Diminution and Closure of Large Ventricular Septal Defects After Pulmonary Artery Banding

By Zoltan G. Mesko, M.D., Jimmy E. Jones, M.D., and Alexander S. Nadas, M.D.

SUMMARY

In a period of 15 years (1957–1972), 114 infants (51 males, 63 females) with the diagnosis of ventricular septal defect and congestive heart failure underwent pulmonary artery banding at The Children’s Hospital Medical Center in Boston. A second study performed in 55 of these revealed the left-to-right shunt through the ventricular septal defect to be greatly diminished in seven and completely closed in three.

Pulmonary artery banding proved clinically beneficial to all patients restudied. Important hemodynamic changes, documented at the second study in all ten patients, included a significant decrease in left-to-right shunt and a drop in pulmonary arteriolar resistance to normal range. The right ventricular pressure, post banding, ranged from suprasystemic levels in those with muscular defects to half systemic levels in the majority of those with membranous defect.

The muscular ventricular septal defects closed by muscular tissue, the membranous defects by aneurysm formation at the “exit” of the left-to-right shunt on the right ventricular septal surface.

There is a surprisingly high closure rate (19%) in these large ventricular septal defects after pulmonary artery banding noted. Hemodynamic mechanisms to account for closure are proposed.

Additional Indexing Words:
Spontaneous closure of ventricular septal defects
Congestive heart failure Pulmonary vascular resistance
Aneurysm of ventricular septum Right ventricular systolic pressure

SPONTANEOUS CLOSURE of ventricular septal defects (VSDs) which do not precipitate congestive heart failure (CHF) happens at an estimated rate of 20–35% during infancy. These defects are thought to be small or medium sized (5-6 mm) in diameter, and are restrictive, with a left ventricular (LV) to right ventricular (RV) systolic pressure gradient, and a small left-to-right shunt. Large VSDs (diameter greater than 1.0 cm) with RV pressure near systemic level and a sizable left-to-right shunt, with accompanying CHF, close spontaneously at a rate of 5–7%. The procedure of pulmonary artery banding (PAB) was introduced in 1952 by Muller and Damman. Seven cases of spontaneous closure of VSD after PAB have been recorded. The present report summarizes our experience at The Children’s Hospital Medical Center in Boston with spontaneous closure or diminution of large VSDs after PAB.

Clinical Data

Between 1957 and 1972 114 infants (51 males, 63 females) with the principle diagnosis of large VSD and severe CHF, confirmed at cardiac catheterization, underwent PAB at The Children’s Hospital Medical Center in Boston. Preparatory to complete correction, 55 of these patients (24 males, 31 females), who had been banded 22 months to 11 years (mean 4.3) previously underwent cardiac catheterization. In seven of these children, the shunt through the VSD was greatly diminished and in three others the defect was thought to be completely closed. These ten children form the basis of the present report.

Results

Preoperative Profile

At their first hospital admission, nine of the infants were under five months of age (six of them two months old or less) and one baby was ten months old. The histories revealed normal birth weights in all except one, whose weight at birth was 2.3 kg. They were all in poor general condition, below the third percentile in weight.
Cardiac examination showed the auscultatory findings of a large VSD in all, and in one baby (pt. 5), the murmur of patent ductus arteriosus also. There was significant cardiomegaly with a cardiac index greater than 0.6 in all and increased pulmonary blood flow. Biventricular hypertrophy was demonstrated on the electrocardiogram of nine infants; one baby (pt. 4) had pure right ventricular hypertrophy.

Hemodynamic studies (table 1) confirmed the diagnosis of VSD with a Qp/Qs ratio of 3:1 or more in all but two infants (pts. 3, 5). The presence of an atrial septal defect was suggested in one baby (pt. 10) by a 17% step-up at the right atrial level and was confirmed by angiocardiogram at the second catheterization. In four infants (2, 6, 7, 10) a patent ductus arteriosus was demonstrated by passage of the catheter. A mild gradient (20–31 mm Hg) between the ascending and descending aorta was demonstrated in four babies (1, 3, 5, 6). The RV systolic pressure was at or near systemic level in eight patients; in two (4, 9) the RV pressure was half systemic level. LV end-diastolic pressure was above 5 mm Hg in seven patients. Pulmonary arteriolar resistance was 2 units/m² or more in all but two infants (8, 9).

