Banding of the Pulmonary Artery

Results in 111 Children

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SUMMARY
The results of banding of the pulmonary artery in 111 children with cardiac malformations associated with excessive pulmonary blood flow are presented. Thirty-three of these children died, eleven either of late band complications or after a corrective operation. Patients with ventricular septal defect represent the largest group of malformation among the 111 patients.

The overall mortality for banding in patients with isolated ventricular septal defect is 10%, as compared to 36% in patients with ventricular septal defect complicated by an associated lesion. In all infants with ventricular septal defect banded under age 3 months the mortality is 59%, as compared to a mortality of 21% in those banded after age 3 months and only 7% if banded after age 1 year. The overall mortality figures for pulmonary arterial banding have not changed appreciably since 1966; future improvement in banding mortality will depend on improved postoperative management of these infants.

Serial hemodynamic studies in patients banded under the age of 2 years have shown a fall in the pulmonary resistance toward normal, with none showing a progression of pulmonary vascular disease. In several patients thickening of the pulmonary valve occurred as a complication of banding.

Additional Indexing Words:
Ventricular septal defect Transposition of great vessels

Banding of the pulmonary artery, since its introduction more than 15 years ago, has proved to be a useful palliative operation in children with congenital cardiac malformations associated with excessive pulmonary blood flow and pulmonary hypertension due to a large ventricular communication. Many patients have since undergone pulmonary artery banding, although published reports of postoperative results have consisted of small groups of patients with either short-term assessment or incomplete hemodynamic data.

The purposes of this report are to describe:

1. the operative mortality of pulmonary banding in 111 children,
2. the hemodynamic effects of banding in patients with ventricular septal defect (VSD),
3. result of operative release of the band and closure of the VSD and,
4. the development of pulmonary valvular changes and obstruction of the right ventricular outflow tract secondary to the banding.

Materials and Methods
Observations were made on 111 children in whom banding of the pulmonary artery was performed between 1960 and 1970. In 108 patients, the pulmonary artery banding was performed at the University of Minnesota Hospitals. In three other children the operation was done at another institution. Among the 111 children, VSD was the most frequent cardiac malformation, being present in 62 (table 1). In 40 of these 62 children the VSD was present as an isolated anomaly whereas in the remaining 22,
Table 1

Distribution of Anomalies Among the 111 Patients Undergoing Pulmonary Artery Banding

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No. of patients</th>
<th>Survived band</th>
<th>Died*</th>
<th>Mortality (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Early</td>
<td>Late</td>
<td></td>
</tr>
<tr>
<td>Ventricular septal defect</td>
<td>62</td>
<td>50</td>
<td>7</td>
<td>5</td>
</tr>
<tr>
<td>Isolated</td>
<td>40</td>
<td>36</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Associated defects</td>
<td>22</td>
<td>14</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>ASD†</td>
<td>8</td>
<td>6</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>PDA†</td>
<td>5</td>
<td>4</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Coarctation</td>
<td>5</td>
<td>3</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>PDA and coarctation</td>
<td>4</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>A-P window</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Transposition of great vessels</td>
<td>16</td>
<td>7</td>
<td>7</td>
<td>2</td>
</tr>
<tr>
<td>With VSD</td>
<td>14</td>
<td>7</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>With B-H</td>
<td>8</td>
<td>6</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Without B-H</td>
<td>6</td>
<td>1</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Intact ventricular septum</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Corrected transposition of great vessels</td>
<td>6</td>
<td>3</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Single ventricle</td>
<td>11</td>
<td>9</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Tricuspid atresia</td>
<td>6</td>
<td>3</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Other malformations</td>
<td>10</td>
<td>6</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>AV canal</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Double-outlet RV</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Pulmonary stenosis‡</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Mitral atresia‡</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Truncus arteriosus</td>
<td>2</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>111</td>
<td>78</td>
<td>23</td>
<td>10</td>
</tr>
</tbody>
</table>

*Early death always occurred within 4 days of banding. Late deaths always occurred more than 1 month after banding.
†One patient had both a PDA and an ASD.
‡Banded in error.

Abbreviations: ASD = atrial septal defect; PDA = patent ductus arteriosus; A-P = aortic-pulmonary; B-H = Blalock-Hanlon; AV = atrioventricular; RV = right ventricle.

associated defects were present. In the other 49 of the 111 patients, there were 16 patients with transposition of the great vessels (TGV), 11 with single ventricle, six with corrected transposition of the great vessels and VSD, six with tricuspid atresia, three with atrioventricular canal, three with double-outlet right ventricle, two with truncus arteriosus, one with pulmonary stenosis (banded in error), and one with mitral atresia.

