Abnormal Motion of the Interventricular Septum in Right Ventricular Volume Overload

Experimental and Clinical Echocardiographic Studies

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SUMMARY
Abnormal systolic motion of the interventricular septum has previously been noted using echocardiography in patients with atrial septal defect and has been ascribed to right ventricular volume overload. To evaluate the mechanism responsible for this abnormality, right ventricular volume overload was created in open-chest dogs by pumping blood from the left atrium to the right atrium. In seven of eight dogs changes in interventricular septum motion were produced acutely at shunt flow as low as 500 cc/min. When the shunt was decreased or stopped, interventricular septum motion returned to normal. Shunts producing left ventricular volume overload (aorta to left atrium and right atrium to left atrium) caused no change in interventricular septum motion.

Clinical observations showed that six of seven patients with partial anomalous pulmonary venous connection and no atrial septal defect displayed abnormal interventricular septum motion on echocardiography. Twenty-one patients undergoing surgery for atrial septal defect were studied before and after operation. Sixteen of the 21 had abnormal interventricular septum motion preoperatively; the abnormal interventricular septum motion returned to normal in only one of the 16 patients during a postoperative follow-up period of up to one year. Three patients were studied only postoperatively up to six years after closure of atrial septal defect; all three retained abnormal interventricular septum motion.

Since the abnormal motion of the interventricular septum seen in patients with atrial septal defect can be produced and reversed acutely by creating a left-to-right atrial shunt in an experimental model, this abnormality must indeed be related to right ventricular volume overload. However, failure of interventricular septum motion to revert to normal following surgical correction in patients with atrial septal defect suggests that additional factors are involved in the persistence of this abnormal motion.

Additional Indexing Words:
Ultrasound  Partial anomalous pulmonary venous connection  Atrial septal defect
Ventricular compliance

Diagnostic ultrasound has been utilized in congenital cardiac lesions: conditions such as single ventricle, hypoplastic left and right heart syndromes and double-outlet right ventricle have been shown to display recognizable echocardiographic patterns. Patients with atrial septal defect (ASD) and tricuspid regurgitation were noted to have abnormalities of interventricular septum (IVS) motion during ventricular ejection. Similar abnormalities were not present in cases of ventricular septal defect, patent ductus arteriosus or pulmonic stenosis, and it was concluded that this abnormal motion of the interventricular septum is a manifestation of right ventricular volume overload (RVVO). However, recent studies have shown that surgical correction of atrial septal defect in children does not change the abnormal IVS motion to normal in a substantial proportion of cases. This casts some doubt on the hypothesis that RVVO is indeed the cause of the abnormal IVS motion.

The present study was designed to evaluate this hypothesis, by means of an experimental model and further clinical observations.
Methods

Animal Studies

An experimental model of RVVO was created in eight dogs. The animals were anesthetized with sodium pentobarbital, 30 mg/kg, and respiration was maintained by a Harvard respirator via a cuffed endotracheal tube. Periodic inflation of the lungs was performed to prevent atelectasis. A midline thoracotomy was performed, the pericardium incised and the heart exposed and lifted slightly in a pericardial sling. The right and left atrial appendages were cannulated with #22 Bardic polyvinyl multihole catheters which were connected to a Sarns heart pump with 5 in rollers. Blood was pumped from left atrium to right atrium at flow rates which were increased in stepwise fashion from 100 to 2000 cc/min (usually in increments of 100 cc/min) creating a left-to-right shunt and selective RVVO of varying magnitude. Echocardiographic recordings were obtained at each new level of shunt flow.

In four animals left ventricular volume overload (LVVO) was created by cannulating the ascending aorta and pumping blood from the aorta to left atrium (three dogs) or from right atrium to left atrium (one dog), also at progressively increasing flow rates.

Echocardiograms were recorded on a commercially available ultrasonoscope (Smith-Kline Ekoline 20, Smith-Kline Instruments, Inc., Palo Alto, California). In the animal studies the ultrasound transducer was placed lightly on the exposed anterior surface of the heart and held in place by a stationary arm. The ultrasound beam traversed the IVS and left ventricular cavity perpendicular to the endocardium/blood interfaces. Identification of the recorded echoes was verified by the rapid injection of 5 cc of normal saline through a left ventricular catheter. Cavitation at the catheter tip produces microbubbles from which the ultrasound beam is reflected; the resultant “cloud” of echoes outlines the borders of the left ventricular chamber and verifies the identification of echoes from the IVS and posterior heart wall8 (fig. 1).

