The Mechanism of Abnormal Septal Motion in Atrial Septal Defect: Pre- and Postoperative Study by Radionuclide Ventriculography in Adults


SUMMARY The mechanism of abnormal interventricular septal wall (IVS) motion in atrial septal defect (ASD) was studied by radionuclide cineventriculography before and within 2 weeks of ASD closure in 11 adult patients. Pre- and postoperative right ventricular/left ventricular volume ratio (RV/LV volume), LV peak filling rate (PFR) and LV ejection fraction (EF) were measured and compared with measurements in 13 normal adults.

In normal subjects the configuration of the left ventricle was ovoid in diastole and the IVS curvature was convex toward the right ventricle. In all 11 ASD patients increased RV volume caused the IVS either to flatten during diastole or reverse its normal direction of curvature, becoming convex toward the left ventricle and resulting in a crescentic LV configuration. In early systole the IVS bulged anteriorly as the left ventricle reassumed its normal ovoid configuration and thereafter contracted normally. Postoperatively, RV volume decreased and both diastolic LV configuration and diastolic IVS curvature returned to normal in nine of the 11 patients. Postoperatively, mean RV/LV volume (± SD) decreased (3.6 ± 0.5:1 preop vs 2.1 ± 0.8:1 postop, p < 0.001; normal subjects 1.3 ± 0.1:1). PFR increased (2.13 ± 0.57/sec vs 3.16 ± 1.19/sec, p < 0.01; normal subjects 2.92 ± 1.28/sec) and EF was unchanged (0.62 ± 0.12 vs 0.69 ± 0.09; NS; normal subjects 0.66 ± 0.08). In three older patients a low LV EF returned to normal postoperatively.

Systolic anterior IVS motion in ASD is caused by an initial abnormal curvature of the IVS during diastole to accommodate increased RV volume, and the IVS curvature returns to normal when this is relieved. The increased RV/LV volume ratio decreases and indexes of LV filling and ejection may improve early after ASD closure in adults.

Although paradoxical systolic anterior motion of the interventricular septal wall (IVS) is an echocardiographic finding in atrial septal defect (ASD), its cause is uncertain. It has been reported to be caused by exaggerated systolic anterior motion of the entire heart, posterior displacement of the septum from right ventricular (RV) overload or anterior septal displacement at the onset of systole. Weyman et al., using short-axis cross-sectional echocardiography, suggested the abnormal septal motion was due to a change in the diastolic shape of the left ventricle caused by RV volume overload. Contrast left ventriculography in the left anterior oblique (LAO) projection has not shown these changes, but foreshortening of the left ventricular (LV) cavity is imaged in this view, and changes in IVS and LV shape may be obscured.

Equilibrium-gated radionuclide cineventriculography using a slant-hole collimator allows sufficient...
angulation for the IVS to be visualized normal to its longitudinal axis, which is displayed without foreshortening. IVS and LV diastolic motion can be studied by this technique before and after ASD closure. Changes in the RV/LV volume ratio and LV function were also measured.

Patients and Methods

Eleven patients, seven males and four females, ages 24–66 years (mean 41 years) with ostium secundum ASDs, were studied by equilibrium-gated radionuclide ventriculography before and within 2 weeks (mean 10 days) after ASD closure. Thirteen normal adult subjects were studied by the same technique for comparison. Informed consent was obtained for pre- and postoperative study in each patient. Diagnosis in each case was established by routine cardiac catheterization. The clinical, hemodynamic and radionuclide ventriculographic data are summarized in table 1. The pulmonary-to-systemic flow ratios ranged from 1.5 to 5.0:1 (mean 2.4:1). Two older patients (cases 1 and 2) presented with congestive heart failure and had elevated right-heart pressures.