In each patient cardiac catheterization and angiocardiography were performed to establish the diagnosis. After a time interval varying from 1 day to more than 1 year after cardiac catheterization, the pulmonary arterial band was placed. In 22 patients additional operative procedures were performed either prior to or at the time of banding. These included ligation and division of a patent ductus arteriosus (nine patients), resection of a coarctation of the aorta (nine patients), and creation of an atrial septal defect by the Blalock-Hanlon procedure for TGV (eight patients). The pulmonary arterial banding was done by a standard procedure as previously described. All but 13 of the pulmonary bands were placed prior to age 2 years.

Results

Ventricular Septal Defect

Sixty-two patients with ventricular septal defect have undergone banding of the pulmonary artery. Of these patients, the VSD occurred as an isolated defect in 40. Among the 22 patients with VSD and coexistent cardiac malformations the additional defects were: atrial septal defect (eight), patent ductus arteriosus (five), coarctation of aorta (five), coarctation and patent ductus arteriosus (four), and aortic-pulmonary window (one).

In the total VSD group of 62 patients, 12 have died, yielding a 19% mortality rate.
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In the 22 patients banded before age 2 years, excessive pulmonary blood flow was present in each. Cardiac catheterization performed from 1 to 5 years after banding revealed reduction in magnitude of the left-to-right shunt to 50% (2:1) or less in all but three cases (fig. 1). In five of the 22 patients, a right-to-left shunt developed as a result of the band. In the one patient banded under the age of 2 years who did not show a significant fall in total pulmonary resistance (TPR), a second band was placed with beneficial effects.

Comparison of pre- and postbanding data in 22 patients banded before age 2 years. In each patient the per cent left-to-right shunt decreased, and in most this was associated with development of an outflow tract gradient. The percentage indicated on five curves represents magnitude of the right-to-left shunt which developed secondary to banding.

associated with banding. Of the 12 who died, seven died within 4 days of operation, while the other five died of medical causes from 1.5 to 26 months after the banding. No specific cause of death, other than “cardiorespiratory failure,” could be determined for the early deaths. Of the late deaths four seemed to be related to chronic congestive heart failure (CHF), and the fifth death occurred secondary to perforation of the pulmonary artery at the band site. The presence of coexistent malformations altered the mortality rate considerably; whereas among the 40 patients with isolated ventricular septal defect the mortality was only 10%, it was 36% when coexistent cardiac anomalies were present.

Thirty-two of the patients have had both pre- and postbanding cardiac catheterizations. These 32 patients have been divided into two groups depending upon whether they were banded before or after the age of 2 years.

Changes in total pulmonary resistance (TPR) with age in 22 patients banded before 2 years of age. Heavy broken line represents normal curve of TPR. Dotted extensions of lines indicate values obtained after corrective operation. TPR has tended to fall to nearly normal levels.
narrowing of the pulmonary trunk was evident following removal of the band. Three of the 24 patients (13%) died on the first postoperative day. Two of the three deaths occurred in children with multiple ventricular septal defects; the other death was associated with operatively acquired complete heart block.

Thirteen of the 21 survivors of the corrective operation have undergone postoperative cardiac catheterization at least 1 year postoperatively. In each patient, the TPR continued to fall toward normal (fig. 2). Seven of the patients were found to have small residual left-to-right shunts (fig. 5), detectable by the Freon inhalation method.8

Major residual pulmonary outflow gradients were present in three patients. In two of these, the obstruction was related to persistent narrowing of the pulmonary trunk at the band site (fig. 6). One of these patients has had another operation, with placement of a larger

In these 22 patients the TPR fell toward a normal level after banding. Although in most patients the TPR remained high, it was uncommonly greater than 750 dyne-sec-cm⁻² (fig. 2).

Among the 10 patients banded after the age of 2 years, seven had bidirectional shunts prior to operation. In only one of the six recatheterized patients has a significant outflow pressure gradient been achieved (fig. 3). In these patients the TPR remains significantly elevated, but has improved subsequent to banding (fig. 4). Progression of pulmonary vascular disease has occurred in only one patient.

Twenty-four patients have undergone operative closure of the VSD and removal of the pulmonary arterial band. In 22 of these patients the defect was closed by a transatrial approach, while in the other two the defect was closed through a ventriculotomy. In 11 cases (46%), a Teflon patch was inserted in the anterior wall of the pulmonary artery because

Changes in TPR with age in 10 patients with VSD banded after the age of 2 years. Heavy broken line represents normal curve of TPR. Dotted line indicates value obtained following corrective operation. In one patient, TPR has risen, and in six others it remains greater than 1000 dyne-sec-cm⁻².

