Indications and Outcomes of Surgical Closure of Ventricular Septal Defect in Adults

François-Pierre Mongeon, MD,* Harold M. Burkhart, MD,† Naser M. Ammash, MD,*
Joseph A. Dearani, MD,† Zhuo Li, MS,‡ Carole A. Warnes, MD,*
Heidi M. Connolly, MD*

Rochester, Minnesota

Objectives We sought to review our experience with surgical ventricular septal defect (VSD) closure in adults.

Background Outcome data of VSD closure in adults on which to base recommendations are limited. Guidelines recommend closure of adult VSD for a Q_p/Q_s ratio ≥ 2, left ventricular volume overload, or endocarditis.

Methods We retrospectively reviewed 46 patients (mean age 33.6 ± 11.2 years, 24 women) who underwent surgical VSD closure (1958 to 2008).

Results VSDs were classified according to the Society of Thoracic Surgeons as type 2 (membranous, 72%) or type 1 (subarterial, 26%). Aortic regurgitation (AR) (28%), left ventricular dilation (20%), and pulmonary hypertension (20%) were the most common indications for closure. Associated lesions justified surgery in 39% of patients. There were no early deaths. Morbidity included 1 high-grade atrioventricular block requiring permanent pacemaker and a residual VSD in 7 patients. Late mortality was 5% (mean follow-up: 10.3 ± 12.4 years). Patient survival did not differ from expected survival in a reference population (p = 0.75). Late residual VSDs were more common after suture closure (6 of 8 patients). Late moderate AR developed in 5 patients (4 with a type 1 VSD) with aortic valve or sinus of Valsalva repair. The use of intraoperative transesophageal echocardiography was associated with fewer residual VSDs and less $\geq$ moderate tricuspid valve regurgitation and AR.

Conclusions Associated heart defects and AR were common indications for VSD closure in adults, which was performed with low mortality and morbidity. Patch closure and use of intraoperative transesophageal echocardiography improve surgical outcomes. Important residua emphasize the need for life-long informed follow-up.   (J Am Coll Cardiol Intv 2010;3:290–7) © 2010 by the American College of Cardiology Foundation

Ventricular septal defect (VSD) is a very common congenital heart defect in children, but due to spontaneous and surgical closure, it is less commonly encountered in adults. Studies have reported the long-term outcome of small VSDs that were not closed during childhood (1–3). About one-third of patients with VSD initially managed medically required surgical intervention later in life (1).

The outcome of adult patients with an unoperated VSD is not uniformly favorable. An increased incidence of sudden death has been noted regardless of the defect size (1). Five-year survival without endocarditis or surgery was reported to be 95.5%, with only 3 of 222 patients requiring surgical closure (3). In a tertiary referral population, Neumayer et al. (2) reported that 25% of patients with small VSDs had serious complications, such as aortic valve regurgitation (AR) and endocarditis; these were the most common indications for surgical VSD closure in adults, occurring in 10.6% of this series (2).

Current practice guidelines recommend surgical closure of adult VSDs if there is a left-to-right shunt with a Q_p/Q_s ratio >2 and evidence of left ventricular (LV) volume overload or associated infective endocarditis (4).

Most surgical series of VSD closure in adults describe the earlier experience (5,6), making contemporary outcome data on which to base recommendations very limited (7). We gathered the largest contemporary single center experience with surgical VSD closure in adults.

Methods

Patient selection. Patients with a VSD who underwent surgical closure were identified from the Adult Congenital Heart Disease Clinic, Cardiac Surgery, and Echocardiography databases at Mayo Clinic. For inclusion, patients had to be 18 years of age or older at the time of surgical VSD closure done at Mayo Clinic in Rochester, Minnesota. The exclusion criteria were the presence of any of the following: 1) a double inlet or double outlet ventricle; 2) transposition of the great arteries; 3) atresia of any valve; 4) a conotruncal abnormality; 5) an atrioventricular septal defect; or 6) Ebstein anomaly. The protocol was approved by the Mayo Clinic Institutional Review Board.

Three hundred eighty-nine patients were identified among which 343 were excluded because they met exclusion criteria or did not undergo VSD closure as part of their operation. The remaining 46 patients formed the study group.

