Surgery of ventricular septal defect and pulmonary vascular resistance

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In a series of 65 patients operated upon for ventricular septal defects (VSD), no mortality was attributed to pre-operative raised pulmonary vascular resistance. Eight patients with a severe degree of pulmonary vascular resistance made a full recovery after surgical closure of the ventricular septal defect. Three patients were cyanotic at the time of operation. Mortality in the series was the result of inadequate perfusion during operation, haemorrhage, cardiac tamponade, mediastinal infection, complete heart block or the development of post-operative aortic incompetence. Raised pulmonary vascular resistance and clinical evidence of a dominant left-to-right shunt in the absence of serious intracardiac anatomical complications does not contraindicate surgery for the repair of VSD.

CLINICAL MATERIAL

Sixty-five patients were operated upon for ventricular septal defect (VSD) in the years 1961 to 1969 in the Cardio-thoracic Department of the Royal Infirmary, Edinburgh. Cases were not included in this series if the VSD was part of a tetralogy of Fallot, a persistent common atrioventricular canal, an aneurysm of the sinus of Valsalva, pulmonary stenosis with a small and haemodynamically insignificant VSD, if associated with marked aortic valvular incompetence, if the origin of both arteries (pulmonary and aortic) was from the right ventricle, or if there was corrected transposition of the great arteries.

Patients whose VSD resulted from trauma or myocardial infarction were not included.

The diagnosis was made early in life in the majority of these patients by the presence of abnormal heart sounds, usually a systolic murmur over the left parasternal region, or the presence of other cardiac manifestations for which they were referred to the Cardiology Department for advice in establishing the diagnosis and management. Clinical examination was supplemented by radiographic examination of the chest, electrocardiograms, cardiac catheterization and angiocardiography, if necessary. These children were kept under constant observation and examination and were referred for surgical treatment when this was considered to be necessary.

After examination all cases were categorized, according to Kirklin (1965), depending on the size of the VSD; pulmonary artery pressure, pulmonary blood flow, and pulmonary vascular resistance ratios were determined (Table I).

Pulmonary arterial hypertension was considered mild when the ratio between the mean pressure in the pulmonary artery and that in a systemic artery was between 0·25 and 0·45, moderate when it was 0·45 to 0·75, and severe if higher than 0·75.

Pulmonary blood flow was considered mild if the ratio between the pulmonary and systemic

TABLE I

Group	Pulmonary Blood Flow / Systemic Blood Flow			Mean Pulmonary Blood Pressure / Systemic Arterial Pressure			Pulmonary Vascular Resistance / Systemic Vascular Resistance		
	Mild < 1·4	Moderate 1·4–1·8	Severe > 1·8	Mild < 0·25– 0·45	Moderate 0·45–0·75	Severe > 0·75	Mild < 0·25– 0·45	Moderate 0·45–0·75	Severe > 0·75
A Aged <4 yr (5 cases)			5			5	5		
B Aged 4–15 yr (50 cases)	6	18	26	12	28	10	20	24	6
C Aged 16–34 yr (10 cases) ..	1	5	4	1	6	3	2	6	2

blood flow was less than 1·4, moderate if it was between 1·4 and 1·8, and large if above 1·8.

Pulmonary vascular resistance was regarded as mild if the ratio between the total pulmonary and total systemic vascular resistance was between 0·25 and 0·45, moderate if between 0·45 and 0·75, and severe if higher than 0·75.

There were 35 males and 30 females.

According to age, the 65 patients were divided into three groups.

GROUP A Five children below the age of 8 months, each with a large ventricular septal defect, large pulmonary blood flow and mild elevation of pulmonary vascular resistance, developed intractable left ventricular failure. Banding of the main pulmonary artery as a palliative procedure, as suggested by Muller and Dammann (1952) and Goldblatt, Bernhard, Nadas, and Gross (1965), was performed. They await full repair of the VSD.

GROUP B This group comprised 50 children between 4 and 15 years of age. They were operated upon because of failure of spontaneous closure of the VSD, increasing breathlessness, repeated pulmonary infections, a history of sub-acute bacterial endocarditis (5 cases, incidence of 10%), progressive cardiac enlargement, cyanosis during exercise or exertion, failure to grow, and general deterioration.