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Patch across the band site. However, recatheterization 6 months later revealed no improvement in the degree of narrowing. The third patient (fig. 7) was found to have a 65-mm Hg gradient across the pulmonary valve (pulmonary valve index = 0.4 cm²/m²). At reoperation in this patient, commissural fusion and adherence of one cusp to the pulmonary trunk at the band site were found.

The right ventriculograms of 20 patients, performed prior to corrective operation, were available for review. Thirteen of these 20 ventriculograms revealed thickening and partial doming of the pulmonary valve (fig. 8), whereas in the other seven the valve was normal. The development of these valvular changes is related to distance between the pulmonary annulus and the pulmonary band (table 2). When this distance was greater than 15 mm there were no pulmonary valvular changes, but when it was less than 15 mm, the valve cusps appeared thick. Neither duration of the band nor magnitude of the gradient appeared to be important determinants in the genesis of the valvular alterations. The gross anatomic features of such an altered pulmonary valve are shown in figure 9.

Effect of operative closure of VSD and removal of band on magnitude of left-to-right shunt and outflow gradient in 13 patients. In three patients, a right-to-left shunt was present (per cent given in parentheses) prior to band removal. In seven patients, a small residual left-to-right shunt was demonstrated, but only by use of a sensitive indicator.

Lateral view of two right ventriculograms in patient with VSD, banded at age 10 months. (A) 4.2 years after band. Tight band is present with a pressure difference of 88 mm Hg between distal pulmonary artery and right ventricle. Note placement of band in distal pulmonary artery, 17 mm from pulmonary annulus; no valvular changes are present. (B) 1 year after corrective operation. Despite use of anterior pulmonary artery patch, severe gradient of 115 mm Hg has persisted. Note persistent posterior indentation at band site.

Figure 5

Figure 6
Complete Transposition of the Great Vessels

Pulmonary artery banding has been performed in 16 patients with TGV (Table 1). In 14 a VSD was present, while in the remaining two the ventricular septum was intact. The status of the ventricular septum was determined by cardiac catheterization and angiography. In none of the patients was a Rashkind procedure operated.

At the time of the banding operation, a Blalock-Hanlon procedure was also done in eight patients, each with a VSD. Six of these eight survived. In the other six patients with TGV and a VSD, a Blalock-Hanlon procedure was not done at the time of the banding, and only one patient survived.

The seven patients with TGV and VSD who survived banding appeared to have benefited clinically with presumed reduction in excessive pulmonary blood flow and decreased pulmonary vascular resistance. Hemodynamic data comparable to that obtained in patients with VSD as the primary cardiac defect, however, is not available. Four patients of

Table 2

Factors Influencing Development of Pulmonary Valvular Changes as Determined from Right Ventriculograms

<table>
<thead>
<tr>
<th>Distance of band from valve annulus (mm)</th>
<th>No. of cases</th>
<th>Pulmonary valve cusps</th>
<th>Duration of band (yrs.)*</th>
<th>Pressure difference across band and/or valve (mm Hg)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>8 – 15 (11.2)</td>
<td>13</td>
<td>Thickened†</td>
<td>1.0 – 6.5</td>
<td>0 – 115</td>
</tr>
<tr>
<td>17 – 26 (21.6)</td>
<td>7</td>
<td>Normal</td>
<td>1.9 – 8.0</td>
<td>0 – 100</td>
</tr>
</tbody>
</table>

Distance of band from the annulus appeared important, whereas neither the duration of the banding nor the pressure difference across the banding site appeared important.

*No significant difference between the two groups.

†As determined by angiographic appearance.
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banded by age 1 year, the mean age at banding being 10 months. Three of the six patients did not survive.

Detailed hemodynamic data is not available in the surviving patients. However, they have clinically appeared to have benefited from reduction of excessive pulmonary blood flow. No corrective surgical procedure has been attempted in this group.

Tricuspid Atresia

Six patients with tricuspid atresia and increased pulmonary blood flow have undergone banding of the pulmonary artery. Banding was performed prior to age 10 months in four, and at age 1 and 3 years in the remaining two. Three patients died after banding. The three surviving patients seem to have benefited clinically from reduction in pulmonary blood flow. No postbanding cardiac catheterizations have been performed to date.