Radionuclide Ventriculography

Equilibrium-gated radionuclide cineventriculography was performed using a method similar to that reported by Parker et al.\textsuperscript{10} Red cells were labeled in vivo by 15–20 mCi technetium-99m pertechnetate preceded by 0.01 mg/kg stannous ion.\textsuperscript{11} After equilibration imaging was performed in the modified LAO projection using a gamma camera (Ohio Nuclear 100) equipped with a 30° slant-hole, high-resolution collimator and interfaced to a PDP 11/40 computer for data acquisition. The LAO projection was adjusted until maximum separation between RV and LV blood pools was achieved and was modified by 30–45° of caudal tilt until the IVS and left ventricle were displayed without foreshortening.\textsuperscript{10}

For cineventriculographic study, counts gated to the patient’s ECG were collected by histogram mode in 11 equal time frames within each RR cycle up to 5 million counts. The composite cardiac cycle was formed by summing the 11 corresponding time segments of each cardiac cycle for the duration of the study. These were replayed in cine mode for assessment of ventricular septal and free wall motion.\textsuperscript{10}

Septal motion and shape were analyzed by three experienced observers without knowledge of the clinical data, and a consensus was obtained. No difference between observers occurred in the normal patients or in those with ASD. Blind independent observation was attempted but was not possible because the dilated right ventricle and characteristic septal motion on cine display easily identified patients with ASD from normal subjects.

In patients with ASDs, septal motion and curvature were compared with the normal patients and after the patients had correction of ASDs. Viewed in cine display, the changes in septal motion as well as ventricular dimensions that occurred after the operation were identified and agreed upon by all three observers.

Figure 1 shows the end-diastolic and end-systolic frames from the cineventriculographic study in a normal subject.

RV/LV Volume Ratio

The RV/LV volume ratio was estimated from the ratio of counts in the RV and LV blood pools at end-diastole (fig. 2). RV and LV regions of interest (ROIs)

<table>
<thead>
<tr>
<th>Pt</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Qp/Qs</th>
<th>Pressures (mm Hg)</th>
<th>RV/LV volume ratio</th>
<th>LV PFR</th>
<th>LV EF</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>RV</td>
<td>PA</td>
<td>Preop</td>
<td>Postop</td>
</tr>
<tr>
<td>1</td>
<td>M</td>
<td>52</td>
<td>3.0:1</td>
<td>60/15</td>
<td>60/22</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>66</td>
<td>2.3:1</td>
<td>40/4</td>
<td>40/10</td>
<td>3.0:1</td>
<td>1.6:1</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>43</td>
<td>2.3:1</td>
<td>23/7</td>
<td>23/11</td>
<td>3.7:1</td>
<td>2.8:1</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>32</td>
<td>1.7:1</td>
<td>22/7</td>
<td>22/12</td>
<td>3.0:1</td>
<td>1.4:1</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>31</td>
<td>2.0:1</td>
<td>28/1</td>
<td>26/6</td>
<td>4.4:1</td>
<td>2.2:1</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>52</td>
<td>2.7:1</td>
<td>35/8</td>
<td>35/10</td>
<td>3.9:1</td>
<td>3.7:1</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>35</td>
<td>2.3:1</td>
<td>23/9</td>
<td>23/8</td>
<td>3.2:1</td>
<td>1.2:1</td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>24</td>
<td>2.5:1</td>
<td>22/6</td>
<td>22/6</td>
<td>3.7:1</td>
<td>2.0:1</td>
</tr>
<tr>
<td>9</td>
<td>F</td>
<td>37</td>
<td>5.0:1</td>
<td>28/7</td>
<td>26/7</td>
<td>3.7:1</td>
<td>1.5:1</td>
</tr>
<tr>
<td>10</td>
<td>M</td>
<td>25</td>
<td>1.5:1</td>
<td>26/6</td>
<td>25/8</td>
<td>4.1:1</td>
<td>2.6:1</td>
</tr>
<tr>
<td>11</td>
<td>F</td>
<td>52</td>
<td>1.5:1</td>
<td>26/6</td>
<td>20/8</td>
<td>3.4:1</td>
<td>1.9:1</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Mean</td>
<td>3.6:1</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>sp</td>
<td>0.5</td>
</tr>
</tbody>
</table>