Data collection. We used the Society of Thoracic Surgeons (STS) classification of VSDs (8) as proposed in national practice guidelines (4). Conal, subpulmonary, infundibular, supracristal, and doubly committed juxta-arterial defects are grouped under STS type 1 VSDs. Perimembranous, paramembranous, and conoventricular defects form the STS type 2. The STS type 3 VSDs include inlet and atrioven-

tricular canal varieties, and muscular VSDs are designated as STS type 4 (4). Details regarding past medical and surgical history, intracardiac hemodynamics, ventricular function, associated cardiac anomalies, pre- and post-operative Warnes-Somerville ability index (9) and New York Heart Association functional class, surgical indication, and both early and late outcomes were abstracted from a retrospective review of clinical notes, surgical protocols, and imaging study reports.

Follow-up started on the day of surgery. The early post-operative period was defined as 30 days after surgery or as the surgical hospitalization, whichever was longer. Follow-up ended on the latest date at which the patient's vital status could be ascertained. All pre-operative evaluations were performed by the patients' personal physicians at Mayo Clinic. Follow-up clinical evaluations and imaging studies performed at Mayo Clinic were reviewed, external clinical documents on file at Mayo Clinic were reviewed when Mayo Clinic evaluation was not performed.

Surgery. Standard techniques for cardiopulmonary bypass were used. Cold potassium, blood cardioplegic solution has been used for myocardial protection since 1977. The use of suture or patch closing technique and the approach to the ventricular septum were noted.

The largest VSD diameter or the diameter of the largest defect at surgical inspection was used as the gold standard for assessment of defect size. When a VSD diameter was not reported in the surgical note, the diameter reported in pre-operative imaging studies was used.

Echocardiography. Use of state-of-the-art equipment and protocols (10,11) could be ensured for the majority of comprehensive 2-dimensional and Doppler echocardiograms. Data were abstracted from clinical reports.

The LV size and ejection fraction were assessed with 2-dimensional echocardiographic guidance (12), by visual estimate (13), or using the Simpson biplane method (14). Tricuspid valve regurgitation (TR) and AR were assessed semiquantitatively as absent, trivial, mild, moderate, moderate-to-severe, and severe. Tricuspid regurgitant jet systolic velocity by continuous wave Doppler was used to estimate the right ventricular systolic pressure (15). The diameter of the VSD was measured on 2-dimensional imaging. The peak flow velocity through the VSD was measured by continuous wave Doppler.

Catheterization. Hemodynamic calculations were verified using reported data from pre-operative cardiac catheteriza-

Abbreviations and Acronyms

AR = aortic valve regurgitation

IOTEE = intraoperative transesophageal echocardiography

LV = left ventricle

LVEF = left ventricular ejection fraction

STS = Society of Thoracic Surgeons

TR = tricuspid valve regurgitation

VSD = ventricular septal defect

tion performed at Mayo Clinic or at referring institutions when a recent study was performed there.

Statistical analysis. Descriptive statistics for categorical variables are reported as frequency and percentage whereas continuous variables are reported as mean and standard deviation. Kaplan-Meier method was used to estimate the survival rate at 5, 10, and 15 years. Log-rank test was used to compare the survival of our patient group to the Minnesota white population. All statistical tests were 2-sided with the alpha level set at 0.05 for statistical significance.

Results

Baseline characteristics. The baseline characteristics of our study population are presented in Table 1. Most of the patients were 30 years of age or younger at the time of surgery. The oldest patient was 64 years of age. The STS type 2 VSDs were the most common. Five patients (11%) presented with a residual VSD after attempted closure, and 1 patient had multiple septal defects. The pre-operative functional class was good in most patients.

