SURGERY FOR CONGENITAL HEART DISEASE

TECHNIQUES AND RESULTS IN THE MANAGEMENT OF MULTIPLE VENTRICULAR SEPTAL DEFECTS

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Objective: The management of patients with multiple ventricular septal defects remains controversial. Primary closure, interventional catheter techniques, and palliative surgery all may have a role, and specific management guidelines remain undefined. **Methods:** We reviewed the records of all 33 patients with multiple ventricular septal defects undergoing repair between January 1988 and October 1996. Pulmonary artery hypertension was present in 21 patients (group 1), and pulmonary stenosis was present in the remaining 12 (group 2). Closure was accomplished from a right atriotomy alone in most patients, although an apical left ventriculotomy was used for apical defects. Among group 1 patients, the mean age at repair was 5.9 ± 0.9 months. Major associated anomalies included coarctation ($n = 6$), straddling tricuspid valve ($n = 1$), and critical aortic stenosis ($n = 1$). Reoperation was performed in two patients for residual ventricular septal defects. Among group 2 patients, the mean age at repair was 6.6 ± 3.2 years. Major associated anomalies included tetralogy of Fallot ($n = 2$), pulmonary stenosis ($n = 4$), double-outlet right ventricle with hypoplastic left ventricle ($n = 1$), and isolated left ventricular hypoplasia ($n = 1$). Three required reoperation for residual ventricular septal defect. **Results:** There were no early or late deaths, no episodes of heart block, and no significant residual ventricular septal defects among group 1 patients. All group 1 patients remain free of significant residual cardiovascular conditions at a mean of 23.4 ± 5.1 months. Among group 2 patients, there was one early death in a patient with double-outlet right ventricle and left ventricular hypoplasia. Complete heart block occurred in two patients and one required late mitral valve replacement. There were no late deaths, seven remain alive without significant residual defects at a mean of 36.2 ± 8.0 months, and two required transplantation for left ventricular failure. **Conclusions:** Primary repair for infants with multiple ventricular septal defects is associated with good late outcomes. The right atrial approach is satisfactory for most muscular defects, although limited apical left ventriculotomy was used for apical defects. Pulmonary artery banding should be limited to patients with complex associated defects. (J Thorac Cardiovasc Surg 1998;115:848-56)

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The optimal management of patients with multiple ventricular septal defects (VSDs) remains controversial. Primary closure, interventional catheter techniques, and pulmonary artery banding all may have a role, and specific management guidelines remain undefined.¹⁻⁴ Despite advances in diagnosis and surgical treatment, the morbidity and mortality in patients with multiple VSDs has remained high, primarily resulting from difficulties associated with inadequate exposure and incomplete closure.⁵⁻⁷ Visualization of the VSDs from the right side of the septum often leads to uncertainties in defining the true edges of the VSDs because of the presence of numerous muscular trabeculations. These defects, however, when viewed from the left side of the septum, will generally appear as a discrete, often single, defect with easily discernible margins. The use of an apical left ventriculotomy to improve exposure has led to better visualization and has facilitated more accurate repair.⁸ Although this has been a satisfactory approach without important late sequelae in some series, including our own,^{9,10} others have reported a significant risk of myocardial dysfunction after left ventriculotomy in addition to late apical aneurysm formation.¹¹ In an effort to further define patient and procedural risk factors associated with the management of patients with multiple VSDs, and to describe the surgical techniques used, this report reviews the experience with the management of multiple muscular VSDs since 1988 at The University of Michigan Medical Center.