Clinical Studies

Seven pediatric and adult patients with partial anomalous pulmonary venous connection and intact atrial septum were examined by echocardiography. Catheterization criteria for the diagnosis of anomalous venous connection without atrial septal defect included evidence of a left-to-right shunt by oximetry at the level of the right atrium or superior or inferior vena cava, demonstration of an early appearance of cardiogreen dye following a bolus injection into one pulmonary artery with a normal appearance following injection in the other pulmonary artery (sampling from right atrium), and failure to cross the atrial septum with a Gensini or Brockenbaugh catheter passed upward from the right femoral vein. Three patients also had pulmonary angiograms which showed no angiographic dye crossing the atrial septum. The method of Flamm et al.9 was used to estimate mixed venous return to enable calculation of Qp/Qs. Three patients underwent surgery: one (W.S.) for correction of the anomalous venous return, one (B.K.) for associated coronary artery disease, and one (B.E.) had previously been operated on to close a secundum type ASD (the anomalous vein was not corrected). In these patients presence of an anomalous vein and absence (or

![Figure 1](Path of the ultrasound beam through the heart; normal interventricular septal motion (posterior during systole) is present. A rapid injection of saline through a left ventricular catheter produces a “cloud” of echoes from microbubbles (arrow).)
previous closure) of an atrial septal defect was confirmed.

Twenty patients undergoing corrective surgery for ASD during the same period were studied by echocardiography in the preoperative period and during the early postoperative period (3–10 days) after they were stable and all indwelling catheters had been removed. When possible, further postoperative studies were obtained during follow-up clinic visits. None of these patients had a pulmonary artery systolic pressure exceeding 50 mm Hg or a right-to-left shunt. Three additional patients who had surgical correction of an ASD before echocardiography was available at this institution were examined for from one to six years following operation. None of these 23 postoperative patients had clinical or radiographic evidence of a persistent left-to-right shunt. One patient (L.L.) demonstrated persistent cardiomegaly following surgical repair of an ASD; on recatheterization no intracardiac shunt was found. In patients, standard techniques for the echocardiographic recording of the motion of the interventricular septum and posterior heart wall were employed. These involve placing the transducer on the chest wall, usually in the fourth intercostal space at the left sternal border, and initially recording the mitral valve echo. The transducer is then rotated laterally and inferiorly until echoes from the posterior ventricular wall and IVS are detected.5

Motion of the IVS, in both animal and clinical studies, was classified according to the criteria of Diamond et al.5 Normally the IVS echo begins to move posteriorly (away from the transducer) 0.04 to 0.06 sec after the onset of the QRS complex and continues to move posteriorly toward the posterior heart wall until the T wave of the ECG (fig. 2). This has been designated type N (normal) motion.5 Two abnormal patterns of IVS motion have been described. In type A motion the IVS moves anteriorly instead of posteriorly, resulting in parallel motion of the IVS and posterior heart wall, instead of the normal opposing motion of these two structures during ventricular systole. In type B abnormal motion the IVS echoes are flattened and move relatively little during ventricular ejection. Both types A and B have been ascribed to RVVO.6

The right ventricular dimension (RVD) was measured as the end-diastolic distance, in centimeters, from the right ventricular epicardial echoes to the right side of the IVS.6 The left ventricular diameter (LVD) was measured from the left side of the IVS to the echo from the endocardial surface of the posterior heart wall. In the clinical studies the RVD was divided by the body surface area and expressed as an RVD index (RVDI), in cm/m². Normal RVDI is 0.3 to 1.1 cm/m².5

Results

Animal Studies

In seven of the eight animals studied abnormalities of IVS motion were produced by left atrium to right atrium shunts (table 1). Type B septal motion was first seen in five dogs at an average shunt flow of 620 cc/min. Type A septal motion was initially noted in five dogs at an average flow of 520 cc/min. In three dogs progressive changes in IVS motion from normal through type A to type B were produced as the shunt was increased (fig. 3); in two dogs, only type B motion could be produced despite very high shunt flows (fig. 4). As shunt flow was decreased IVS motion returned to normal (fig. 5). The abnormal motion could be reproduced several times in each animal by restoring shunt flow to previous levels. RVD measurements showed anticipated changes, rising from a control mean of 0.9 cm to 1.1 cm at average shunt flow of 620 cc/min (where type B motion was first noted), to 1.3 cm at 820 cc/min (type A motion first noted) and to 1.5 cm at 1640 cc/min, the average highest shunt achieved.