The VSD diameter was measured at time of surgery in 37 patients. An echocardiography-derived VSD diameter was

Table 1. Baseline Characteristics (n = 46)

Age at surgery, yrs	33.6 ± 11.2
Female sex	24 (52)
Society of Thoracic Surgeons type of VSD	
Type 1 (subarterial)	12 (26)
Type 2 (membranous)	33 (72)
Type 3 (inlet)	1 (2)
Type 4 (muscular)	0
VSD diameter, mm	11.4 ± 10.5
Associated lesions	
Coronary cusp prolapse	15 (33)
With sinus of Valsalva aneurysm	5 (11)
Patent foramen ovale	7 (15)
Bicuspid aortic valve	6 (13)
Mitral regurgitation	6 (13)
Right ventricular outflow tract obstruction	6 (13)
Atrial septal defect	5 (11)
First degree atrioventricular block	3 (7)
Patent ductus arteriosus	3 (7)
Persistent left superior vena cava	3 (7)
Coarctation of the aorta	2 (4)
Warnes-Somerville ability index	
1	31 (67)
2	13 (28)
3	2 (4)
New York Heart Association functional class	
I	30 (65)
II	15 (33)
III	1 (2)

Data are n (%) or mean ± SD.
   VSD = ventricular septal defect.

Table 2. Pre-Operative Echocardiographic and Catheterization Data

Echocardiography		
Left ventricular end-diastolic diameter, mm	39	55 ± 8
LVEF, %	42	61 ± 7
Tricuspid regurgitation peak velocity, m/s*	26	2.81 ± 0.46
Tricuspid regurgitation ≥ moderate	34	6 (18)
Aortic regurgitation ≥ moderate	35	6 (17)
Catheterization		
Q_p/Q_s	19	1.6 ± 0.5
Left ventricular end-diastolic pressure, mm Hg	16	12 ± 3
Right ventricular systolic pressure, mm Hg*	20	50 ± 27
Mean pulmonary artery pressure, mm Hg	20	25 ± 16
Pulmonary vascular resistance index, WU · m²	18	5.6 ± 4.5

Data are n (number of patients for which the data were available), n (%), or mean ± SD. *Patients with right ventricular outflow tract obstruction and double chamber right ventricle were excluded from the analysis of this parameter.
   LVEF = left ventricular ejection fraction.

used in 4 patients, and 5 had no measure of the defect diameter. One-half of the patients had a VSD diameter smaller than 7 mm. The largest VSD was a 50-mm STS type 2 defect in a patient who had associated right ventricular outflow tract obstruction due to previous pulmonary artery banding.

Associated cardiac lesions are presented in Table 1. A sinus of Valsalva aneurysm without cusp prolapse was present in 2 patients. Peripheral pulmonary stenosis, double chamber right ventricle, pulmonary valve regurgitation, subaortic stenosis, valvular aortic stenosis, an aneurysm of the ascending aorta, and mitral valve prolapse were each present in 1 patient.

Pre-operative echocardiographic and catheterization data. A pre-operative echocardiogram was available in 42 patients and a pre-operative catheterization was available in 21 patients (Table 2). The LV was enlarged in the majority of patients (mean LV end-diastolic diameter 55 mm, range 39 to 79 mm). The systolic function was preserved or mildly impaired (LV ejection fraction [EF] range 42% to 74%). Any degree of AR and TR were present in 24 (69%) and in 32 (94%) patients, respectively. There was no clustering of AR or TR according to the VSD type. The peak velocity of flow across the VSD was reported in 23 patients. It was high (mean 4.9 ± 0.7 m/s, range 3.3 to 6.7 m/s) suggesting that most defects were restrictive. Right ventricular and pulmonary artery pressures and resistance were moderately elevated. A selection bias may exist with the patients most likely to have pulmonary hypertension or those in whom noninvasive assessment of pulmonary pressures was not possible being submitted to catheterization.

Indications for VSD closure. The surgical indications for VSD closure are listed in Table 3 in decreasing order of frequency. Coexisting congenital or acquired structural heart lesions were the primary motivation for surgical repair in 18 patients (39%). In addition, surgery was justified by
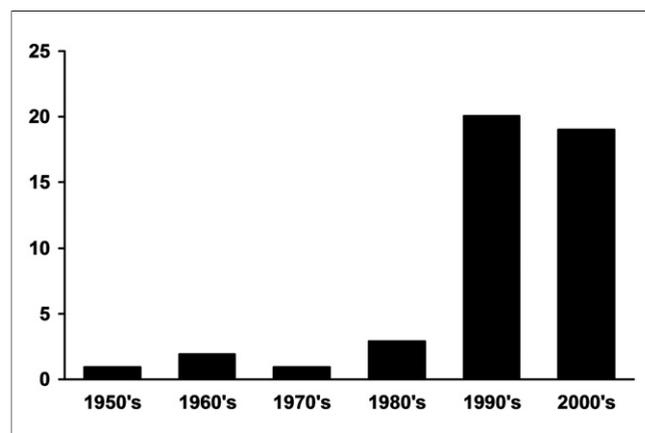
J A C C : C A R D I O V A S C U L A R I N T E R V E N T I O N S , V O L . 3 , N O . 3 , 2 0 1 0
M A R C H 2 0 1 0 : 2 9 0 – 7