Patients and methods

Between January 1988 and October 1996, 33 patients underwent surgical repair of multiple VSDs at C. S. Mott Children's Hospital, University of Michigan Medical Center. Pulmonary artery hypertension was present in 21 patients (group 1). In the remaining 12 patients (group 2), the pulmonary vascular bed was protected either by naturally occurring or surgically created pulmonary stenosis. Anatomic definitions of muscular VSDs were based on the classification described by Kirklin and associates.¹² Indications for operation were congestive heart failure or failure to thrive (or both) in all of the group 1 patients. Cyanosis caused by a right-to-left shunt was the indication for operation in most of the group 2 patients. In all patients, preoperative diagnosis was made by two-dimensional echocardiography and Doppler color flow mapping. Cardiac catheterization was performed in 28 patients. In five infants, the anatomy and hemodynamics were well defined by Doppler echocardiography in addition to the clinical presentation, and cardiac catheterization was not considered to be necessary. Twenty-five patients had major associated congenital cardiac conditions (Table I). Five patients had undergone pulmonary artery banding

Table I. Associated cardiovascular conditions in 25 patients*

Associated condition	Group 1 (No.)	Group 2 (No.)
Patent ductus arteriosus	5	3
Atrial septal defect	7	5
Coarctation	6	0
Tetralogy of Fallot	0	2
Pulmonic stenosis	0	2
Critical aortic stenosis	1	0
Straddling tricuspid valve	1	0
L-Transposition	0	1

*Twenty-one patients had more than one associated condition.

procedures at outside institutions. A total of 38 procedures were performed to close the VSDs, including reoperation in five patients. The surgical approach was a right atriotomy alone in 26 procedures, combined right atriotomy and apical left ventriculotomy in 11, and right ventriculotomy alone in 1 procedure. All patients underwent complete two-dimensional Doppler echocardiography before discharge to assess the adequacy of the cardiac repair and to examine for residual VSDs.

Hypothermic cardiopulmonary bypass (25° to 28° C) was used in 27 patients with cannulation of the ascending aorta and bicaval venous return. In the six patients with coarctation of the aorta, a single cannula was placed in the right atrium and the procedure was performed during a single period of deep hypothermia and circulatory arrest for repair of both the coarctation and the VSDs. Primary resection of the coarctation and end-to-end anastomosis was performed before closure of VSDs. The surgical approach was based on the localization of VSDs, which was assessed before and during the operations. The ventricular septum was examined through both the tricuspid and mitral valves (via the atrial septum) to facilitate accurate identification of the true edges of the muscular VSDs. Transatrial closure of the VSDs was performed if possible.

Surgical approaches for muscular defects

Right atrial approach with division of septal and/or moderator bands (Fig. 1). VSD closure was accomplished either exclusively ($n = 26$ procedures) or in combination with an additional approach ($n = 11$ procedures) through the right atrium. This exposure was sufficient to close all muscular VSDs, regardless of location, with the exception of 11 of the 12 apical defects and one anterior muscular defect. The latter was approached through a right ventriculotomy, the only such ventriculotomy in this series. The coarse multiple trabeculations on the right ventricular side of the septum, including the moderator band and lower end of the septal band, were divided to facilitate accurate exposure of midmuscular and anterior VSDs as necessary. This technique effectively allowed the VSDs to appear like a single defect. Suturing in the area away from the conduction tissue was performed by placing the patch on the left ventricular side of the VSD for more secure closure.

Right atrial approach with oversized patch (Figs. 2 and 3). In some patients, the division of right ventricular

Fig. 1. Appearance of midmuscular and anterior muscular VSDs as viewed through the tricuspid valve. Division of muscle trabeculations, including the septal and moderator bands, is used to facilitate exposure and delineate the edges of the defects.

muscle trabeculations required to adequately expose the defects proved to be too extensive. A technique of inserting an "oversized" patch from the right side of the septum was used in two of the patients in this series. This technique involved dividing only enough of the right ventricular muscle trabeculations to provide access to the left side of the septum. A patch of polytetrafluoroethylene is cut slightly larger than the total VSD area and a series of sutures is placed through the patch before it is positioned in the heart. The patch, with the attached sutures, is then pushed through the center of the VSDs into the left ventricle and the sutures are brought back across the septum from left to right. Relatively few sutures are required because the patch is held in position by the higher left ventricular pressures.