GROUP C Ten patients aged 16 to 34 years were referred for surgery as the above symptoms were noticed during this age period.

OTHER CONGENITAL CARDIAC ABNORMALITIES PRESENT

Patent ductus arteriosus Two patients were operated upon several years previously before a repair of the VSD was attempted.

Pulmonary stenosis In four patients with hypertrophy of the crista supraventricularis, a wedge of muscle was excised during repair of the VSD. In one case the pulmonary valve was stenosed; this required dilatation by a dilator.

Presence of left superior vena cava In four cases this drained into the right atrium; in two cases into the coronary sinus. No surgery for this anomalous vein was required.

Atrial septal defect One case was discovered by pre-operative catheterization; the other patient became desaturated after the VSD had been repaired. A large atrial septal defect was discovered and was repaired.

SURGICAL TREATMENT Repair of the VSD was done using extracorporeal cardiopulmonary by-pass. Through a mid-line sternal-splitting incision the pericardium was opened vertically. A search was made for patent ductus arteriosus, left superior vena cava and anomalous pulmonary venous connections. The femoral artery was used before 1964 and the ascending aorta after this date for arterial perfusion during operation which was maintained by a Melrose disc oxygenator. The superior and inferior venae cavae were cannulated through the right atrium for venous drainage. A Rygg pump oxygenator with disposable bag was used after 1964. Closure was achieved using interrupted silk sutures in cases where the VSD was less than 1·5 cm. in diameter (34 cases). In 25 cases a Dacron patch was used where the VSD diameter was found to be more than 2 cm. in diameter. In one patient a pericardial patch was used. In cases of multiple ventricular septal defects (Swiss cheese septum) repair was performed using interrupted silk sutures. Recently, interrupted silk mattress sutures passing behind these defects and tied over the surface of the heart on a piece of Teflon have been used. When these sutures are tied, the defects close.

Post-operatively respiratory assistance, using an endotracheal tube and a Bird respirator, was required for about 15 hours. In patients with a high pulmonary resistance, respiratory assistance was required for longer periods of up to three days.

RESULTS

GROUP A Five children with banding of the pulmonary artery are clinically improved and awaiting full correction.

GROUP B Fifty children aged between 4 and 15 years all survived, except three children who died from the following causes.

A child of 14 years had a defect 2 cm. in diameter with moderate pulmonary vascular pressure and moderate vascular resistance. Death was due to mediastinal infection. A child of 7 years with moderate pulmonary vascular pressure and moderate vascular resistance developed cardiac tamponade and cardiac arrest following evacuation of the haematoma. A child of 12 years with moderate pulmonary vascular pressure and mild vascular resistance after closure of the VSD

TABLE II
PRE-OPERATIVE HAEMODYNAMICS

Age and Sex		Indication for Operation	QP/QS	PP/PS	RP/RS	Post-operative Progress
20	M	VSD diagnosed age 6 yr. Increased breathlessness on exertion	1·8	0·5 (moderate)	0·5 (moderate)	No clinical evidence of increased pulmonary vascular resistance. Asymptomatic for 8 yr following closure of VSD
22	F	VSD noticed age 5 yr. Complained of tiredness, dyspnoea on exertion, unable to do housework. History of subacute bacterial endocarditis	2	0·9 (severe)	0·19 (severe)[1]	Satisfactory progress since closure of VSD in 1965. Symptom markedly improved
20	M	VSD noticed age 8 yr. Breathlessness on exertion for one year	4·4	0·8 (severe)	0·45 (moderate)	Asymptomatic. No clinical evidenc of increased pulmonary vascular resistance since closure of VSD in 1965
17	M	VSD discovered age 3 yr. Attack of subacute bacterial endocarditis	1·8	0·43 (mild)	0·4 (mild)	Satisfactory since closure of VSD in 1965. Asymptomatic
18	M	VSD discovered age 5 yr. Repeated attacks of subacute bacterial endocarditis	1·6	0·5 (moderate)	0·46 (moderate)	Full activity since closure of VSD in 1965
34	M	VSD discovered age 1 yr. Clinical evidence of reverse of shunt: L–R to R–L across VSD	2	1·3 (severe)	1·1 (severe)[1]	Asymptomatic. No clinical evidence of pulmonary vascular resistance since closure of VSD in 1965. Repeated catheterization in 1969 showed PP/PS: 0·4; RP/RS: 0 4
26	F	Dyspnoea on exertion. Occasional cyanosis. Patent foramen ovale discovered during catheterization	1·4	0·48 (moderate)	0·4 (mild)	No symptoms since closure of VSD and patent foramen ovale in 1966
24	F	VSD discovered following investigation for tiredness and breathlessness	2·5	0·8 (severe)	0·5 (moderate)	Satisfactory progress. Normal activity since 1969 when VSD was closed