Mongeon *et al.*   293
Surgical VSD Closure in Adults

Table 3. Indications for Surgery

Aortic regurgitation	13 (28)
Left ventricular dilation	13 (28)
Pulmonary hypertension	9 (20)
Endocarditis	8 (17)
Symptoms	6 (13)
Left ventricular dysfunction	4 (9)
Sinus of Valsalva aneurysm	5 (11)
Ruptured	3 (7)
Not ruptured	2 (4)
Coronary cusp prolapse	3 (7)
Double chamber right ventricle	3 (7)
Mitral regurgitation	3 (7)
$Q_p/Q_s \geq 2$	3 (7)
Subaortic stenosis	1 (2)

Data are n (%). Total is >100% because many patients had multiple indications.

Table 4. Surgical Data (n = 46)

Prior cardiac surgery	8 (17)
Prior ventricular septal defect closure	5 (11)
Type of closure	
Primary closure	22 (48)
Patch closure	24 (52)
Dacron	15 (33)
Pericardium	1 (2)
Gore-Tex	2 (4)
Teflon	4 (9)
Unspecified patch	2 (4)
Surgical approach	
Transatrial	23 (50)
Right ventricular	16 (35)
Aorta	4 (9)
Pulmonary artery	2 (4)
Concomitant procedures	34 (74)
Aortic valve or sinus of Valsalva repair	12 (26)
Tricuspid valve repair	11 (24)
Atrial septal defect closure	9 (20)
Resection of right ventricular or subpulmonary diaphragm	4 (9)
Mitral valve repair	4 (9)
Aortic valve replacement	3 (7)
Cardiopulmonary bypass time, min	84 ± 42
Aortic cross clamp time, min	52 ± 29

Data are n (%) or mean ± SD.

the following conditions in 4 patients: cyanosis due to a large VSD with an obstructive pulmonary artery band, large atrial septal defect, severe aortic stenosis, and severe coarctation of the aorta. To be allowed to serve in the military, 1 patient requested a VSD closure.

Surgical data. The earliest surgery was done in 1958, but most were performed after 1980 (Fig. 1). Suture closure was used in 22 patients (48%) (Table 4). Nearly all of them (18 of 22) had a VSD diameter ≤10 mm or described as small. The most common approach for VSDs was from the atria. An LV approach was never required. A majority of patients (74%) underwent concomitant repair of other cardiac defects.

