of circumferential fiber shortening was normal, it must be interpreted with caution in patients with ASD and reversed septal motion because the left ventricle does not contract or relax concentrically. When patients were divided into two groups on the basis of age — those older than and those younger than 60 years old — to investigate the effects of old age on left ventricular function, no differences in cavity mechanics were demonstrable despite the presence of right-heart failure in six patients older than 60 years (group 2). Left ventricular cavity size in group 2, however, was slightly, although not significantly, increased. In addition, mean left ventricular end-diastolic pressure and cardiac index in the patients catheterized were normal, indicating that global or left ventricular cavity function in patients with ASD in these two groups was normal both hemodynamically and echocardiographically.

For further investigation of global left ventricular function, patients with ASD were arbitrarily divided into subgroups hemodynamically — those with pul-
monary artery pressure greater and less than 40 mm Hg, right atrial pressure greater and less than 10 mm Hg, right ventricular end-diastolic pressure greater and less than 8 mm Hg, left ventricular end-diastolic pressure greater and less than 10 mm Hg, Qp/Qs greater and less than 2.5, and cardiac index greater and less than 2.5 l/min/m². Patients were also subdivided into those with normal and those with abnormal septal motion, and in patients in group 2, into those with and those without right ventricular failure. Global function did not differ significantly among these subgroups.

Since left ventricular cavity (pump) function may remain normal even in the presence of major regional abnormalities of myocardial behavior, we also examined regional muscle function. Septal motion was abnormal in the majority of patients; thus, it makes a smaller contribution to cavity obliteration during ejection than in normal subjects, in whom the septum and posterior wall approximate and thereby reduce cavity diameter by almost equal amounts. However, in spite of reversed septal motion, the duration of rapid filling and peak filling rate were normal. This may be explained in part by two compensatory mechanisms: 1) increased percentage systolic thickening of the septum and 2) increased percentage systolic thickening of the posterior left ventricular wall. The latter compensatory mechanism has also been observed in patients with abnormal septal motion due to anteroseptal infarction, in patients with IHSS, and now in patients with abnormal septal motion due to ASD. Peak septal thickness occurred earlier in patients with ASD than in normal subjects, but the close time relation between peak posterior wall thickness and minimum left ventricular cavity dimension was preserved.

The left ventricular cavity is where abnormalities of myocardial muscle function resulting from increased stiffness or compliance are expressed. With computer analysis, muscle function can be quantitated in terms of peak rates of septal and posterior wall systolic thickening and diastolic thinning, and these parameters have proved to be sensitive indices of regional myocardial performance. Changes in the properties of the septum and the posterior wall, which are primary manifestations of myocardial disease, precede abnormalities of cavity function and are therefore recognizable earlier. In patients with ASD, not only was cavity function normal but the normalized peak rates of systolic thickening and diastolic thinning of the septum and free wall were also normal regardless of right atrial and pulmonary artery pressure, right and left ventricular end-diastolic pressures, cardiac index, or the size of the left-to-right shunt. Septal and posterior wall dynamics were not significantly different in the patients older than 60 years (group 2) with and without right-heart failure and in patients with normal and those with abnormal septal motion. Percent thickening of the posterior wall

**FIGURE 2.** Similar computer output of representative left ventricular echocardiogram from a 54-year-old patient with secundum atrial septal defect.
and the septum in patients with ASD and normal septal motion, however, was normal, that is, significantly different from that in patients with abnormal septal motion. These data show that the intrinsic function of the septum in these patients was normal and was independent of its direction of motion. The peak rate of thinning of the posterior wall was faster than that of the septum, as in normal subjects, indicating that in both normal subjects and in patients with ASD the left ventricular cavity filling rate was determined to a greater extent by the posterior wall than by the septum. In addition, septal and free wall dynamics were not significantly changed by older age.

We believe that regional (muscle) and cavity (pump) function of the left ventricle in patients with secundum ASD is normal regardless of age and the type of septal motion. When reversed septal motion is present, as in the majority of patients with ASD, its smaller-than-normal contribution to left ventricular cavity filling and emptying is compensated for by enhanced septal and posterior wall thickening similar to that in patients after anteroseptal infarction. The reported low resting cardiac index in no way expresses intrinsic left ventricular muscle dysfunction; rather, it reflects the prediction of Starling’s law, that reduced myocardial fiber stretch due to decreased left ventricular filling results in a lower-than-normal systemic output. The mechanism of reduced left ventricular filling may be explained by systolic and diastolic left-to-right shunting. The previous suggestions that left ventricular filling is restricted by a tight pericardium or reversed Bernheim effect are not well supported and therefore seem unlikely. Experimental evidence from isolated hearts and open-chest dogs with increased right ventricular filling suggests that left ventricular end-diastolic pressure is misleading as an index of left ventricular function and also that estimation of left ventricular volume from pressure-volume curves may result in errors of the order of 50%. These studies indicate that assessment of left ventricular function using indices of contractility that require measurement of either volume or end-diastolic pressure can be unreliable in the presence of right ventricular volume or pressure overload, and such data must therefore be interpreted with extreme caution.

A possible explanation for the role of left ventricular dysfunction in patients with ASD may be an incautious interpretation of data obtained at cardiac catheterization. The reduced preoperative cardiac output response to exercise in patients with ASD may be due to two factors: 1) that the left ventricle is small and therefore its stroke volume is small, and 2) that despite the decrease in the ratio of pulmonary to systemic blood flow from rest to exercise, left ventricular filling is still submaximal because of continuing although relatively reduced left-to-right shunting. The abnormal postoperative left ventricular function with exercise is more difficult to explain, but may be contributed to in part by variability in intraoperative myocardial preservation.

Although the right and left ventricles may fail independently, the myocardial architecture is such that in the terminal stages of right ventricular disease due either to chronic volume overload as in ASD or to chronic pressure overload as in cor pulmonale, left ventricular dysfunction may develop as the heart fails as a whole organ. In this study, left ventricular function in all patients, including six with right heart failure, was normal, indicating that the morbidity and mortality of patients with ASD cannot be ascribed to left ventricular dysfunction. We submit that left ventricular dysfunction due to right ventricular volume overload is rare and plays little part in congestive heart failure except perhaps in patients with concomitant coronary artery disease or severe mitral regurgitation due to commonly associated mitral valve prolapse. However, we had no such patients in this study.

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