Abbreviations: Qp/Qs = pulmonary to systemic flow ratio; RV = right ventricular; PA = main pulmonary artery; RV/LV volume ratio = right to left ventricular volume ratio; PFR = peak filling rate; EF = ejection fraction.
were defined using a joystick and outlining the right and left ventricles in the end-diastolic image. As the pulmonary valve plane was not easily identified, the upper margin of the RV ROI was taken arbitrarily to be at the same level as the aortic–mitral valve plane and therefore may have included some portion of the main pulmonary artery. RV and LV background activity (bkg) were estimated from a semiannular area adjacent to the right and left ventricles at end-systole. The RV/LV volume ratio was calculated as

\[
\frac{\text{RV ROI counts} - \text{RV bkg counts}}{\text{LV ROI counts} - \text{LV bkg counts}}
\]

One experienced observer measured RV/LV ratios. Thirty analyses on assorted patients showed the RV/LV volume ratio could be reproduced to an accuracy of ± 5% over a range of 1.03 to 3.63:1. In the 13 normal subjects the mean RV/LV volume ratio (± SD) was 1.3 ± 0.1:1. This technique is similar to that described by Maddahi et al. Pre- and postoperative RV/LV volume ratios were measured in 10 of the 11 ASD patients but were not measured in case 1, in whom the ventricular dimensions were visually assessed from videotape recordings of his pre- and postoperative studies.

LV Function

The LV time-activity curve was obtained by a second data acquisition period with count collection restricted to a region over the left ventricle. Counts were collected by histogram mode for 48 equal time frames within each RR interval up to 1.5 million counts. The method of obtaining the LV time-activity curve from this collected data has been reported. The LV ejection fraction (EF) was calculated from the curve after seven-point quadratic smoothing. The normalized peak filling rate (PFR) was derived from the diastolic portion of the curve using a least-squares technique to fit the data to a third-degree polynomial.

In the 13 normal subjects with a mean heart rate of 66 ± 11 beats/min, mean EF was 0.66 ± 0.08 and mean normalized PFR was 2.92 ± 1.28 end-diastolic volumes (edv)/sec.
Results

Surgical Results

The secundum ASD was closed in all patients by direct suture or pericardial patch and residual left-to-right shunt excluded by dye-dilution study postoperatively. In case 6, a residual shunt was detected and resuture of a pericardial patch that had become partially detached was performed on the second postoperative day. The physical signs of congestive heart failure in cases 1 and 2 were not present after surgery.

IVS Curvature and Motion

Normal Subjects

Figure 1 shows the end-diastolic and end-systolic frames of the radionuclide ventriculographic study of a normal subject. At end-diastole the left ventricle is relatively ovoid in configuration. The IVS curvature is convex toward the right ventricle. In systole the left ventricle contracts concentrically, that is, the IVS and LV free wall move inward toward each other. All 13 normal subjects had a similar LV and IVS configuration in diastole and concentric contraction of LV walls in systole.

Atrial Septal Defect

Preoperative

Preoperatively, the IVS moved abnormally in diastole to accommodate the increased RV volume. The diastolic curvature of the IVS was reversed from normal, and instead of the normal convexity toward the right ventricle it became convex toward the left ventricle in nine patients and flattened in two. The increased RV volume and abnormal IVS curvature caused the left ventricle to assume a thin crescentic shape during diastole. In early systole the IVS bulged anteriorly as the normal ovoid configuration of the left ventricle was reassumed. When the IVS had returned to its normal curvature after the onset of systole, it then contracted inward, as in the normal heart.