Apical left ventriculotomy (Fig. 4). When the VSD is located in the apical portion of the septum, a short "fish-mouthed" incision is made in the apex of the left ventricle away from and parallel to the anterior and posterior descending coronary arteries. Care is taken to avoid damaging the anterior papillary muscle of the mitral valve. This incision can be considerably smaller than the VSD itself because the exposure afforded is excellent and the margins of the defect are immediately apparent along the left side of the VSD. The apex of the VSD is the ventriculotomy itself. To close the left ventriculotomy, a double layer of running polypropylene is placed to incorporate the patch used to close the VSDs. This approach was used in 11 patients. In the additional patient with an apical VSD, closure was accomplished entirely from a

Fig. 2. Oversized patch technique used to close multiple muscular VSDs. A patch is cut slightly larger than the total area of septal defects. Right ventricular muscle trabeculations are divided to expose enough of the defects to allow insertion of the patch into the left ventricle. A series of mattress sutures are placed through the patch before it is inserted. The patch and the sutures are then placed across the septum into the left ventricle (*inset*). *RV*, Right ventricle; *LV*, left ventricle.

right atriotomy by placing a series of mattress sutures supported by felt buttresses from the outside of the apex of the heart into a prosthetic patch that was used to close the apical defect in addition to multiple anterior muscular defects.

Sandwich technique (Figs. 5 and 6). The sandwich technique was used in two patients for closure of multiple small anterior muscular VSDs by transfixing the muscular edge of the defects to the anterior free wall of the ventricle. Mattress sutures are placed through the septal muscle, continued through the anterior wall of the ventricle away from the left anterior descending coronary artery, and finally through a felt buttress to effectively close the defects by "sandwiching" them against the anterior wall of the heart.

Results

In the 21 patients with pulmonary artery hypertension (group 1), the age at operation was 5.9 ± 5 months (mean $\pm$ standard error of the mean) and

Fig. 3. Oversized patch technique used to close multiple muscular VSDs. The sutures are brought back across the septum from the left to the right side and secured over felt buttresses away from the edges of the defect. The patch is held in position on the left side of the septum by the sutures and the higher left ventricular pressures (*inset*). RV, Right ventricle; LV, left ventricle.

the weight was 5.3 ± 1.8 kg. In contrast, the 12 patients whose pulmonary artery beds were protected (group 2) had a mean age of 79.2 ± 38.4 months and weighed 20.4 ± 17.2 kg ($p = 0.006$ and 0.005 for age and body weight, respectively) at the time of the operation. Only one of the group 1 patients was older than 1 year of age, whereas in group 2 only one patient was younger than 1 year of age. The locations and surgical approaches of the VSDs are shown in Table II. There was no significant difference in the distribution of the VSDs between groups.

No early or late deaths occurred among group 1 patients. Significant complications included hemidiaphragmatic paralysis in one patient necessitating early diaphragm plication, one sternal dehiscence, three instances of transient complete heart block, and neurologic dysfunction in one patient with Down syndrome. One early death occurred among group 2 patients. This occurred in a patient with double-outlet right ventricle, hypoplastic left ventricle, and mitral stenosis who had previously undergone placement of a pulmonary artery band and multiple attempts at device closure of apical and anterior muscular VSDs. Death was due to intractable ventricular arrhythmias. The complications in group 2 patients included progressive biventricular failure in two patients requiring late orthotopic cardiac transplantation and two occurrences of complete heart block necessitating pacemaker placement. In one

Fig. 4. Apical left ventriculotomy used to close apical VSDs. The incision is placed to avoid major coronary branches and can be considerably smaller than the defect. The apex of the VSD is the incision itself and the patch used for closure is incorporated into the closure of the incision.

patient, mitral valve injury during the operation was repaired, but progression of mitral regurgitation led to mitral valve replacement 5 months later.

Five patients had hemodynamically significant residual VSDs and required reoperation (Table III). Hemodynamically significant residual VSDs were considered to be present if the patient had low cardiac output, congestive heart failure, pulmonary hypertension, or a combination of these problems. These procedures were all performed during the same hospitalization. Two of the group 1 patients required reoperation for closure of residual VSDs, located along the margin of the VSD patch in one and in the apical portion of the septum in the other. The residual apical defect had not been appreciated on the preoperative study. Three patients in group 2 required reoperation for residual defects, located in the anterior muscular septum in two and in the midmuscular septum in the other. One of the residual anterior defects occurred in the only patient who died in the hospital and was also not diagnosed before the operation. In two additional patients, the intraoperative transesophageal Doppler echocardiogram performed immediately after separation from cardiopulmonary bypass detected previously undiagnosed VSDs. The VSDs in both were located in the anterior septum and were

Fig. 5. Sandwich technique used for closure of anterior muscular VSDs. A series of mattress sutures are placed through the septal muscle, continued through the anterior wall of the ventricle, and then through a felt buttress. Care is taken to avoid the left anterior coronary artery.

successfully closed before the patients left the operating room.