One animal showed no change in IVS motion during left atrium to right atrium shunt up to 1000 cc/min, at which point ventricular fibrillation suddenly developed; resuscitation efforts were unsuccessful.

Three animals underwent aorta-to-left atrium shunts, and one a right atrium-to-left atrium shunt. Despite shunt flows up to 2000 cc/min no abnormalities of IVS motion were produced (fig. 6). Mean LVD rose from a control of 1.6 cm to a high of 2.2 cm during these shunts.

Clinical Studies

Partial Anomalous Pulmonary Venous Connection

with Intact Atrial Septum (table 2)

Six of the seven patients with PAPVC displayed abnormal motion of the IVS, four type A and two type B (fig. 7). One patient (K.H.), with the smallest left-to-right shunt in the series, showed
Table 1

<table>
<thead>
<tr>
<th>Dog</th>
<th>Control RVD or LVD (cm)</th>
<th>Type B IVS motion first noted (RVD or LVD (cm))</th>
<th>Type A IVS motion first noted (RVD or LVD (cm))</th>
<th>Highest shunt flow achieved (cc/min)</th>
<th>RVD or LVD (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1*</td>
<td>1.0</td>
<td>—</td>
<td>—</td>
<td>3000</td>
<td>2.6</td>
</tr>
<tr>
<td>2*</td>
<td>1.3</td>
<td>—</td>
<td>—</td>
<td>1000</td>
<td>1.7</td>
</tr>
<tr>
<td>3</td>
<td>0.8</td>
<td>500</td>
<td>1.1</td>
<td>900</td>
<td>1.2</td>
</tr>
<tr>
<td>4†</td>
<td>0.9</td>
<td>Not seen</td>
<td>—</td>
<td>1000</td>
<td>1.4</td>
</tr>
<tr>
<td>5</td>
<td>0.7</td>
<td>800</td>
<td>0.8</td>
<td>1400</td>
<td>0.9</td>
</tr>
<tr>
<td>6</td>
<td>0.9</td>
<td>600</td>
<td>1.5</td>
<td>1000</td>
<td>0.9</td>
</tr>
<tr>
<td>7</td>
<td>0.6</td>
<td>600</td>
<td>0.7</td>
<td>2000</td>
<td>1.5</td>
</tr>
<tr>
<td>8</td>
<td>1.3</td>
<td>600</td>
<td>1.5</td>
<td>1600</td>
<td>1.9</td>
</tr>
</tbody>
</table>

**Left atrium to right atrium shunts**

**Aorta to left atrium shunts**

**Right atrium to left atrium shunt**

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Abbreviations: IVS = interventricular septum; RVD = right ventricular dimension; LVD = left ventricular diameter.

*Shunt began at 500 cc/min and was increased in increments of 500 cc/min. In animals 3-8 shunt flow began at and was increased in increments of 100 cc/min.

†Ventricular fibrillation occurred at 1000 cc/min shunt flow; no abnormalities of IVS motion seen up to this point.

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normal IVS motion. One patient (W.S.) underwent corrective surgery. IVS motion remained abnormal at four months after surgery.

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Atrial Septal Defect (table 3)

Five of 20 patients with ASD studied preoperatively had normal motion of the IVS despite

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**LA → RA SHUNT**

**Control**

**500cc/min**

**1000cc/min**

---

*Figure 3*

Experimental left atrium to right atrium shunt. Normal septal motion present initially becomes type B at 500 cc/min and type A at 1000 cc/min. RVW = right ventricular wall; IVS = interventricular septum; EKG = electrocardiogram; LVW = left ventricular wall. ECG retouched to accentuate QRS.
Experimental left atrium to right atrium shunt. Normal septal motion present initially becomes perceptibly more flattened at shunt flow of 300 cc/min and frankly abnormal (type B) at 600 cc/min. (Type A motion was not produced despite shunt flow increases up to 1400 cc/min) ECG retouched to accentuate QRS. See fig. 3 for abbreviations. LA = left atrium; RA = right atrium.

