Surgical treatment of ventricular septal defect in infancy

Primary repair versus banding of pulmonary artery and later repair¹

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SUMMARY Results of primary closure of ventricular septal defects are compared with those of two-stage repair, with banding of the pulmonary artery followed by debanding and closure.

Apart from the high incidence of unsatisfactory results after banding and a significant morbidity with the two-stage approach, the mortality for primary repair (2.4%) is considerably lower than that achieved with the staged repair (19.3%). Primary repair of ventricular septal defect is advocated for infants resistant to maximal medical treatment. A more flexible policy is adopted for patients with multiple ventricular septal defects and those with associated anomalies.

Intractable congestive heart failure, failure to thrive, and repeated chest infections are the main indications for surgical treatment of ventricular septal defect with a large left-to-right shunt during the first year of life. Raised pulmonary vascular resistance alone is rarely an indication for surgical treatment of ventricular septal defect before the age of 1 year. The increasing number of encouraging reports on the results of primary closure of ventricular septal defect in infancy (de Leval *et al.*, 1974; Johnson *et al.*, 1974; Barratt-Boyes *et al.*, 1976; Kirklin *et al.*, 1976; Subramanian, 1976; Rein *et al.*, 1977; Sigmann *et al.*, 1977) is certainly in favour of the one-stage rather than two-stage repair (pulmonary artery banding, followed by closure of ventricular septal defect and debanding at a second stage). It is, however, of some concern that comparable data from the two management programmes are not available. A randomised trial of both approaches from the outset would have been ideal, but the initial reports of primary closure have been so favourable that many would regard a trial at this stage as unjustifiable. Because of differences in

patient material and surgical practice, comparison of the two approaches from different centres, or from one centre during different eras, is of limited value. In an attempt to make a more valid comparison we have reviewed our experience at The Hospital for Sick Children, Great Ormond Street, London, with these two approaches during the same period, January 1971 to December 1976.

Clinical material

In the 6-year period, 1971 to 1976, 24 infants (i.e. children less than 12 months of age) with ventricular septal defect as their dominant lesion have undergone pulmonary artery banding (group 1). Their ages at time of operation ranged from 6 days to 7 months (mean 3.0 months), and their weights from 2.0 to 6.1 kg (mean 3.8 kg) (Fig. 1 and 2).

During the same period, closure of ventricular septal defect was undertaken in 41 infants aged 1 to 11 months (mean 6.2 months), weighing 2.5 to 7.5 kg (mean 4.8 kg) (Fig. 1 and 2) (group 2). The cardiac lesions in the two groups are listed in Table 1. Under single high ventricular septal defect, we have included ventricular septal defects in the region of the membranous septum and/or the infundibular septum. Several patients in that group had an atrial septal defect and/or a persistent ductus

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Fig. 1 *Banding versus primary repair. Age distribution and hospital mortality.*

arteriosus in addition. Under ventricular septal defect and coarctation of the aorta, we have included patients with single (4 patients) or multiple septal defects (3 patients). All these patients were submitted to an identical intensive medical regimen, consisting of full digitalisation, diuretics, nasogastric tube feeding in many cases, and assisted ventilation in some. Antibiotic therapy was used only when an infection was present. In the infants with a tight coarctation, this was resected as an emergency before the trial period of medical treatment. Preoperative investigations in all patients included a full cardiac catheterisation with selective angiocardiography. This included a left ventriculogram with the patient positioned to profile the interventricular septum in order to determine the number and location of the

Fig. 2 *Banding versus primary repair. Weight distribution and hospital mortality.*

Table 1 *Primary operation in infants with ventricular septal defect: banding or primary closure*

Lesion	Banding		Primary closure	
	No.	%	No.	%
Single high ventricular septal defect	15	62.5	39	95
Multiple ventricular septal defects	2	8.3	2	5
Ventricular septal defect + coarctation	7	29.2		
Total	24	100	41	100

ventricular septal defects. All the patients had a pulmonary/systemic flow ratio greater than 2.5:1. The decision to band or to proceed with intra-cardiac repair was not randomised. In 6 infants early in this series, closure of the ventricular septal defect was performed after failure of banding to alleviate cardiac failure. Later, some patients who required urgent operation underwent pulmonary artery banding, as our limited facilities precluded open-heart repair at short notice. For patients with coarctation of the aorta, banding was carried out at the time of operation for correction of coarctation when the left-to-right shunt at ventricular level was considered to be a major component in inducing the heart failure.