Follow-up is complete in group 1 patients (100%) and all are alive and free of significant residual cardiovascular conditions at a mean follow-up of 23.4 ± 5.1 months (range 1 to 77 months). Fourteen of these patients are free of symptoms and free of medications, and the remainder receive a combination of digoxin, diuretics, and/or afterload reduction at the discretion of the referring cardiologist. All are in normal sinus rhythm with left ventricular ejection fractions of $66\% \pm 7\%$ (range 49% to 72%). Follow-up is available for nine patients (75%) in group 2 at a mean of 36.2 ± 8 months (range 1 to 88 months). Two of these patients underwent late orthotopic cardiac transplantation for severe ventricular failure and seven others remain free of significant residual cardiovascular conditions. All seven of these patients are free of symptoms, free of medications, and have a left ventricular ejection fraction of $72\% \pm 9\%$ (range 58% to 80%). Six patients are in normal sinus rhythm and one remains in complete heart block and is pacemaker dependent. None of the patients in either group has apical

Fig. 6. Sandwich technique used for closure of anterior muscular VSDs. The defects are closed by "sandwiching" them against the anterior wall of the heart.

ventricular aneurysms or significant residual left-to-right shunts.

Discussion

Despite advances in preoperative diagnosis, improved surgical techniques, and postoperative care, a significant morbidity and mortality still exists in the treatment of patients with multiple VSDs. This has led to considerable controversy regarding the optimal management of these patients. Banding of the pulmonary artery to protect the pulmonary vascular bed and relieve symptoms of congestive heart failure has traditionally been used in an effort to delay definitive repair. In addition, this procedure may allow time for, or even promote, spontaneous closure of some of the defects, possibly increasing the likelihood of a successful surgical outcome. Recently, the introduction of device closure for muscular VSDs has been recommended to avoid the need for surgical intervention or to simplify operative repair.⁴ This approach is also associated with significant complications, however, and has not produced uniformly successful results. Accurate diagnosis is essential to enhance the chances for a satisfactory outcome. A combination of cardiac catheterization and two-dimensional Doppler echocardiography with complete color flow mapping is indicated in the majority of patients. Five infants

Table II. Anatomic location of VSDs

	Age	Location	Perimembranous	Muscular defect					Subpulmonary	Approach	Residual	
				Posterior	Midmuscular	Anterior	Apical	VSD			Reoperation	
Group 1												
1	1 mo	Pe, Ap, An	+			+	+			RA, LV	An	No
2	5 mo	Pe, Ap, An	+			+	+			RA, RV	Ap	Yes
3	4 mo	Po, Ap		+			+			RA, LV	No	No
4	4 mo	Mi, Ap			+		+			RA, LV	No	No
5	6 mo	Mi, Sp, Ap			+		+	+		RA, PA	No	No
6	8 mo	Pe, Po	+	+						RA	No	No
7	8 mo	Pe, An, Ap, Mi	+		+	+	+			RA, LV	Mi	No
8	9 mo	Pe, Po	+	+						RA	No	No
9	8 mo	Pe, Po, An	+	+		+				RA	Ap	No
10	9 mo	Pe, Inf	+					+		RA	Ap	No
11	10 mo	Pe, Mi	+		+					RA	No	No
12	11 mo	Pe, Ap	+				+			RA, LV	No	No
13	17 mo	Pe, An/Ap	+				+			RA, LV	No	No
14	6 days	Ap, Pe	+				+			RA, LV	No	No
15	20 days	An, Mi			+	2+				RV	No	No
16	6 mo	Pe, An	+			+				RA	No	No
17	4 mo	An, Mi			+	+				RA	No	No
18	12 mo	Multiple Mi			3+					RA	Patch leak	Yes
19	8 days	Multiple Mi			2+					RA	No	No
20	15 days	Multiple An				2+				RA	No	No
21	1 mo	Multiple An				3+				RA, LV	No	No
Group 2												
1	4 mo	Multiple Po		2+						RA	No	No
2	4 yr	Po, (An), Pe	+	+		device+				RA	No	No
3	4.5 yr	Mi, Pe	+		+					RA	An	Yes
4	2 yr	Mi, Pe	+		+					RA	No	No
5	14 mo	Mi			multi+					RA	No	No
6	2.75 yr	Inf, Mi			+			+		RA	No	No
7	11 yr	Pe, Mi	+		+					RA	Ap	No
8	37 yr	Pe, Mi	+		+					RA	No	No
9	4.5 yr	Pe, Po, (Ap, An)	+	+		device+	device+			RA	An	Yes
10	6 yr	Mi, An, Ap			+	+	+			RA	No	No
11	6 yr	Pe, Ap	+				+			RA, LV	An	Yes
12	11 yr	Po, Ap		+			+			LV	No	No