Figure 3 demonstrates IVS motion from four selected frames in the preoperative radionuclide ventriculographic study of case 8. In late diastole (frame 1) the increased RV blood pool reverses the normal direction of IVS curvature, which is now convex toward the left ventricle and results in an abnormal crescentic configuration of the LV cavity. During early systole (frame 2), the IVS bulges anteriorly to reassure a normal anterior convex curvature, returning the left ventricle to a normal ovoid configuration. The IVS then contracts inward toward the LV free
wall and at end-systole (frame 3) the left ventricle is similar in size and shape to that seen at end-systole in the normal heart. In early diastole (frame 4) the IVS curvature is lost as it becomes flattened by increased RV filling, and in late diastole (frame 1) becomes convex toward the left ventricle again.

Postoperative

Figure 3B shows a typical postoperative result recorded from case 8 on the eighth postoperative day. In end-diastole (frame 1), RV volume is reduced from preoperatively and the normal ovoid configuration of the left ventricle has returned. The IVS curvature is convex toward the right ventricle, as in the normal heart, in contrast to the convex curvature toward the left ventricle preoperatively. There is normal inward contraction of the IVS and LV free wall from the onset of systole (frame 2).

Figure 4 shows the preoperative and postoperative radionuclide cineventriculograms in case 3. The preoperative study demonstrates a flat rather than curved IVS at end-diastole with IVS returning to a normal curvature in systole. The postoperative study demonstrates that although RV size has not diminished as much as that demonstrated in the previous case (fig. 3), the end-diastolic IVS curvature has changed markedly and is rounded and convex toward the right ventricle as in the normal heart. The systolic frame shows normal LV contraction.

RV/LV Volume Ratio and IVS Curvature

Qualitatively the decrease in RV cavity dimensions was very evident in most of our patients, as demonstrated by case 8 (fig. 3). The pre- and postoperative RV/LV volume ratios and diastolic IVS curvature in 10 of the 11 ASD patients are compared in figure 5. One patient (case 1), not shown in figure 5, was assessed from videotape recordings of his pre- and postoperative studies. This patient had near-normal RV size postoperatively and the reversed diastolic IVS curvature preoperatively had returned to normal. Preoperatively the RV/LV volume did not correlate with either age or pulmonary-to-systemic flow rates and ranged from 3.0 to 4.4:1 (mean 3.6:1) (normal 1.3 ± 0.1:1 p < 0.001). Postoperatively RV/LV volume ratio was decreased, ranging from 1.2 to 3.7:1, mean 2.1:1, and the average decrease was 42% (p < 0.001). Only case 6, who required reoperation, showed no significant change in RV/LV volume postoperatively.

Preoperatively the IVS was displaced during diastole, with reversal of the normal direction of IVS curvature in nine patients. In the other two patients IVS curvature was flattened, but reversal of curvature

![Figure 4](http://circ.ahajournals.org/doi/abs/10.1161/01.CIR.63.1.146)

**Figure 4.** Preoperative (A) and postoperative (B) picture taken from radionuclide cineventriculograms in case 3 demonstrating a flat septum at end-diastole (ED) preoperatively returning to a rounded curvature convex toward right ventricle (RV) postoperatively. Interventricular septal wall curvature has returned to normal despite less reduction in right ventricular size than seen in figure 3. The left ventricular ejection fraction increased from 0.47 preoperatively to 0.63 postoperatively. ES = end-systole; AO = aorta; PA = pulmonary artery; RA = right atrium; LA = left atrium.
postulated by Weyman et al.; namely, RV overload changes the diastole shape of the left ventricle. Preoperative equilibrium-gated radionuclide cineventriculography demonstrated in our patients that the increased RV volume in ASD caused the IVS to become flattened or bowed convex toward the left ventricle in diastole. This is opposite to what is seen in the normal heart, in which the IVS curvature is convex toward the right ventricle. As a result, the LV cavity forms a thin crescent instead of the ovoid shape seen in the normal heart. At the onset of systole the IVS bulges anteriorly toward the right ventricle and the left ventricle reassumes the normal ovoid shape. When the IVS has returned to its normal anterior convex curvature, it contracts inward toward the LV free wall, and at end-systole the size and shape of the left ventricle are normal. Normal inward contraction of the IVS in the late portion of systole in patients with ASD has been seen by contrast left ventriculography in studies performed by Popio et al. and by Mueller et al. They probably did not see the abnormal diastolic curvature because they used the LAO projection without caudal angulation. Meyer et al. suggested the paradoxical motion of the septum was due to the anterior systolic motion of the whole heart. An exaggerated anterior swing of the left ventricle during systole was not evident from the LAO projection in our subjects, suggesting that this is not a major mechanism for paradoxical IVS motion. The posterior displacement of the septum described by Hagan et al. is compatible with the change in shape of the IVS and left ventricle found in this study.