Sixteen patients (15 ASD and 1 PAPVC) showed abnormal preoperative motion, 14 type A and 2 type B. Fifteen of these displayed persistently abnormal motion on early and late postoperative studies up to one year following surgery (fig. 8). Four of the patients showed a postoperative change from type A to type B motion (fig. 9); no patients changed from type B preoperative to type A postoperative.

One patient (R.L.) showed a gradual change from type A motion preoperative to type B motion

Experimental left atrium to right atrium shunt. Same animal as fig. 3. As shunt flow is reduced, septal motion changes from type A (500 cc/min) to type B (200 cc/min) to normal (control). See fig. 3 for abbreviations. ECG retouched to accentuate QRS.
ABNORMAL MOTION OF IV SEPTUM

**Figure 6**

Experimental aorta to left atrium shunt. Normal septal motion persists despite shunt flow of 2000 cc/min. Turbulent flow produces microbubbles in the left ventricle from which echoes are recorded (white streaks in right picture). See fig. 3 for abbreviations. ECG retouched to accentuate QRS.

The mean RVDI of the five patients with ASD and normal IVS motion was 1.9 cm/cm²; the mean RVDI of the 15 patients with ASD and type A or B IVS motion was 1.8 cm/cm². Following surgery mean RVDI decreased to 1.2 cm/cm² in the patients

Table 2

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex/Age (years)</th>
<th>Qp/Qs</th>
<th>IVS motion</th>
<th>RVDI (cm/cm²)</th>
<th>Anatomy</th>
</tr>
</thead>
<tbody>
<tr>
<td>W.S.</td>
<td>M/35</td>
<td>2.8</td>
<td>A</td>
<td>3.0</td>
<td>Right lung to SVC. &quot;Small probe-patent foramen ovale&quot; at surgery.</td>
</tr>
<tr>
<td>B.K.</td>
<td>M/60</td>
<td>1.6</td>
<td>B</td>
<td>0.6</td>
<td>Right lung to SVC. Died during surgery for coronary disease; atrial septum confirmed intact.</td>
</tr>
<tr>
<td>W.F.</td>
<td>M/42</td>
<td>2.9</td>
<td>A</td>
<td>1.4</td>
<td>Right lung to right atrium.</td>
</tr>
<tr>
<td>K.H.</td>
<td>F/23</td>
<td>1.2</td>
<td>normal</td>
<td>1.2</td>
<td>Left lung to innominate vein.</td>
</tr>
<tr>
<td>W.M.</td>
<td>M/49</td>
<td>2.1</td>
<td>B</td>
<td>1.2</td>
<td>Left lung to innominate vein. Coarctation of aorta.</td>
</tr>
<tr>
<td>C.H.</td>
<td>F/5</td>
<td>1.3</td>
<td>A</td>
<td>1.4</td>
<td>Right lung to SVC.</td>
</tr>
<tr>
<td>B.E.</td>
<td>M/14</td>
<td>*</td>
<td>A</td>
<td>2.4</td>
<td>Right lung to SVC noted at surgery for ASD in 1968.</td>
</tr>
</tbody>
</table>

Abbreviations: Qp/Qs = pulmonic/systemic flow ratio; IVS = interventricular septum; RVDI = right ventricular dimension index; SVC = superior vena cava; ASD = atrial septal defect.

*Not remeasured since ASD surgery.
Table 3

Echocardiographic Examinations of 24 Patients Before and After Surgery for ASD or PAPVC