Corrected Transposition of the Great Vessels

Banding of the pulmonary artery has been performed in six patients with corrected transposition of the great vessels, each with coexistent VSD. Five of the six patients were

these seven have undergone a Mustard procedure, and three have died. Two of the three deaths occurred late following corrective operation, and were associated with chronic pulmonary parenchymal and vascular disease even though banding was performed prior to age 4 months.

Figure 8

Typical angiographic illustration of pulmonary valvular changes occurring subsequent to band placement in the proximal main pulmonary artery. The band had been placed 3 years prior to obtaining this lateral view right ventriculogram. At the time of this catheterization at age 3.5 years, there is a combined gradient across the band site and valve of 115 mm Hg. The proximal and distal borders of the band are indicated by dotted lines. The proximal arrows indicate the valve annulus and the distal arrow indicates the point of apposition of the valve cusps. In systole, the valve cusps can strike against the narrowed pulmonary artery. Note the thickened valve cusps.

Figure 9

Photograph of pulmonary valve at autopsy in patient age 28 months who died in first postoperative day after closure of VSD and band removal. Band had been placed at age 3 months in the proximal main pulmonary artery. Catheterization prior to corrective surgery revealed a gradient of 40 mm Hg across the band and 5 mm Hg across the pulmonary valve. Looking down on the pulmonary valve, note the thickened and distorted valve cusps (arrows).
Table 3

Effect of Age and Type of Malformation upon Operative Mortality

<table>
<thead>
<tr>
<th>Age range (months)</th>
<th>No. of patients</th>
<th>No. of deaths*</th>
<th>Mortality of patients (%)</th>
<th>Band deaths (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 - 2.9</td>
<td>27</td>
<td>4/8</td>
<td>16/27</td>
<td>59</td>
</tr>
<tr>
<td>3 - 5.9</td>
<td>28</td>
<td>2/16</td>
<td>7/28</td>
<td>25</td>
</tr>
<tr>
<td>6 - 8.9</td>
<td>16</td>
<td>2/10</td>
<td>4/16</td>
<td>25</td>
</tr>
<tr>
<td>9 - 11.9</td>
<td>12</td>
<td>3/8</td>
<td>5/12</td>
<td>42</td>
</tr>
<tr>
<td>&gt;12</td>
<td>28</td>
<td>1/20</td>
<td>2/28</td>
<td>7</td>
</tr>
<tr>
<td>Total</td>
<td>111</td>
<td>12/62</td>
<td>12/33</td>
<td>34/111</td>
</tr>
</tbody>
</table>

*Numerator indicates number of deaths; denominator, total number of patients in the group.
†Includes all patients with VSD as the primary defect.

Single Ventricle

Pulmonary artery banding has been performed in 11 patients with single ventricle, seven of whom had "d-transposition" and four, "l-transposition." Among these 11, nine survived the operative procedure. Three of the nine survivors had had postoperative catheterizations at intervals of 3 months to 2 years after banding. Marked reduction in the left-to-right shunt and TPR occurred in two cases. No change in left-to-right shunt or TPR was present 2 years after banding (age 27 months) in the third patient. Although no postbanding hemodynamic data is available in these other six survivors, they all seem to have benefited clinically from reduction in pulmonary blood flow and pressure.

Other Malformations

Pulmonary artery banding has been performed in 10 other children with a variety of conditions (table 1). Two patients should not have been banded, and both died after the operation. One had a hypoplastic left ventricle; in early infancy the second patient was banded at the time of ligation and division of the PDA because of a clinical diagnosis of VSD. Subsequently he was shown to have isolated pulmonary stenosis and did not survive pulmonary valvulotomy and band removal.

Banding of the pulmonary artery has been clinically effective in the six surviving patients, all of whom had excessive pulmonary blood flow and pulmonary hypertension prior to banding. This has been verified by postbanding catheterization in the one survivor with atroventricular canal.

Influence of Age

In addition to the influence of associated defects on banding mortality, age at the time of operation is an important determinant of mortality (table 3). Considering all cardiac defects, there was a 58% mortality for banding in the first 3 months of age compared to 6% mortality after age 2 years. In patients with VSD as the primary cardiac defect, there was a 50% mortality in infants banded under 3 months of age, 15% mortality between 3 and

Table 4

Summary of Factors Related to Mortality of Banding of the Pulmonary Artery

<table>
<thead>
<tr>
<th>Possible explanation</th>
<th>Related to early death</th>
<th>Related to late death</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Rapidly deteriorating clinical condition before banding</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>2. Inadequate banding</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>3. Incorrect diagnosis; clinical status made worse by banding</td>
<td>Yes</td>
<td>Possible</td>
</tr>
<tr>
<td>4. Inadequate operative and/or postoperative management</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>5. Presence of hemodynamic situation not improved by banding</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>6. Irreversible pulmonary vascular disease</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>7. Band placed too tightly</td>
<td>No</td>
<td>No</td>
</tr>
</tbody>
</table>
12 months of age, and a 5% mortality if banded after age 1 year. In the 16 patients with VSD banded beyond the age of 2 years no patients died.