In order to assess the cumulative morbidity and mortality of a two-stage repair, we have also reviewed our results of intracardiac repair after previous banding (group 3). The repair in this group of patients was performed during the same period, 1971 to 1976. There were 58 patients aged between 2 and 9 years (mean 5.4 years). All had been banded in infancy (10 days to 10 months) (mean 5.2 months). There were 52 single ventricular septal defects and 6 multiple ventricular septal defects (Table 2). Patients with a failed banding who also underwent closure during infancy are not included in this group.

SURGICAL TECHNIQUES AND RESULTS IN 3 GROUPS

(1) Pulmonary artery banding

Our technique of banding the pulmonary artery has

Table 2 *Closure of ventricular septal defect + debanding*

Lesion	Patients	Mortality	
		No.	%
Single high ventricular septal defect	47	2	4.2
Midseptal ventricular septal defect	2	0	0
Apical ventricular septal defect	3	0	0
Multiple ventricular septal defects	6	2	33.3
Total	58	4	6.8

been described previously (Stark *et al.*, 1969). There were 2 operative deaths in this group (mortality rate of 8.3%), one in a 1-month-old infant and the other in a 6-day-old baby; the latter patient also had a coarctation of the aorta which was resected. Pulmonary artery banding was not effective in controlling congestive heart failure in 8 patients (33.3%). These patients remained in congestive heart failure despite a diminution in the pulmonary artery pressure, often after a short period of improvement. One patient died 10 weeks after operation in heart failure. In addition, he had a tracheo-oesophageal fistula repaired and a ventriculoatrial shunt constructed for hydrocephalus. One infant was banded again successfully and the other 6 patients underwent closure of the ventricular septal defect and debanding of the pulmonary artery. These 6 patients are also included in the group who underwent closure of ventricular septal defect in infancy. The mortality rate for the first group is, therefore, 3 patients out of 24 (12.5%).

(2) Closure of ventricular septal defect in infancy

Forty-one infants underwent closure of ventricular septal defect in infancy between 1971 and 1976. Conventional cardiopulmonary bypass with moderate hypothermia was used in 30 patients (73%). The technique of deep hypothermia and total circulatory arrest was used in 11 patients (27%). The surgical approach is summarised in Table 3, which indicates our preference for closure of ventricular septal defect through the right atrium. The transventricular approach was used mainly in the beginning of the series before we had acquired more experience with the transatrial closure. At the present time, the majority of ventricular septal defects are closed through the right atrium. The defects were closed with a prosthetic patch in 37 patients and by direct suture in 4 patients. When the defect was closed through the right atrium, the septal and anterior leaflets of the tricuspid valve were retracted but not detached. Interrupted or running sutures were employed. An apical left ventriculotomy was used in 2 patients with a defect in the lower part of the

muscular septum (Singh *et al.*, 1977). The ventricular septal defect was closed through a short right ventriculotomy in 14 patients. When a persistent ductus arteriosus was present, this was dissected intrapericardially and doubly ligated before cannulating the ascending aorta. The ductus arteriosus was routinely dissected in all patients whose operations were done under deep hypothermia and circulatory arrest when there was pulmonary hypertension, even if the investigation had failed to demonstrate its patency. In these patients, we think that an aortogram does not reliably rule out a small persistent ductus arteriosus that would be a source of air embolus when the circulation is arrested and the right heart opened. All defects in the atrial septum, no matter how small, were routinely closed to prevent any right-to-left shunts in the postoperative period. The postoperative complications are listed in Table 4. Arrhythmias in the immediate postoperative period were relatively frequent (19%) but were all temporary. One patient required reoperation for wound infection and another patient for a residual ventricular septal defect. The latter patient did well after the second closure of the ventricular septal defect; one suture had cut through the tricuspid valve and the patient had a large shunt from the left ventricle to the right atrium. A cardiac arrest occurred 36 hours after operation in another patient. After resuscitation he had convulsions and his subsequent development indicates major cerebral damage. This patient has also remained in congestive heart failure. A repeat cardiac catheterisation showed a significant shunt between the left ventricle and the right atrium. A second patient had convulsions, but these ceased and there was no evidence of permanent brain damage.