An, Anterior muscular; Ap, apical muscular; Inf, infundibular; Mi, midmuscular; Pe, perimembranous; Po, posterior; LV, left ventricle; PA, pulmonary artery; RA, right atrium; RV, right ventricle; SP, subpulmonary.

with discrete defects in this series did not undergo cardiac catheterization because it was believed that the anatomy of the VSDs was clearly defined by Doppler echocardiography alone. In none of these patients was any important VSD overlooked. However, although the correct diagnosis was achieved before the operation in 11 of 12 patients with right ventricular outflow tract obstruction, only 17 of the 21 patients with pulmonary hypertension received a complete diagnosis before the operation. It is likely that the increased difficulty in accurately diagnosing both the presence and hemodynamic significance of muscular VSDs, particularly those located in diffi-

Table III. Location of residual VSDs

Location	Group 1	Group 2
Midmuscular	0	1
Apical	1	0
Anterior	0	2
Patch	1	0

cult to visualize locations in the anterior and apical regions of the septum, are related to uncertainties associated with multiple small defects when they exist in association with larger defects. The large left-to-right shunt and pulmonary hypertension re-

sulting from the larger defects may obscure the magnitude of the smaller ones. This underscores the importance of fastidious preoperative evaluation in preparing for surgical correction.

The timing of corrective surgery is influenced by the location of the defects, symptoms, and the presence of associated cardiovascular conditions. The presence of associated major cardiac lesions and the complexity of the underlying malformation is a significant factor in the mortality associated with complete repair of multiple VSDs.¹² Although the group of patients with normal pulmonary artery pressure caused by naturally occurring pulmonary stenosis or surgically placed pulmonary artery bands were older at referral and were well compensated, this group fared less well than those who underwent primary repair in infancy. The explanation for this difference is not entirely clear from these data, and multiple factors are likely to influence outcome, including the complexity of the associated malformation itself. However, it was clear that the younger patients without pulmonary artery stenosis undergoing primary repair were no more likely to have residual defects after closure than those undergoing operation at an older age, despite the fact that there was no difference in the number or location of the VSDs between the two groups. It has been our impression that closure of muscular VSDs was actually easier in the younger age group and that accurate definition of the defect edges was more precise. In the older patients with long-standing right ventricular hypertension, the ventricular septum was extremely hypertrophied, making exposure more difficult. More extensive division of right ventricular muscle trabeculations was required, which may adversely affect ventricular function after the operation. Because the development of pulmonary vascular disease must also be factored into the decision to intervene,¹³ our experience indicates that palliation with pulmonary artery banding is unnecessary and that early repair is associated with excellent results.