Discussion

Considerable experience with pulmonary artery banding has been gained since the original description of this procedure in 1952. Subsequently, other reports have presented the technique of pulmonary artery banding and the efficacy of banding in both decreasing excessive pulmonary blood flow and preventing the development of pulmonary vascular disease. 

Recently the operative mortality of banding of the pulmonary artery in patients with VSD has been reported. Although the mortality ranged from 5–26% in these studies, they do indicate an increased operative mortality both in infants under 3 months of age and in patients with associated cardiac malformations. The mortality figures in our series (table 1) indicate similar findings, with a 5% operative mortality in patients with isolated VSD and a 23% operative mortality in those with associated malformations. When late deaths after pulmonary arterial banding are included, the overall banding mortality becomes 10% for patients with isolated VSD and 36% in those with associated defects.

The cause of death after banding of the pulmonary artery may be related to several factors (table 4). Deaths occurring in our patients comprised two distinct groups. Each of the early deaths occurred within 4 days, and usually within 2 days, after banding and were associated with one or more of the first five factors listed. All other deaths occurred late, at least 1 month after banding. The indicated association of each factor listed in table 4 with early and/or late banding mortality is based entirely on results in our series.

Improved diagnostic methods and early, aggressive medical and operative treatment have certainly lessened the importance of the first four factors listed in table 4. Further improvement in banding mortality will require continuing refinements in postoperative management. Deaths in the present series were thought to be unrelated to the band being too restrictive, and no patient who left the operating room with a presumed adequate band was later found to have a band that was too tight. The reason for this is that a tight band is apparent at the time of placement and is corrected immediately.

The mortality associated with banding in patients with VSD has not appreciably changed since 1961. Since 1966, the banding mortality for all defects has been relatively constant, perhaps because known improvements in early, accurate diagnosis and improved surgical technique have been at least partly counterbalanced by the selection of sicker and/or younger infants for banding.

Pulmonary artery banding is an important palliative or even life-saving operation in patients with operable as well as inoperable cardiac malformation. The hemodynamic effects of banding in patients with VSD have been the reduction of both pulmonary blood flow and pulmonary vascular resistance and the creation of a significant gradient across the band. No patient banded before age 2 years has developed progressive, irreversible pulmonary vascular disease.

In patients banded after the age of 2 years, the hemodynamic benefits of banding have been variable. There have been no deaths associated with banding (10 cases). Of these ten patients, four should have had corrective surgery rather than banding, but these bandings were all performed 5–9 years ago. Three of the ten patients have successfully undergone corrective surgery, but moderate pulmonary vascular disease persists in one of these patients 1 year after corrective surgery (fig. 4).

Six of the 10 patients banded after age 2 years had bidirectional shunting and marked elevations in TPR when first evaluated and were not, therefore, candidates for corrective surgery. A significant gradient at the band site could not be achieved in any patient, but only one patient has had subsequent progression of pulmonary vascular disease. The TPR has fallen in the remaining five patients, but remains
significantly above normal values. However, the fact that significant improvement in TPR can occur suggests that banding of children with irreversible pulmonary hypertension secondary to VSD may be beneficial even though all remain inoperable. How banding will affect life expectancy in this group is, of course, unknown.

The ultimate aim in banding the pulmonary artery in patients with VSD is to prepare them for ultimate operative correction. Of the 62 patients with VSD originally banded, 50 survived. To date, 24 of these patients have undergone operation to remove the pulmonary arterial band and close the VSD. In this group there have been three deaths, yielding an operative mortality of 12.5%. The postoperative hemodynamic data has revealed continual fall in TPR. Among the patients with other forms of congenital heart disease, our hemodynamic data are too limited to draw conclusions regarding effects of banding upon the pulmonary vasculature.