Surgery

At operation, usually within days after catheterization, the main pulmonary artery was constricted by cotton tape, reducing distal pulmonary artery pressure by at least 50%. During this maneuver, pressure in the systemic artery usually increased while the systemic arterial oxygen saturation did not change significantly. During the PAB operation the patent ductus arteriosus was divided in the four patients with this defect, and in three the coarcted segment of the aorta was resected (3, 5, 6).

Postoperative Profile

The postoperative period was uneventful. Clinical status improved, CHF disappeared, the babies rapidly gained weight and their growth rate was accelerated. Most were asymptomatic at the end of the first year and continued to thrive (table 2).

At the time of the second catheterization, auscultation showed harsh ejection systolic murmur, loudest at the upper left sternal border, without a diastolic rumble, and a widely split second sound with a soft pulmonic component. The electrocardiogram showed pure RV hypertrophy in seven patients; in addition, severe RV "strain" was noted in patient 1 with severe RV hypertension. In the

<table>
<thead>
<tr>
<th>Pt. no.</th>
<th>Sex</th>
<th>Age at cath</th>
<th>PAP mm Hg</th>
<th>PPA mm Hg</th>
<th>PPA mm Hg</th>
<th>Qp/Qs</th>
<th>Rp U/m²</th>
<th>Type of VSD</th>
<th>Additional lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>2 mo</td>
<td>100/5</td>
<td>107/33</td>
<td>112/56</td>
<td>3/1</td>
<td>6.6</td>
<td>Mu</td>
<td>Coarctation of Aorta</td>
</tr>
<tr>
<td>5 yr</td>
<td></td>
<td></td>
<td>117/54</td>
<td>1/1.1</td>
<td>1.0</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>2 mo</td>
<td>65/4</td>
<td>60/25</td>
<td>75/40</td>
<td>3/1</td>
<td>2.0</td>
<td>Mu</td>
<td>PDA</td>
</tr>
<tr>
<td>2 yr</td>
<td></td>
<td></td>
<td>88/65</td>
<td>1.1/1</td>
<td>1.0</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>2 mo</td>
<td>107/40</td>
<td>85/53</td>
<td>2.5/1</td>
<td>7.0</td>
<td></td>
<td>Mu-Multiple</td>
<td>Coarctation of Aorta</td>
</tr>
<tr>
<td>3 yr</td>
<td></td>
<td></td>
<td>135/3</td>
<td>130/75</td>
<td>1.1</td>
<td>2.2</td>
<td></td>
<td>Me-Single</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>5 mo</td>
<td>62/8</td>
<td>54/33</td>
<td>105/69</td>
<td>3/1</td>
<td>3.0</td>
<td>Me</td>
<td></td>
</tr>
<tr>
<td>3½ yr</td>
<td></td>
<td></td>
<td>121/65</td>
<td>1.2/1</td>
<td>1.6</td>
<td></td>
<td></td>
<td></td>
<td>Coarctation of Aorta</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>3 mo</td>
<td>86/8</td>
<td>73/23</td>
<td>89/33</td>
<td>2.6/1</td>
<td>2.9</td>
<td>Me</td>
<td>PDA</td>
</tr>
<tr>
<td>4 yr</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>10 mo</td>
<td>64/2</td>
<td>60/33</td>
<td>76/49</td>
<td>3/1</td>
<td>2.8</td>
<td>Me</td>
<td>PDA</td>
</tr>
<tr>
<td>5 yr</td>
<td></td>
<td></td>
<td>120/80</td>
<td>1.1</td>
<td>1.4</td>
<td></td>
<td></td>
<td></td>
<td>Me-Multiple</td>
</tr>
<tr>
<td>7</td>
<td>F</td>
<td>4 mo</td>
<td>70/6</td>
<td>70/45</td>
<td>98/50</td>
<td>3/1</td>
<td>2.2</td>
<td>Me</td>
<td>PDA</td>
</tr>
<tr>
<td>6 yr</td>
<td></td>
<td></td>
<td>124/64</td>
<td>1.2/1</td>
<td>1.0</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>F</td>
<td>1 mo</td>
<td>50/6</td>
<td>60/40</td>
<td>4/1</td>
<td>1.0</td>
<td></td>
<td>Me</td>
<td></td>
</tr>
<tr>
<td>7½ yr</td>
<td></td>
<td></td>
<td>102/70</td>
<td>1.1</td>
<td>1.0</td>
<td></td>
<td></td>
<td></td>
<td>Single</td>
</tr>
<tr>
<td>9</td>
<td>M</td>
<td>4 mo</td>
<td>65/5</td>
<td>58/38</td>
<td>102/50</td>
<td>3/1</td>
<td>1.2</td>
<td>Me</td>
<td></td>
</tr>
<tr>
<td>11 yr</td>
<td></td>
<td></td>
<td>95/72</td>
<td>1.3/1</td>
<td>1.2</td>
<td></td>
<td></td>
<td></td>
<td>Single</td>
</tr>
<tr>
<td>10</td>
<td>F</td>
<td>2 mo</td>
<td>88/7</td>
<td>86/32</td>
<td>90/32</td>
<td>3/1</td>
<td>2.2</td>
<td>Me</td>
<td>PDA</td>
</tr>
<tr>
<td>6 yr</td>
<td></td>
<td></td>
<td>120/65</td>
<td>1.6/1</td>
<td>1.0</td>
<td></td>
<td></td>
<td></td>
<td>ASD</td>
</tr>
</tbody>
</table>