The need for left ventriculotomy has not been a significant deterrent to primary closure in our experience, even in the neonate. In a separate study from this institution, nine of 13 patients undergoing apical left ventriculotomy for VSD closure underwent prospective assessment of myocardial function at a mean of 47 months after repair.¹⁰ Left ventricular ejection fraction was normal in all (median 55%), including patients undergoing operation as early as 1 week of age. It is important, however, to empha-

size that all these patients had limited incisions confined to the apex of the left ventricle to close apical defects only. Defects located in the more anterior portions of the septum would require a larger incision in the left ventricle for exposure, which would be more likely to result in ventricular dysfunction. Although apical left ventriculotomy is avoided wherever possible in favor of a transatrial approach, when used as described herein, we have seen no adverse effect on ventricular function.

A careful, systematic approach to closure of muscular VSDs should result in excellent exposure and complete closure in the majority of patients, regardless of location. All defects are initially explored through the right atrium and, when necessary, the left atrium through the atrial septum. Dividing muscle trabeculations along the right side of the septum improves the exposure and facilitates accurate identification of the true margins. The base of the septal band and the moderator band may be safely divided when necessary. Tricuspid valve function should be carefully assessed and a valvuloplasty performed if tricuspid regurgitation results from distortion of papillary muscles. When the number of muscle trabeculations obscuring the VSD from view is considerable and division is judged to be too extensive, the use of an "oversized" patch placed into the left ventricle has proved to be a useful technique. Precise delineation of the defect margins becomes less important because the patch will effectively "seal" the left side of the septum and is held in place by the higher left ventricular pressure after it is anchored by a few sutures.

Our experience indicates that primary repair for infants with multiple VSDs is associated with good long-term results. A transatrial approach is satisfactory for all muscular defects with the exception of those defects located in the apical muscular portion of the septum, which may require limited apical left ventriculotomy for proper exposure. Repair through a limited apical left ventriculotomy did not add to the risk of the procedure, was associated with normal late left ventricular function, and should be considered for optimal visualization. Symptomatic infants may safely undergo primary repair, and pulmonary artery banding is unnecessary.

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Discussion

Dr. John W. Brown (*Indianapolis, Ind.*). Dr. Durham and his coauthors from Ann Arbor have demonstrated that patients without associated complex lesions can undergo repair in infancy without mortality, without permanent heart block, and without significant residual shunts. They state that pulmonary artery banding alone is needed only in the very complex subgroup.

The timing of the operation in group 1 patients was influenced by the location of the defect, symptoms, and the presence of associated lesions. The authors have the impression that closure of multiple VSDs in infants is easier than in older patients and that accurate definition of the edges of the defects is more precise. The mean age and weight of the patients in group 1 was 6 ± 5 months, and the weight was 5 ± 2 kg. In our practice, the majority of infants who have multiple VSDs become symptomatic in the neonatal period, when they usually weigh less than 3 kg.

I have four questions for Dr. Durham. How early in infancy will you attempt complete repair, and do you have a threshold weight for surgical intervention? Would you attempt repair or banding in a 2 kg neonate who is ventilator-dependent with congestive heart failure?

Although there were no deaths, significant residual shunts,

or pulmonary hypertension at last follow-up in your patients, a third of them were still on a program of digitalis, diuretics, and afterload reduction. Why? Was right ventricular function impaired by your aggressive approach?

In group 2, one patient died of ventricular arrhythmia and two required cardiac transplantation. What was the cause of their ventricular dysfunction after the operation?

Finally, how do you keep the multiple sutures organized when you place the VSD patch through the VSD and anchor it on the left ventricular side? How do you avoid injury to the mitral valve?

Dr. Thomas L. Spray (*Philadelphia, Pa.*). If you saw a residual defect now by echocardiography, would you immediately reoperate on all patients and not take the patient out of the operating room? Were any of these reoperations done later? You said they were done at the same hospitalization. Were they done at the same operation in all cases?

I also noticed the relatively small incidence of anterior muscular defects in this series. In my experience, those are the hardest ones to see, especially through the right atrium. The apical defects are usually fairly easy to find. Do you have any tricks to find the very high anterior muscular defects up underneath the pulmonary valve? That area is difficult to get to in some cases.

Were the patients who died or required transplantation those patients who had apical incisions for apical defects or were they patients who had biventricular dysfunction from prolonged pulmonary banding? Do you have any comments about that?