Banding of the pulmonary artery is associated with changes in the pulmonary valve and the pulmonary trunk. The most frequent complication of pulmonary arterial banding found in our cases was alteration of the structure of the pulmonary valve. Such changes have consisted of thickening of the valve cusps, usually associated with valve doming, and can be correlated with proximity of the band to the pulmonary valve. No patient with distal placement of the band has developed pulmonary valvular changes even though the resultant obstruction might be severe.

The valvular changes are probably secondary to the continual trauma to the valve cusps as they strike the narrowed pulmonary artery at the band site. In many of our cases, as illustrated in figure 8, the valvular cusps can be seen to actually hit the band during each systole, resulting in thickening of the cusps and adherence of the cusps to the pulmonary arterial wall.

Hemodynamic manifestations of the acquired pulmonary valvular changes have usually been negligible, consisting of small valvular gradients of 5–10 mm Hg in almost all cases. The long-term effect of such valvular changes, however, is unknown. That this is a potentially significant problem, however, is evidenced by the fact that one of our patients evaluated after band removal did have acquired valvular stenosis of a degree sufficient to require subsequent pulmonary valvulotomy.

A second complication has been persistent postoperative narrowing of the pulmonary trunk at the site of the band. Eleven of the 24 patients with VSD who have undergone corrective surgery have required a pulmonary artery patch because the pulmonary artery remained narrowed when the band was removed. Even though a large patch has been placed anteriorly at the band site, in two of our 11 patients evaluated after corrective surgery persistent posterior narrowing at the band site has remained and caused a significant residual gradient. In the patient previously reported, operative revision of the band site was also unsuccessful due to the marked persistent posterior narrowing at the band site (also see fig. 7). Another minor acquired problem secondary to banding has been the development of mild infundibular pulmonary stenosis in three of the 11 patients evaluated after corrective surgery. This is of no apparent hemodynamic significance.

In 1966 Osborn and his coworkers reported another complication of pulmonary banding. Their patient, banded at age 5 months, developed progressive cyanosis at age 8 months and died at age 10 months. At necropsy, the pulmonary trunk proximal to the band was almost totally occluded by a thrombus. In addition, necrosis in the proximal pulmonary trunk was present. This necrosis was thought to have resulted from ischemia secondary to occlusion of the vasa vasorum by the band and to, therefore, be the underlying cause of thrombus formation. One of our patients, banded at age 6 months, came to us 26 months later with a history of recent onset of marked cyanosis. Death occurred before catheterization or emergency surgery.
could be attempted. Necropsy revealed extensive destruction of the pulmonary artery proximal to the band. Rupture of the main pulmonary artery had occurred, allowing communication with a large false aneurysm. The histologic appearance of the pulmonary trunk was that of interruption of the vessel wall at the site of banding and of organized parietal thrombosis and necrosis of the pulmonary arterial wall. There was neither clinical nor pathologic evidence of subacute bacterial endocarditis. Therefore, as in the case reported by Osborn, it appears that pressure necrosis of the pulmonary artery secondary to presence of the band may cause such complications. Since this complication occurred in only one of the 78 patients in our series who have survived banding for more than 1 year, an incidence of 1.3%, it is obvious that this complication is rare, although certainly devastating.

Spontaneous closure of the VSD after pulmonary artery banding has been reported recently in three separate case reports. Although the incidence of spontaneous closure of a small VSD in children surviving infancy may well be in the range of 25 to 30%, the incidence of closure of a large VSD must be very low. Thus, despite the theoretical possibility that this may occur, none of the 78 patients in this series who survived the first year of life has undergone spontaneous closure of his VSD.

Despite potential complications of banding, such as pulmonary valvular thickening and residual narrowing at the band site, pulmonary arterial banding represents an important palliative procedure in critically ill infants with excessive pulmonary blood flow and pulmonary hypertension. On the basis of clinical evidence, it is also apparent that pulmonary artery banding can be very beneficial in patients with inoperable cardiac defects who are symptomatic secondary to excessive pulmonary blood flow and pulmonary hypertension. As surgical techniques and postoperative management continue to improve, it is quite likely that pulmonary banding for VSD should be reserved for those patients under age 1 year, and probably under age 6 months, with primary surgical correction being performed in older infants. The high mortality in infants under age 3 months can only be improved by earlier diagnostic and surgical intervention plus improved pre- and postoperative management. Pulmonary artery banding has no place as a palliative procedure in patients older than age 2 years with uncomplicated VSD. However, in patients inoperable due to pulmonary vascular disease, banding may prevent or delay progression of pulmonary vascular changes. In our series, all the patients who developed irreversible pulmonary vascular disease had done so by the age of 2 years.

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References


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