Abbreviations: Pt. = patient; cath = catheterization; P = pressure; RV = right ventricle; PA = pulmonary artery; FA = femoral artery; Qp = pulmonary flow; Qs = systemic flow; Rp = pulmonary arteriolar resistance; Rs = systemic resistance; Mu = muscular; Me = membranous; ASD = atrial septal defect; PDA = patent ductus arteriosus.

*Distal to pulmonary artery band.
Table 2

Development of Patients (in % of Normal Growth*) with Large Ventricular Septal Defect and Pulmonary Artery Band at the First and Second Cardiac Catheterization

<table>
<thead>
<tr>
<th>Pt. no.</th>
<th>Weight 1st cath</th>
<th>Height 1st cath</th>
<th>Weight 2nd cath</th>
<th>Height 2nd cath</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>&lt;3%</td>
<td>&lt;3%</td>
<td>10%</td>
<td>10%</td>
</tr>
<tr>
<td>2</td>
<td>&lt;3%</td>
<td>&lt;10%</td>
<td>10%</td>
<td>50%</td>
</tr>
<tr>
<td>3</td>
<td>&lt;3%</td>
<td>&lt;25%</td>
<td>25%</td>
<td>50%</td>
</tr>
<tr>
<td>4</td>
<td>&lt;3%</td>
<td>&lt;3%</td>
<td>25%</td>
<td>25%</td>
</tr>
<tr>
<td>5</td>
<td>&lt;3%</td>
<td>&lt;3%</td>
<td>25%</td>
<td>25%</td>
</tr>
<tr>
<td>6</td>
<td>&lt;3%</td>
<td>&lt;3%</td>
<td>25%</td>
<td>25%</td>
</tr>
<tr>
<td>7</td>
<td>&lt;3%</td>
<td>&lt;25%</td>
<td>97%</td>
<td>97%</td>
</tr>
<tr>
<td>8</td>
<td>&lt;3%</td>
<td>&lt;3%</td>
<td>10%</td>
<td>75%</td>
</tr>
<tr>
<td>9</td>
<td>&lt;3%</td>
<td>&lt;10%</td>
<td>50%</td>
<td>50%</td>
</tr>
<tr>
<td>10</td>
<td>&lt;3%</td>
<td>&lt;10%</td>
<td>10%</td>
<td>10%</td>
</tr>
</tbody>
</table>

Abbreviations: Pt. = patient; cath = catheterization.

*From height and weight charts constructed by H. C. Stuart and associates, Harvard School of Public Health, Boston, Massachusetts.

remaining three patients (6, 9, 10), all with RV pressure at half systemic level, RV conduction delay was the only abnormal sign present. The chest X-ray showed a normal-sized heart in all but one (8) in whom the cardiothoracic index was 0.60. The pulmonary blood flow was normal, but the main pulmonary artery segment was prominent in all.

At catheterization (table 1) no left-to-right shunt could be demonstrated at ventricular level by oxygen saturation data in four patients (1, 3, 5, 6),

Figure 1

Patient #5. Levophase of right ventricular cineangiogram. Intact ventricular septum. Catheter is in right ventricular outflow position.
though in one of these (1) a trivial right-to-left shunt was suggested by a systemic saturation of 92%. A small step-up in oxygen saturation was documented in five children, with a mean Qp/Qs of 1.3/1 (1.6–1.1/1).