Tricuspid valve repair was performed in 11 patients (24%) (Table 4) because of tricuspid tissue obstructing the VSD. Pre-operative severe TR was uncommon (6%). The tricuspid valve was repaired in 4 patients with mild or less TR (Fig. 2).
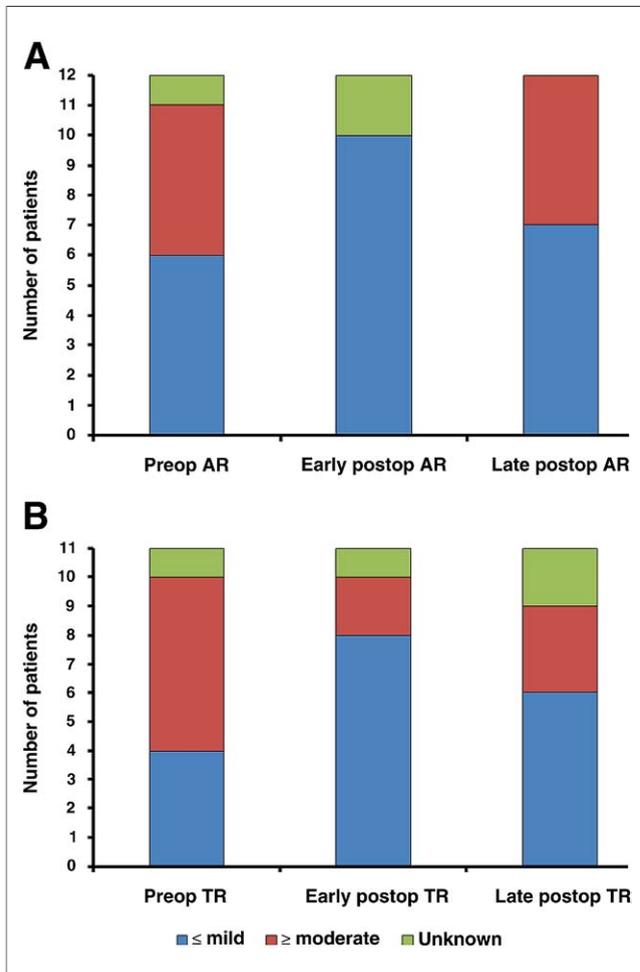
Aortic valve or sinus of Valsalva repair were performed in 12 patients (26%) (Table 4). Among them, 6 patients had mild or less AR pre-operatively (Fig. 2); in 3 patients, a ruptured sinus of Valsalva aneurysm justified surgery. Of the remaining 3 patients, 2 had cusp prolapse.

In addition, 2 patients had surgical enlargement of the right ventricular outflow tract; 2 had closure of a patent ductus arteriosus; and pulmonary valvotomy, resection of subaortic stenosis, Maze procedure, resection of a membranous septal aneurysm, repair of coarctation of the aorta and LV myectomy, ligation of a left superior vena cava, and an ascending aortoplasty were performed in 1 patient each.

Outcomes. There was no operative mortality. Post-operative atrial fibrillation occurred in 9% of patients. There was 1 case of high-grade atrioventricular block requiring early post-operative permanent pacing. Significant pleural and pericardial effusions occurred in 3 patients and 1 had cardiac tamponade 2 months after surgery. Post-operative morbidity also included hemorrhage (not leading to reoperation), thrombophlebitis, retained temporary pacemaker wire, and an episode of unexplained fever and abdominal pain, occurring in 1 patient each. Among 40 patients with an early post-operative echocardiogram, the mean LV end-diastolic diameter and LVEF were 50 ± 8 mm and 55 ± 9%, respectively.

Follow-up beyond hospital dismissal, available in 40 patients, ranged from 20.5 months to 47 years after surgery.

Figure 1. Distribution of Surgical VSD Closures at Mayo Clinic

Distribution of surgical ventricular septal defect (VSD) closures per decade at Mayo Clinic. Only 4 patients were operated before 1980.

Figure 2. Outcomes of Valve Repair at Time of Surgical VSD Closure

Outcomes of valve repair at the time of surgical ventricular septal defect (VSD) closure. **(A)** Aortic valve regurgitation (AR) before and after aortic valve or sinus of Valsalva repair in 12 patients (7 Society of Thoracic Surgeons [STS] type 1 VSDs and 5 STS type 2 VSDs). **(B)** Tricuspid valve regurgitation (TR) before and after tricuspid valve repair in 11 patients, all with a STS type 2 VSDs. Preop = pre-operative; postop = post-operative.

A late post-operative echocardiogram was available for 34 patients. Only 3 late studies were performed outside Mayo Clinic. Late mortality was low (5%) (Table 5). Survival rates at 5, 10, and 15 years after surgery were 100%, 95%, and 88%, respectively, and did not differ significantly from expected survival rates (p = 0.75) (Fig. 3). Patients had a good functional status (Table 5). The cause of the 2 late deaths in our cohort could not be identified. We found no record of sudden cardiac death. One patient (3%) had documented nonsustained ventricular tachycardia and 10% of patients developed atrial fibrillation at late follow-up. One patient underwent 2 reoperations: a residual VSD was closed 42 years after the initial surgery, along with coronary artery bypass grafting and 2 successive aortic valve replacements. One patient had atrioventricular node ablation and