There was 1 hospital death (2.4%) and 1 late death. The hospital death occurred in an 11-month-old infant who sustained a sudden cardiac arrest 24 hours after operation. The ventricular septal defect was securely closed and the reason for his sudden death remains unexplained. The late death was not related to the heart defect. It occurred in a child

Table 3 Closure of ventricular septal defect in infancy — surgical approach

Surgical approach	No.
Right atriotomy	27
Right ventriculotomy	14
Left ventriculotomy	2
Total	41*

*2 patients required 2 incisions (left ventriculotomy and right atriotomy) for closure of multiple defects.

Table 4 Closure of ventricular septal defect in infancy — postoperative complications

Postoperative complications	No.
Supraventricular tachycardia	2
Nodal rhythm	1
1st degree AV block	1
2nd degree AV block	2
3rd degree AV block	2
Convulsions	2
Residual ventricular septal defect	2
Wound infection	2

Table 5 Closure of ventricular septal defect and debanding — surgical approach

Surgical approach	No.
Right atriotomy	36
Right ventriculotomy	22
Apical left ventriculotomy	3
Total	58*

*3 patients required 2 incisions for closure of all ventricular septal defects.

with thoracic dystrophy who died from progressive chronic respiratory failure one year after operation. Apart from this late death, and the patient who has serious brain damage, all other patients are making satisfactory progress. In 4 patients there is a persistent systolic murmur without any sign of heart failure. The nature of the murmur has not yet been determined by repeat cardiac catheterisation.

(3) Intracardiac repair and debanding of pulmonary artery

Morbidity related to previous banding of the pulmonary artery was significant. Twenty-seven patients (46.5%) had a decreasing exercise tolerance and developed cyanosis before debanding and closure of the ventricular septal defect. All patients had cardiac catheterisation and angiocardiography before operation. Six (10.3%) had a raised pulmonary vascular resistance ($R_p/R_s \geq 0.5$).

The surgical repair was performed using cardiopulmonary bypass with moderate hypothermia and intermittent cross-clamping of the aorta. The surgical approach is shown in Table 5. In 3 patients with multiple ventricular septal defects, 2 incisions were made to close all the defects. In 8 patients the defect was closed by direct suture. A prosthetic patch was used for the other patients. There were 4 hospital deaths (6.8%). The first patient was 5 years old, and the pulmonary artery had been banded at 3 months of age for a single mid-septal ventricular septal defect. The band had migrated beyond the bifurcation of the pulmonary artery. The origin of the right pulmonary artery was completely occluded and the left pulmonary artery was hypoplastic. A satisfactory reconstruction of the pulmonary arteries was not achieved at the time of operation and the patient died 8 days after operation. The necropsy confirmed that the right pulmonary artery remained occluded and histological examination showed pulmonary vascular changes in the left lung, compatible with a moderate degree of pulmonary vascular disease. The second patient had a single high ventricular septal defect and a left superior vena cava draining into the coronary sinus. Banding had been performed at 5 months of age, and the

ventricular septal defect was closed and the pulmonary artery debanded at the age of 3 years. An unroofed coronary sinus (left superior vena cava draining to the coronary sinus and the left atrium) was not recognised and she became severely hypoxic after operation. At reoperation the following day, the left superior vena caval blood was redirected to the right atrium, but the patient died 24 hours later in a low cardiac output state. The other patients who were 3 years and 6 years of age had multiple ventricular septal defects and died after operation in a low cardiac output state. The younger patient had required reconstruction of both pulmonary arteries to relieve the band which had migrated beyond the bifurcation.

Patch enlargement of the main pulmonary artery was used in 47 patients (81%). Excision of the banded segment with end-to-end anastomosis was used in 6 patients; this is at present our technique of choice. Migration of the band beyond the bifurcation required extensive reconstruction of the pulmonary arteries in 5 patients, including the use of a 'dacron' tube from the main pulmonary artery to left or right pulmonary artery in 3 patients. Infundibular resection of the right ventricle was necessary in 11 patients (18.9%), and a pulmonary valvotomy in 2 (3.4%). A gradient between the right ventricle and the pulmonary artery exceeding 20 mmHg remained in 12 patients (20.6%).

Discussion

As for most operations on the heart, the determination of the ideal time for surgical repair of ventricular septal defect depends on: (1) the operative risk in relation to age; (2) the quality of the repair; (3) the preoperative detrimental effects of the disease on the myocardium and pulmonary vasculature and their reversibility; and (4) to some extent, on the psychosocial impact of the disease and its treatment on the child and its family.