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex/Age (years)</th>
<th>Qp/Qs</th>
<th>Preop IVS motion (cm/m²)</th>
<th>RVDI (cm/m²)</th>
<th>Early postop IVS motion (cm/m²)</th>
<th>RVDI (cm/m²)</th>
<th>Late postop IVS motion (cm/m²)</th>
<th>RVDI (cm/m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>L.J.</td>
<td>F / 5</td>
<td>2.1</td>
<td>A</td>
<td>3.3</td>
<td>B†</td>
<td>2.5</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>T.M.</td>
<td>F / 5</td>
<td>2.4</td>
<td>A</td>
<td>2.0</td>
<td>A</td>
<td>2.0</td>
<td>A (6 wks)</td>
<td>1.4</td>
</tr>
<tr>
<td>R.L.</td>
<td>F / 40</td>
<td>3.6</td>
<td>A</td>
<td>2.7</td>
<td>B</td>
<td>2.5</td>
<td>N (7 mos)</td>
<td>1.1</td>
</tr>
<tr>
<td>D.H.</td>
<td>M / 6</td>
<td>2.8</td>
<td>A</td>
<td>1.6</td>
<td>A</td>
<td>1.2</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>H.S.</td>
<td>F / 55</td>
<td>2.4</td>
<td>A</td>
<td>0.8</td>
<td>B</td>
<td>0.7</td>
<td>B (2 mos)</td>
<td>0.6</td>
</tr>
<tr>
<td>M.S.</td>
<td>F / 28</td>
<td>2.7</td>
<td>A</td>
<td>0.4</td>
<td>A</td>
<td>0.4</td>
<td>A (1 yr)</td>
<td>0.4</td>
</tr>
<tr>
<td>H.W.</td>
<td>F / 46</td>
<td>2.3</td>
<td>A</td>
<td>1.3</td>
<td>A</td>
<td>1.3</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>C.T.</td>
<td>F / 5</td>
<td>2.1</td>
<td>B</td>
<td>2.9</td>
<td>B</td>
<td>1.6</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>L.G.</td>
<td>F / 7</td>
<td>2.6</td>
<td>N</td>
<td>1.7</td>
<td>N</td>
<td>1.0</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>T.G.</td>
<td>M / 15</td>
<td>2.5</td>
<td>A</td>
<td>1.5</td>
<td>A</td>
<td>1.1</td>
<td>A (10 wks)‡</td>
<td>0.8</td>
</tr>
<tr>
<td>P.S.</td>
<td>F / 6</td>
<td>1.4</td>
<td>B</td>
<td>2.5</td>
<td>B†</td>
<td>2.4</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>J.B.</td>
<td>F / 31</td>
<td>2.5</td>
<td>N</td>
<td>1.9</td>
<td>—</td>
<td>—</td>
<td>N (3 mos)</td>
<td>1.1</td>
</tr>
<tr>
<td>W.S.</td>
<td>M / 35</td>
<td>2.8</td>
<td>A</td>
<td>3.0</td>
<td>A</td>
<td>1.1</td>
<td>A (4 mos)</td>
<td>0.7</td>
</tr>
<tr>
<td>C.A.</td>
<td>F / 34</td>
<td>4.2</td>
<td>A</td>
<td>1.6</td>
<td>A</td>
<td>1.7</td>
<td>A (4 mos)</td>
<td>1.0</td>
</tr>
<tr>
<td>R.H.</td>
<td>F / 5</td>
<td>1.6</td>
<td>N</td>
<td>3.2</td>
<td>N</td>
<td>1.5</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>T.C.</td>
<td>M / 10</td>
<td>1.8</td>
<td>N</td>
<td>0.8</td>
<td>N</td>
<td>0.7</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>J.S.</td>
<td>F / 6</td>
<td>1.5</td>
<td>N</td>
<td>1.7</td>
<td>N</td>
<td>1.4</td>
<td>N (2 mos)</td>
<td>1.1</td>
</tr>
<tr>
<td>P.B.</td>
<td>F / 24</td>
<td>2.0</td>
<td>A</td>
<td>1.6</td>
<td>A</td>
<td>2.0</td>
<td>B (6 wks)</td>
<td>0.8</td>
</tr>
<tr>
<td>B.S.</td>
<td>F / 6</td>
<td>2.1</td>
<td>A</td>
<td>2.7</td>
<td>A†</td>
<td>2.5</td>
<td>A (2 mos)</td>
<td>2.0</td>
</tr>
<tr>
<td>M.C.</td>
<td>F / 55</td>
<td>2.8</td>
<td>A</td>
<td>0.7</td>
<td>A</td>
<td>0.4</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>M.M.</td>
<td>F / 4</td>
<td>2.1</td>
<td>A</td>
<td>2.5</td>
<td>A</td>
<td>3.0</td>
<td>A (6 wks)</td>
<td>3.0</td>
</tr>
<tr>
<td>M.NelD.</td>
<td>M / 8</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>B (2 yrs)</td>
<td>1.4</td>
</tr>
<tr>
<td>L.L.</td>
<td>M / 5</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>B (1 yr)</td>
<td>2.2</td>
</tr>
<tr>
<td>F.S.</td>
<td>M / 13</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>A (6 yrs)</td>
<td>1.1</td>
</tr>
</tbody>
</table>

Abbreviations: IVS = interventricular septum; RVDI = right ventricular dimension index; Qp/Qs = pulmonic/systemic flow ratio; N = normal, A = type A, B = type B.