The pressure in the RV at the second catheterization showed a significant increase over the prebanding level in five patients (1–5); in three of these, all with muscular defects, the postbanding pressure was actually suprasystolic (1, 2, 3). In three other patients, postbanding pressures were below the preoperative level (7, 9, 10); in the remaining two (6, 8), RV pressure remained the same as at the time of the first study.

The mean gradient across the PAB was 131 mm Hg in the three patients with the suprasystolic pressure. The mean gradient in the other patients was 47.5 mm Hg (range 93–11 mm Hg).

Among the four patients with coarctation of the aorta, the aortic gradient was abolished after surgery in two (3, 6), increased in spite of surgery in one (5), and remained the same in one unoperated child (1).

The pulmonary arteriolar resistance decreased to within normal limits in all patients.

Postoperative LV cineangiogram showed no residual VSD in three patients (3, 5, 6) (fig. 1) with membranous defects. In five other patients with membranous VSDs a stream of contrast material could be demonstrated crossing the bulging aneurysm during systole (fig. 2). In one patient, with muscular VSD (2), a small jet of contrast material was shown to cross the lower muscular septum during systole. In a second child, with a single large muscular VSD (1), a funnel-shaped deformity of the ventricular septum could be demonstrated during systole, but only in diastole could radiopaque material be seen crossing the septum (figs. 3 and 4).

Four children (1, 2, 3, 6) underwent surgical repair with the division of the pulmonary artery band and repair of the main pulmonary artery by pericardial patch. Inspection of the ventricular septum, through right ventriculotomy, showed it to be intact in two patients (3, 6); in two others (1, 2) a “tiny” VSD could be identified in the muscular

**Figure 2**

Patient #4. Selective left ventricular cineangiogram. Large membranous aneurysm bulging through the ventricular septum into right ventricular cavity.
part of the ventricular septum. In patients 1 and 3 excision of large muscle bundles in RV outflow portion was necessary because of severe infundibular obstruction.

Patient 2 died on the second postoperative day with hypoxia and respiratory arrest. Autopsy revealed a small VSD (7 x 7 mm) on the left ventricular septal side, between the junction of smooth and trabeculated musculature. The defect branched behind the LV orifice in two dichotomous channels, emerging as two muscular VSDs (1 x 2 mm in size), one above and one below the septal band on RV side. The double-channeled VSD traversed in a funnel shape the ventricular septum, from LV to the septal surface of the RV, with marked fibrous proliferation of the intraluminal endocardium.

Patient 1 had an uneventful recovery and postoperative course but died unexpectedly at home three weeks postoperative with an acute pulmonary infection. Autopsy confirmed the VSD at the junction of the proximal conal septum with the right ventricular septal surface. The defect seemed large on the LV septal surface (14 x 11 mm) and was "coning" through the ventricular septum to a "fish-mouth" slit (7 x 1 mm in size), hiding behind

Figure 3

Patient #1. Selective left ventricular angiogram. At the end of ventricular systole the contracted left ventricular chamber is visualized, with a "funnel-shaped" deformity of the lower half of the ventricular septum. No contrast material is seen crossing the ventricular septum into the right ventricular cavity.
the anterior papillary muscle (fig. 5). The endocardium of the slit showed marked endocardial thickening and fibrous proliferation.

The other two children are doing well after surgical repair and are completely asymptomatic. The six remaining patients are on the waiting list for corrective surgery.

Discussion

The purpose of PAB in infants with a large VSD is to decrease the left-to-right shunt, abolish the CHF, and prevent pulmonary vascular obstructive disease. These initial, and substantial benefits have to be balanced against the disadvantages of the necessity for a second operation to close the VSD, to remove the PAB as well as to repair the main pulmonary artery. In recent years, therefore, primary closure of VSDs in infancy has been advocated.

PAB proved to be beneficial in all our patients. The clinical improvement could be documented by the spurt in development, increase in exercise tolerance, as well as disappearance of CHF.

Hemodynamic studies revealed that the defect significantly diminished in size or closed completely after PAB in ten of 55 of our patients (19%). This closing rate is substantially higher than the spontaneous closing demonstrated in cases of large VSD and CHF without PAB. The left-to-right shunt, the pulmonary artery pressure, and the pulmonary arteriolar resistance decreased, and, in fact, returned to normal in all.

The RV pressure measurements revealed a spectrum of values in the ten patients under discussion, with the level of RV pressure ranging from suprasystemic to a normal pressure. RV pressures were highest among the three with muscular defects and were in fact higher than were...