Residual abnormalities in left heart size and function have been documented after closure of ventricular septal defect in older children (Jarmakani *et al.*, 1971) and seem to be reversible if the defects are closed in younger patients (less than 2 years of age (Cordell *et al.*, 1976). The same applies for pulmonary vascular changes which are reversible if the patients are operated upon when less than 2 years of age (DuShane and Kirklin, 1973). It can be argued in the light of several series of primary closure of isolated single high ventricular septal defect in infancy (Johnson *et al.*, 1974; Barratt-Boyes *et al.*, 1976; Kirklin *et al.*, 1976; Subramanian, 1976; Rein *et al.*, 1977; Sigmann *et al.*, 1977) that, if the operative mortality and quality of surgical

repair are the same in infancy as beyond infancy, all large ventricular septal defects should be closed in infancy. However, this argument takes no account of the occurrence of spontaneous closure or diminution in size of the defect. Accordingly, we still believe that elective closure of these defects should not be undertaken before the age of 1 year unless clearly indicated by: (a) the persistence of congestive heart failure and its consequences in spite of a fair trial of medical treatment or, (b) the less common rising pulmonary vascular resistance in that age group.

Blackstone and his collaborators (1976) have recently attempted to bring together all these factors into an elegant equation which sums these possibilities. Their results show that cure after successful primary repair of a single large ventricular septal defect is more likely in the younger patient with a lower preoperative pulmonary vascular resistance and pulmonary artery pressure. The authors define surgical cure as survival for 5 years, with a mean pulmonary artery pressure at that time less than 25 mmHg. Their conclusion is that the optimal age for elective repair is 18 months. Since this figure is based on a higher surgical mortality in infancy than reported here, we might, therefore, conclude that the optimal age for closure is somewhat less.

Our policy for patients with single ventricular septal defect associated with patent atrial septum (excluding atrioventricular defects) or a persistent ductus arteriosus is the same as the one outlined for isolated single ventricular septal defect. For patients with multiple ventricular septal defects the mortality is still high whatever the policy adopted (Blackstone *et al.*, 1976). Whether these patients should undergo a primary closure or banding of the pulmonary artery if they require surgical treatment during infancy remains controversial. We now would consider primary closure of these defects except for the truly multiple small defects of the 'Swiss-cheese type', for which we still prefer banding of the pulmonary artery in infancy and an intracardiac repair at about 2 years of age.

When ventricular septal defect is complicated by coarctation of the aorta, our present policy is to resect the coarctation at the preliminary operation without banding the pulmonary artery, unless the infant is critically ill and deteriorates during resection of the coarctation. In this case, pulmonary artery banding would also be performed.

The difference in the mortality rate between the one-stage and two-stage approaches is considerable. The risk of pulmonary artery banding was less (12%) in this series than in previous years (Stark *et al.*, 1969). More recent reports from other

institutions show a hospital mortality of 10 to 20 per cent (Patel *et al.*, 1973; Dooley *et al.*, 1975). If one assumes that the risk of debanding and closure of ventricular septal defect would be the same in our group 1 patients who survived pulmonary artery banding as in the group 3 patients, one could expect an overall mortality rate with the two-stage approach of 19.3 per cent. This is considerably higher than the operative risk of early primary closure, which was 2.4 per cent in the present series.

This consideration does not take into account the late morbidity and late mortality which remains to be assessed. Our experience so far does not suggest that late complications are more common in patients who had early correction. In addition, pulmonary artery banding may not be effective, as shown by our experience with 7 infants who survived but remained in congestive heart failure. The failure of pulmonary artery banding probably results from the difficulty in evaluating the haemodynamic effect of the band at the time of operation (Utley, 1973) and from changes in pulmonary vascular resistance after banding. If we add the patient in whom increased pulmonary vascular resistance was present at the time of intracardiac repair ($R_p/R_s > 0.5$) to the infants with congestive heart failure after pulmonary artery banding, the failure rate was 43.6 per cent.

Our groups 1 and 2 are not entirely comparable: infants who develop intractable cardiac failure during the first 3 months of age often have more complex anomalies than those with isolated ventricular septal defect(s) and therefore are at high risk. However, if after treatment of the associated lesion(s) they remain in severe congestive heart failure related to the presence of ventricular septal defect(s), we believe our results justify a policy of primary closure rather than banding of the pulmonary artery, and that the age or weight of the patient should not contraindicate or increase the risk of primary closure.

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