*All patients had ostium secundum atrial septal defects except M.S. and P.B. (ostium primum atrial septal defect) and W.S. (partial anomalous pulmonary venous connection).

† = Respiratory variation (see text).
‡ = Depending on transducer position (see text).

Discussion

Abnormal motion of the IVS was found by Diamond et al. in 31 of 33 patients with ostium secundum ASD, and in all of six patients with ostium primum ASD. IVS motion was found to be normal in patients with patent ductus arteriosus, ventricular septal defect, pulmonic stenosis and pulmonic hypertension, but was abnormal if pulmonic hypertension was complicated by tricuspid regurgitation. These findings were interpreted as suggesting that the abnormality of IVS motion during ventricular ejection was a manifestation of RVVO with unequal stroke volume—that is, the right ventricular stroke volume was large and exceeded the left ventricular stroke volume. RVVO alone was not sufficient to produce the abnormality as shown by the finding of normal IVS motion in patients with ventricular septal defect—where both right and left ventricular flow is increased, and the stroke volumes of the two ventricles remain equal.

However, two recent studies of pediatric patients undergoing surgery for ASD have shown that in no patients did the IVS motion return to normal in the early postoperative period, and it remained abnormal in 8 of 18 patients studied up to 13 months postoperative. Since closure of an ASD abolishes the RVVO, persistence of the echocardiographic abnormality suggests that chronic RVVO has produced anatomical or physiologic changes which persist after removal of the overload or that RVVO is not responsible for the echocardiographic abnormality.

The animal model was constructed to evaluate the effect of acute selective RVVO, without an anatomical abnormality of atrial or ventricular septum. Abnormal IVS motion was clearly produced and reversed acutely in this model. In
addition, LVVO did not produce abnormal IVS motion, and this correlates well with the failure to observe abnormal motion in patients with clinical conditions producing LVVO (such as mitral regurgitation, aortic regurgitation, patent ductus arteriosus).

The condition of PAPVC with intact atrial septum is a clinical analogue of the experimental model—that is, selective RVVO without an intra-cardiac defect. If the RVVO hypothesis is correct, patients with partial anomalous pulmonary venous connection (PAPVC) and no ASD should have abnormal motion of the IVS on echocardiography. Six of the seven patients studied did indeed have detectable abnormalities. The patient with normal IVS motion had a very small left-to-right shunt (Qp/Qs = 1.2). McCann et al.10 have recently reported three additional cases in which PAPVC and intact atrial septum are coupled with abnormal IVS motion.

If the chronic RVVO of the patients and the acute RVVO in the animals had produced identical changes, abolition of a left-to-right atrial shunt in patients undergoing surgery for ASD should return postoperative IVS motion to normal. Instead, abnormal IVS motion persisted up to six years.
ATRIAL SEPTAL DEFECT

Preoperative  2mos Postoperative

RVW

IVS

EKG

LVW

Figure 9

Atrial septal defect. Type A abnormal septal motion is present preoperatively and has changed to type B abnormal septal motion (septum is flattened during systole) two months postoperatively.

following closure of ASD or correction of PAPVC. This confirms previous recent observations on patients studied up to 13 months postoperative.6,7 Only one of a total of 24 patients we studied after surgery demonstrated a return to normal IVS motion, at seven months postoperative. Four patients did display a flattening of septal motion during systole—that is, they moved from type A motion preoperative to type B postoperative, while no patients changed from type B to type A motion after surgery.