Table 5. Late Outcomes After Surgical VSD Closure

Follow-up, yrs	40	10.3 ± 12.4
Mortality	40	2 (5)
Warnes-Somerville ability index 1–2	37	37 (100)
New York Heart Association functional class I–II	37	36 (97)
High-grade atrioventricular block	34	0
Permanent pacemaker	35	3 (9)
Ventricular tachycardia	40	1 (3)
Atrial fibrillation	40	4 (10)
Atrial flutter	40	2 (5)
Supraventricular tachycardia	40	4 (10)
Residual VSD	35	8 (23)
Reoperation	40	2 (5)
Echocardiogram at latest follow-up		
Time after surgery, yrs	34	9.7 ± 12.9
Left ventricular end-diastolic diameter, mm	33	52 ± 6
LVEF, %	32	57 ± 8
Tricuspid regurgitation peak velocity, m/s	28	2.66 ± 0.48
Tricuspid regurgitation ≥ moderate	28	6 (21)
Aortic regurgitation ≥ moderate	30	5 (17)

Data are n (number of patients for which the data were available), n (%), or mean ± SD. Abbreviations as in Tables 1 and 2.

pacemaker implantation 8 months after surgery for persistent atrial arrhythmias.

Among the 7 residual VSDs (18% of patients) identified in the post-operative period, 6 were described as small; size was not reported for 1 VSD identified with dye curves. Among the 8 residual VSDs (23% of patients) identified at late follow-up, 7 were small and 1 was of moderate size. It is noteworthy that suture closure was used in 6 of 8 patients with a residual VSD at late follow-up. Echocardiographic screening for a residual VSD was performed in 87% of
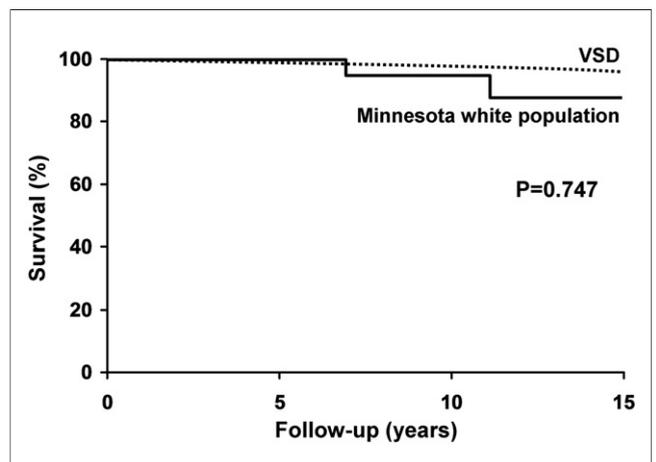
Figure 3. Survival After Surgical VSD Closure Versus Reference Population

Survival after surgical ventricular septal defect (VSD) closure compared with a reference population. There was no difference in 15-year survival of operated patients compared with the Minnesota white population (p = 0.747).

patients post-operatively and in 74% of patients at late follow-up.

Some degree of TR was present in 33 of 34 patients (97%) and in all patients in whom it was assessed by echocardiography at early and late follow-up, respectively. Tricuspid valve regurgitation was moderate or greater in 5 patients (15%) early after surgery. Among the 6 patients with more than moderate TR at late follow-up, 5 had an STS type 2 VSD, 3 had a tricuspid valve repair, 3 had a residual VSD, 2 had pre-operative right ventricular outflow tract obstruction, 2 had a late TR velocity >2.5 m/s, and 1 had the VSD closed with a patch. Moderate or greater TR at late follow-up was present in 27% of patients (3 of 11) who had a tricuspid valve repair (Fig. 2).

Aortic valve regurgitation was present in 22 of 32 patients (69%) and in 23 of 30 patients (82%) with early and late echocardiographic follow-up, respectively. At early follow-up, all cases of AR were mild or less. The AR became moderate in 5 patients (17%) at late follow-up. Among these patients, all underwent aortic valve or sinus of Valsalva repair at the time of VSD closure, 4 had an STS type 1 VSD, and 3 developed an aneurysm of the proximal aorta during follow-up. Among patients who had aortic valve or sinus of Valsalva repair, AR decreased in 5 patients, increased in 2 patients, and did not change in 3 (unknown AR status in 2 patients) during follow-up (Fig. 2). The prevalence of moderate late AR after aortic valve repair was 42%.