Figure 4

Figure 4. Selective left ventricular angiography in the same patient. In ventricular diastole a stream of radiopaque material is visualized across the closing small VSD.
noted during the first catheterization. In the group of seven patients with membranous VSDs, there were two with RV pressure close to systemic level, and in the rest RV pressure either stayed the same or decreased significantly.

From the anatomic point of view, we found a variety of defects including a single large muscular VSD, multiple small muscular "Swiss cheese" defects, a mixed muscular and membranous VSD, and a group of single large VSDs in *pars membranacea septi.*

The mechanism of spontaneous closure was different for the muscular and the membranous defect. The muscular ones, usually funnel shaped in profile, had a large residual orifice on the LV side and a smaller "closing" orifice on the RV septal surface, with endocardial proliferation in the lumen and muscular hypertrophy around the exit of the VSD.

The membranous defects all closed with aneurysms protruding into the RV outflow tract from the septal surface. This type of spontaneous closure has been described previously for small VSDs without previous PAB.20, 21, 22

The various explanations proposed in the literature for spontaneous closure of VSDs are presented in tables 3 and 4. Muscular defects (table 3) usually close by hypertrophy provoked by the "stress" of high velocity of flow through a restrictive type defect. In the case of membranous defects (table 4) spontaneous closure occurs mostly via endocardial structures either by aneurysm forma-
Table 3

Mechanism of Diminution and Spontaneous Closure of Muscular Septal Defects

<table>
<thead>
<tr>
<th>Defect Type</th>
<th>Description</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Relative reduction of VSD with growth</td>
<td>(French, 1918)</td>
<td></td>
</tr>
<tr>
<td>2. Apposition of the margins of the VSD</td>
<td>(Edwards, 1954)</td>
<td></td>
</tr>
<tr>
<td>3. Enlargement of VSD by muscular hypertrophy</td>
<td>(Suzuki, 1967)</td>
<td></td>
</tr>
<tr>
<td>4. Papillary muscle hypertrophy</td>
<td>(Bloomfield, 1964)</td>
<td></td>
</tr>
<tr>
<td>5. Septal hypertrophy</td>
<td>(Evans, 1960)</td>
<td></td>
</tr>
</tbody>
</table>

PAB may promote spontaneous closure of these defects. Further support for this thesis may be found in the case reports of Engle et al., Watson et al., and Jain et al., who proposed that spontaneous development of RV outflow obstruction promotes closure of large VSDs.

The main hemodynamic consequence of PAB is an input of high resistance to flow at exit of the RV, with a consequent change of high flow and high pressure left-to-right shunt to a low flow, high pressure left-to-right shunt. In such a hemodynamic situation the dp/dt of the LV during the isovolumic and early ejection period is higher than that of the RV. It is conceivable that such a change in LV contractility may increase the flow velocity across the septal defect to such a degree that proliferation and membrane formation of the endocardial layers ensues.

References

7. Muller WH Jr, Daman JS Jr: The treatment of certain congenital malformations of the heart by the creation of pulmonic stenosis to reduce pulmonary hypertension and excessive pulmonary flow. Surg Gynecol Obstet 95: 213, 1952
23. FRENCH H: Possibility of a loud congenital heart murmur disappearing when a child grows up. Guy's Hospital Gaz 32: 87, 1918
32. VAN PRAAGH R, CORWIN RD, DAHLQUIST EH, FREEDOM RM, MATTIOLI L, NEBESAR RA: Tetralogy of Fallot with severe left ventricular outflow tract obstruction due to anomalous attachment of the mitral valve to the ventricular septum. Am J Cardiol 26: 93, 1970
33. EDWARDS JE, JAMES JV, DuSHANE JW: Congenital malformation of the heart. Origin of transposed great vessels from the right ventricle associated with atresia of the left ventricular outlet, double orifice of the mitral valve and single coronary artery. Lab Invest 1: 197, 1952
35. LAVOIE R, SESTIER F, GILBERT G, CHAMAIDES L, VAN PRAAGH R, GRONDBJ P: Double outlet right ventricle with left ventricular outflow tract obstruction due to small ventricular septal defect. Am Heart J 82: 290, 1971
41. VAN PRAAGH R: Unpublished data
Diminution and Closure of Large Ventricular Septal Defects After Pulmonary Artery Banding
ZOLTAN G. MESKO, JIMMY E. JONES and ALEXANDER S. NADAS

Circulation. 1973;48:847-855
doi: 10.1161/01.CIR.48.4.847
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1973 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/48/4/847

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/