Unlike the cessation of an acute experimental RVVO, surgical correction of a chronic selective RVVO in humans does not cause an immediate change in IVS motion to normal. Why might this be so? Chronic ventricular volume overload has been shown to produce changes in ventricular size and diastolic compliance,11 and it is presumably these changes which produce and maintain the observed abnormal septal motion.7 Studies in children undergoing closure of large ventricular septal defects have shown that while left ventricular wall mass shows significant postoperative declines, it remains greater than normal for an average of two years following surgery.12 Closure of experimental chronic arteriovenous shunts in dogs produces a fairly rapid but incomplete return toward normal of filling pressure and diastolic compliance. Changes in left ventricular size occur more slowly.11 These observations indicate that the changes in left ventricular filling characteristics associated with chronic ventricular volume overload may be partially irreversible. The same considerations probably apply to the right ventricle in chronic RVVO; angiographic studies have shown elevated right ventricular volume one year following ASD correction in children.13 This would explain the persistent abnormality in IVS motion despite surgical ASD closure. The observation that the mean right ventricular dimension index remained above normal following surgery supports this hypothesis. On the other hand, one might anticipate that the effect of many years of RVVO would make the IVS motion, in the older patients especially, less likely to return to normal following ASD closure, but we were unable to demonstrate differences between adult and pediatric patients in this regard.

Five of our patients with ASD and one with PAPVC had normal IVS motion. Other authors have also noted normal septal motion in some
patients with RVVO. These patients have tended to have small left-to-right shunts; in our own ASD and PAPVC patients the mean Qp/Qs of the six with RVVO and normal IVS motion was 1.9, as compared with a mean Qp/Qs of 2.4 in the 20 RVVO patients with abnormal IVS motion on preoperative echocardiograms (tables 2 and 3). Tajik et al. suggested that the magnitude of the shunt may be an important factor in determining IVS motion. This is also supported by our experimental model, where we found that frankly abnormal IVS motion during ejection was not seen below 500 cc/min of shunt flow in any animal, although subtle changes from control motion were apparent in some animals at lower flows. Abnormal IVS motion has been noted in patients with Qp/Qs as low as 1.15.

Meyer et al. suggested that changes in RVDI discriminated between patients whose IVS motion became normal after surgery and those in whom the motion remained abnormal; in the latter group the reduction of RVDI was less than one third of the preoperative figure. We were unable to confirm this, however, since five of our 15 patients whose abnormal IVS motion persisted demonstrated postoperative reductions ranging from 38% to 77% of the preoperative RVDI. Nor were these ventricular dimension measurements useful in distinguishing between the groups with various types of IVS motion either in the animal or clinical studies. Anticipated increases in RVD and LVD with experimental shunts, and declines in RVDI following corrective ASD surgery, were seen.

Technical factors are important in obtaining satisfactory echocardiograms. Variations in the direction of transducer aim have produced changes in the amplitude of echoes from the septum and posterior wall, with septal motion being exaggerated in its posterior direction during systole when the ultrasonic beam is directed toward the cardiac apex. However, we found that as long as the posterior left ventricular wall and not left atrial wall was recorded the IVS echo remained relatively constant in terms of A, B or normal IVS motion. In one patient (T.G.) the IVS motion appeared to be type A if mitral valve fragments were present and type B if they were not (the left ventricular wall was the posterior echo in both instances). In only one patient (studied five years after ASD closure) did slight variations in transducer aim produce marked changes in IVS motion, from normal to type A or B; this patient was excluded from the study. In three cases (L.J., P.S., B.S.) IVS motion appeared to vary between types A and B with respiration; since inspiration increases venous return to the right heart the magnitude of RVVO presumably increases momentarily and this may affect the recorded motion. In addition, the motion of the chest wall during respiration may have caused alterations in the direction of the ultrasound beam and thus affected the amplitude of the IVS motion, as noted above. In these cases the type of motion designated in table 3 was that observed during held expiration.

We conclude that echocardiographically abnormal motion of the IVS in patients with ASD and PAPVC is indeed related to selective RVVO. The magnitude of the RVVO is probably an important factor in the production of this abnormality, and it may not be present in patients with small left-to-right shunts. The persistence of abnormal IVS motion on ultrasound study following surgical correction of chronic RVVO suggests that additional factors are involved and is further evidence that correction of long-standing congenital or acquired cardiac lesions probably does not fully reverse presurgical abnormalities in ventricular volume, mass, and compliance. One important clinical consequence of these findings is that persistence of abnormal IVS motion following operation for any condition producing RVVO should not be taken to indicate incomplete or unsuccessful surgical correction.

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