Spontaneous closure of ventricular septal defect after banding of pulmonary artery

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Spontaneous closure of ventricular septal defect is the most beneficial culmination in the natural history of the malady. It was first recognized on clinical grounds by French in 1918 and documented with haemodynamic data by Carvalho Azevedo et al. in 1958. It is believed to occur in about 25 per cent of cases (Bloomfield, 1964). The first report of spontaneous closure of ventricular septal defect following banding of the pulmonary artery in infancy was made by Edgett et al. in 1968, 16 years after pulmonary artery banding was first performed by Muller and Dammann (1952). We report two instances of spontaneous closure of the ventricular septal defect following banding of the pulmonary artery in infancy.

**Case reports**

**Case 1** This female infant was referred at the age of 6 months in December 1962 for recurrent chest infections and a systolic murmur over the praecordium. There was nothing relevant in the family history. Physical examination showed a moderately developed child with no cyanosis or clubbing, readily palpable femoral pulses, cardiomegaly, with loud pulmonary second sound, and a pansystolic murmur at the lower left sternal edge. Chest x-ray showed cardiomegaly, dilated pulmonary arteries, and plethoric lung fields. The electrocardiogram appeared to be within normal limits. Cardiac catheterization and angiocardiography showed a large left-to-right shunt at ventricular level with a high pulmonary artery pressure (80/50 mmHg) (Table). In May 1963 the pulmonary artery was banded bringing the pressure to 35/20 mmHg, after which the child improved considerably and was regularly followed up as an outpatient.

In April 1968 when she presented for review she was well developed, with no cyanosis or clubbing. There was cardiomegaly with left parasternal heave, widely split pulmonary second sound, and systolic thrill and murmur over the left sternal edge. Electrocardiogram showed right ventricular hypertrophy. With a view to further corrective surgery she was catheterized again, when the pressure in the right ventricle was found to be 120/0 mmHg and that of the main pulmonary artery beyond the band 15/10 mmHg. Oxy-metry and angiocardiography showed no evidence of a shunt. The absence of shunt was considered to be due to balanced pressures in the ventricles or spontaneous closure of the ventricular septal defect.

In March 1970 surgical exploration was undertaken. It was found that the ventricular septal defect had closed spontaneously and debanding of the pulmonary artery was done with an uneventful recovery. She continues to be well with a grade 1-2/6 systolic murmur over the left sternal edge.

**Case 2** This baby was referred in August 1962 at the age of 2 months for failure to thrive. There was nothing relevant in the family history. Physical examination showed an undernourished child with no cyanosis or clubbing, but obviously dyspnoeic. The femoral pulses were easily palpable. There was no cardiomegaly. Heart sounds appeared normal with a pansystolic murmur at the lower left sternal edge. The liver was palpable and tender. No abnormalities were present in the other systems. Chest x-ray showed no cardiomegaly and the lung fields appeared plethoric. The electrocardiogram did not show any abnormalities. Cardiac catheterization and angiocardiography undertaken in September 1962 (Table) after adequate treatment of cardiac failure showed
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TABLE Catheterization data

<table>
<thead>
<tr>
<th>Chamber</th>
<th>Case 1 1963</th>
<th></th>
<th></th>
<th>Case 2 1962</th>
<th></th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Pressure (mmHg)</td>
<td>Oximetry</td>
<td>Pressure (mmHg)</td>
<td>Oximetry</td>
<td>Pressure (mmHg)</td>
<td>Oximetry</td>
</tr>
<tr>
<td>Superior vena cava</td>
<td>4/0</td>
<td>67%</td>
<td>4/1</td>
<td>59%</td>
<td>4/0</td>
<td>75%</td>
</tr>
<tr>
<td>Right atrium</td>
<td>80/50</td>
<td>85%</td>
<td>120/0</td>
<td>62%</td>
<td>60/10</td>
<td>82%</td>
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<tr>
<td>Right ventricle</td>
<td>5/1</td>
<td>66%</td>
<td></td>
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<tr>
<td>Main pulmonary</td>
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<td>98%</td>
<td></td>
<td>70/30</td>
<td>80%</td>
<td></td>
</tr>
<tr>
<td>artery</td>
<td>67%</td>
<td></td>
<td>59%</td>
<td>4/0</td>
<td>79%</td>
<td></td>
</tr>
<tr>
<td>Inferior vena cava</td>
<td>25/10</td>
<td>85%</td>
<td></td>
<td>15/10*</td>
<td>62%</td>
<td></td>
</tr>
<tr>
<td>Aorta</td>
<td></td>
<td>70%</td>
<td></td>
<td>65%</td>
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</tr>
</tbody>
</table>

* Beyond the band. The aortic pressure in Case 1 was measured in the ascending aorta, the catheter having been passed through the ventricular septal defect. In Case 2 the catheter passed through a persistent ductus arteriosus into the descending aorta. The left-to-right shunting at the ventricular septal defect with right-to-left shunting at the persistent ductus is common in seriously ill infants.

...get enlarged in proportion to the growth of the heart, while Weber (1918) postulated the contraction of the fibrous rim around the defect to be responsible for closure. Based on observations at necropsy (Majka, Ryan, and Bondy, 1960) and during operations (Hoffman et al., 1965; Wade and Wright, 1963), it was considered that the septal leaflet of the tricuspid valve might functionally close a high ventricular septal defect in the membranous septum. Edwards (1953) suggested that the ovoid defect in the muscular portion of the septum in the course of events might lengthen to a point where the two edges almost met and finally got closed by fibrous tissue leaving subtle pathological residue, while Evans, Rowe, and Keith (1960) suggested that septal hypertrophy might close a defect in the muscular portion of the septum. This was also borne out by observations during operations. The jetting left-to-right shunt across the defect occurring with the fall of the pulmonary artery and right ventricular pressures after birth encourages fibroblastic proliferation, intimal hyperplasia, and plaque formation (Rodbard, 1959), and closure of the ventricular septal defect by itself or by superimposition of such fibrosis on the gradual muscular encroachment of the defect (Suzuki and Lucas, 1967).

Both cases under report showed right ventricular hypertrophy after banding of the pulmonary artery, as expected. Whether this has helped the spontaneous closure of the defect cannot be decided for certain, though Hoffman et al. (1960), on the basis of observations of functional closure of muscular defect by the contraction of the hypertrophied muscle during open heart operation, suggest this is so.
Probably analysis of a larger series might help to illuminate the idea further.

Further, the right ventricular pressure is only slightly raised despite the band. The most likely explanation is that the reduction in the diameter of the pulmonary artery is causing only a slight rise in right ventricular pressure now that the systemic pressure from the left ventricle is reduced. The electrocardiographic changes and right ventricular pressure may persist for some years after relief of the cause, e.g. after successful pulmonary valvotomy, and are presumably due to the thickened ventricular wall.

References


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Br Heart J 1971 33: 854-856
doi: 10.1136/hrt.33.6.854