The early postoperative studies further suggest that abnormal curvature of the IVS during diastole is due to RV volume overload, as the normal ovoid configuration of the left ventricle and normal convexity of the IVS toward the right ventricle returned after surgical correction. Because the postoperative left ventricle was a normal ovoid shape at end-diastole, the IVS contracted inward toward the LV free wall from the onset of systole.

The RV size may only partially decrease before diastolic IVS curvature returns to normal, as in case 3 (fig. 4), who showed that the IVS curvature changed markedly when the RV/LV volume ratio postoperatively had changed only 25%. Meyer et al. showed that IVS motion returned to normal on echocardiography when the RV end-diastolic dimension index was reduced by approximately one-third; but when it was reduced by less than one-third, abnormal IVS motion persisted. In our study the only patient showing unchanged IVS curvature postoperatively also had unaltered RV/LV volume ratio. The other patients showed decreases in RV/LV volume ratio, ranging from approximately 25–60% (average 41%). RV dilatation is said to persist in older patients after ASD closure. Despite the long duration of RV volume overload, the RV cavity dilatation of most of our patients, whose average age was 41 years, appeared to be rapidly improved when the ASD was closed. This was evident both qualitatively and from the decreased RV/LV volume ratios postoperatively.

**Discussion**

The mechanism of systolic anterior IVS motion in ASD described in this study agrees with the hypothesis

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

**Figure 5.** The pre- to postoperative change in right ventricular/left ventricular volume ratio (RV/LV volume) and diastolic interventricular septal curvature is shown. Case 1 is not shown, as RV/LV volume ratio was not measured (see text).
Pre- and Postoperative LV Size and Function

LV dysfunction is said to occur in older patients with an ASD, but its pathophysiology is controversial.16–17 When pre- and postoperative LV peak filling rates were compared, LV filling was improved after surgery. Qualitatively postoperative LV cavity size appeared larger after its return to an ovoid shape, when the IVS no longer bowed into the cavity of the left ventricle. This suggests that the RV volume overload may alter LV performance by alteration of the pressure-volume relationships of the left ventricle.17 Three older patients, two of whom were in heart failure with a low EF preoperatively, had a significant increase in EF after closure of the ASD. This small experience suggests the dysfunction of the septum and abnormal LV geometry may adversely affect LV function, but is reversible after the RV overload regresses.

This study confirms that the mechanism of IVS motion seen in ASD is the result of an abnormal IVS curvature during diastole caused by RV volume overload and results in an abnormal configuration of the left ventricle. The abnormal IVS shape LV configuration and indexes of LV diastolic and systolic function may return to normal when RV overload is relieved, even when the RV overload is long-standing. These findings support the concept of functional interdependence of right and left ventricles and illustrate that RV overload influences LV function.

References
The mechanism of abnormal septal motion in atrial septal defect: pre- and postoperative study by radionuclide ventriculography in adults.  
J Hung, R F Uren, D R Richmond and D T Kelly

doi: 10.1161/01.CIR.63.1.142

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231  
Copyright © 1981 American Heart Association, Inc. All rights reserved.  
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:  
http://circ.ahajournals.org/content/63/1/142.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:  
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:  
http://circ.ahajournals.org//subscriptions/