Dr. Durham. Dr. Brown asked how early we would intervene. Depending on the clinical status of the child, we have intervened at less than 1 week of age.

We have no absolute threshold weight. That has to be taken in context to the clinical status of the child. If the child is sick, weight is a consideration, possibly an indication for pulmonary banding. We do not think pulmonary artery banding is necessarily bad, but it does dictate an obligatory second operation, which could be made more difficult by hypertrophy of the septum. Banding itself is not without complications, because of distortion of the pulmonary arteries.

Regarding the treatment of a 2 kg infant who is ventilator dependent, I think we would favor banding.

Your second question concerned right ventricular function in children who underwent primary repair while on a program of digitalis, diuretics, and afterload reduction. Once they are being followed up by the cardiologist, they tend to continue receiving a lot of these medications. The medications are phased out over time. I am not sure why the seven patients whom you mentioned were still receiving this support.

One child who underwent cardiac transplantation had a complex course with multiple attempts at device closure and subsequent failure, which necessitated extracorporeal membrane oxygenator support. He underwent transplantation after that. The other child had complex multiple VSDs and had problems with myocardial preservation during the operation, which resulted in ventricular failure and subsequent transplantation.

As to tricks regarding the oversize patch technique, how we keep the sutures straight, and how we avoid the mitral valve, realistically, it is difficult at best. We attempt to

keep the sutures on rubber shods and keep them straightened out by using three people in the operating room to keep them sorted and assure that they are not mangled. If you are not careful you may twist the patch or put in the eighth suture and find that it has just crisscrossed the previous seven. With an oversized patch, I would restress that we use relatively few sutures because the general higher left-sided pressures hold the patch in place. Seven or eight sutures are needed, not 15 or 20.

Dr. Spray inquired about timing of reoperation. Generally, we perform transesophageal echocardiography on all patients who have had VSD closure. The decision that a residual defect may become hemodynamically significant is somewhat arbitrary in some cases. In two of our patients transesophageal echocardiography identified previously undiagnosed anterior muscular VSDs. The defects were closed because the magnitude of the shunt was believed to be quite large because there were multiple VSDs. If we have any problem weaning the child from bypass or if the child looks unwell after being weaned, then we have to consider going back on bypass and fixing those VSDs.

Commentary

The article by Kitagawa and associates has added substantially to our database and understanding of multiple VSDs. This retrospective study of 33 patients, managed with advanced surgical techniques, demonstrates both considerable progress (mortality was only 3%) and considerable room for improvement, with "significant" morbidity in 33% and a reoperation rate of 15%.

The study also contains several important inferences that seem likely to stand the test of time. Right ventriculotomy adds little to the exposure of these VSDs: most muscular VSDs can be successfully seen and closed from an atrial approach. Infants who have midmuscular, posterior muscular, and infundibular VSDs (almost one third of

this series) do well with early primary surgical repair. The authors contend that pulmonary artery banding is unwarranted in these infants, and the data support that contention.

One inference, however, deserves much more scrutiny. The authors conclude that infants with apical or anterior muscular VSDs can undergo repair early with "good long-term results." Central to this conclusion is their belief that a limited fish-mouthed left ventriculotomy, required in seven of nine infants with apical VSDs, is a benign procedure on late follow-up.

The prior history of ventriculotomy as a cardiac surgical procedure would not seem to warrant such optimism. Late complications of aneurysm formation, reduced global function, and ventricular arrhythmias have been repeatedly observed after ventriculotomy for tetralogy of Fallot, single ventricle with restrictive bulboventricular foramen, and left ventricular apical aortic conduits. Despite this experience, one cannot dismiss left ventriculotomy from the arsenal of therapies that might help in the management of this difficult disease. One should, however, recognize the experimental nature of infantile left ventriculotomy and require careful follow-up. Absence of significant problems on standard clinical follow-up is not enough. A careful program in these patients, studying left ventricular mechanical function, left ventricular electrical function, mitral valve function, and completeness of septal closure by means of sophisticated noninvasive and, when necessary, invasive studies would seem only prudent. Given the unfortunate history of ventriculotomies, the burden of proving its safety lies with those who advocate its increased use.

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