QP=pulmonary blood flow; QS=systemic blood flow; PP=mean arterial pulmonary blood pressure; PS=systemic blood pressure; RP=pulmonary vascular resistance; RS=systemic vascular resistance.
[1] Total pulmonary vascular resistance was 9 units or more and pulmonary arteriolar resistance 6 units or more.

developed a 2:1 heart block; a pacemaker was used but the condition gradually deteriorated with clinical evidence suggesting induced post-operative aortic incompetence. Death occurred two months later during re-operation for aortic incompetence.

GROUP C (10 cases) Two patients died. One, an adult aged 21, with moderate pulmonary blood pressure and moderate pulmonary vascular resistance, had closure of a VSD, 2·5 cm. in diameter, by a Dacron patch. He recovered from operation but developed cardiac tamponade and died during evacuation of the haematoma. The other, an adult aged 26 with a 2-cm. VSD and moderate pulmonary pressure and resistance, died due to cerebral anoxia, probably the result of inadequate perfusion or air embolism. He did not recover consciousness after operation. The other eight patients recovered satisfactorily (Table II).

DISCUSSION

Elevation of pulmonary vascular resistance in patients with ventricular septal defect is a reflection of anatomical changes in the small vessels of the lungs, e.g., endothelial proliferation and accumulation of connective tissue within the intima, increase of elastic tissue in the muscle fibres, muscular hypertrophy and hyperplasia of pulmonary arteries; in severe cases fibroid changes or necrosis of the wall of the arteries may occur (Heath and Edwards, 1958; Wagenvoort, Neufeld, DuShane, and Edwards, 1961; Sandoe, 1963; Fontana and Edwards, 1962).

Similar changes were noticed on pathological examination of the pulmonary vessels in patients who died (Heath, Helmholz, Burchell, DuShane, Kirklin, and Edwards, 1958; Hoffman, 1968).

Cartmill, DuShane, McGoon, and Kirklin (1966) report the hazards of operation when the pulmonary systemic resistance ratio is greater than 0·75.

Our study suggests that, although severe pulmonary resistance before the age of 15 years (6 cases out of 50) and even after 15 years (2 cases) may make the operation risky, careful surgical technique and post-operative respiratory assistance reduce this risk.

One patient aged 34 years with a severe degree of pulmonary hypertension and pulmonary resistance, proved by pre-operative catheterization and at operation, was found four years after closure to have no clinical evidence of pulmonary vascular disease. The pulmonary vascular resistance and the pulmonary blood pressure were normal.

It is difficult to explain how the pulmonary blood pressure and pulmonary vascular resistance return to a normal level after repair in childhood

or adolescence. This may be explained by the elasticity of the pulmonary vessels. In adults, pulmonary vessels, probably closed due to vasospasm before repair of the VSD as a defensive mechanism, may re-open after repair. There was no late mortality nor physical signs indicating progression of pulmonary vascular disease attributed to closure of the VSD.

The assessment of pulmonary vascular disease is essentially a histological one (Edwards, 1957; Heath and Edwards, 1958). No single clinical or haemodynamic findings can be used to assess pulmonary vascular disease (Hallidie-Smith, Hollman, Cleland, Bentall, and Goodwin, 1969). The pulmonary vascular resistance may even increase under hypoxic conditions (Vogel, McNamara, and Blount, 1967) and full oxygenation is required during assessment.

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