IOTEE. Intraoperative transesophageal echocardiography (IOTEE) was used in 38 patients (82.6%). An early and a late residual VSD were present in 6 patients (15.8%) and in 4 patients (10.5%) who had an IOTEE compared with 1 patient (12.5%) and 4 patients (50.0%) who did not have an IOTEE. Moderate or more AR (n = 2, 25.0%) and TR (n = 2, 25.0%) at late follow-up were more common in patients who did not have an IOTEE. In patients who had an IOTEE, late ≥ moderate AR was present in 3 patients (7.9%) and late ≥ moderate TR was present in 4 patients (10.5%).

Early surgical experience. We identified 4 patients operated on between 1950 and 1980. The only meaningful difference in late outcomes is the presence of a residual VSD in 3 patients (75%) compared with 5 patients (12%) in the 1980 to 2008 group.

Discussion

Summary of results. We report the largest contemporary (91% of patients operated after 1980) series of adults who underwent surgical VSD closure at a single institution. The STS type 2 VSDs (membranous) were the most common. Severe TR and AR were uncommon.

The most common surgical indications were AR, LV dilation, and pulmonary hypertension. Patch closure and a transatrial approach were most frequently used. Suture closure was most commonly used in patients with a residual VSD, which was present in 18% of patients post-operatively and in 24% at late follow-up.

There were no perioperative deaths. Late mortality was 5%. Survival at 15 years was not different from the expected survival in a reference population. There was a low incidence of ventricular arrhythmias. The use of IOTEE was associated with fewer residual VSDs and less late ≥ moderate AR and TR.

Indications. Most surgical indications were consistent with previous series (2,6,7). Moderate pulmonary hypertension was the only indication for VSD closure in 2 patients and factored in the decision for surgery in 8 patients. These patients' VSDs measured 4 to 20 mm and 7 of the 8 patients did not have another lesion to account for the pulmonary hypertension. One patient had moderate-to-severe mitral regurgitation. Otterstad et al. (6) mentioned pulmonary hypertension among their surgical indications for medium and large VSDs only. It is, however, known that small VSDs can also be associated with pulmonary hypertension (1,16). Numerous congenital heart defects were present in our patients (Table 1). A bicuspid aortic valve was present in 13% of patients, an association already reported by Neumayer et al. (2). Although some of the coexisting lesions prompting operative repair were independent from the VSD, coronary cusp prolapse, often complicated by aneurysm of the sinus of Valsalva and always associated with AR, was the most common (n = 8). The AR was mild or less in 7 of 8 patients and 3 patients had a ruptured aneurysm. These observations suggest that associated heart defects are as important as the hemodynamic consequences of the VSD in deciding to perform surgical repair.

Early mortality. The absence of operative mortality in our series differs from the 6% reported by Kidd et al. (1) but is similar to another adult series (7). Most surgeries in our cohort were performed during the last 2 or 3 decades on patients in good pre-operative condition. Other adult VSD closure series reflect the earlier experience and report operative mortalities of 4.7% and 9.8% (5,6).

Late mortality. Mortality at a mean follow-up of 10 years was low in our series, similar to other reports (5–7) but lower than the late mortality (11% [55 of 483] of patients) of the surgically managed patients in the NHS-2 (Second Natural History Study) trial (1).

Arrhythmias. The low prevalence of ventricular arrhythmias at follow-up potentially explains the low mortality in our cohort. The previously reported incidences of sudden death (39% of all deaths) and serious arrhythmias (ventricular tachycardia 14.8%) in VSD patients, especially those treated surgically, were substantially higher (1,16). We cannot speculate on the influence of a ventricular or atrial approach on late survival because numerous patients in our cohort had a ventriculotomy to access another heart defect. The patient in our study who had nonsustained ventricular tachycardia

had an atriotomy and also had a large atrial septal defect with right ventricular volume overload. Echocardiographic follow-up was not available for this patient.

The common use of an atriotomy and the presence of various structural heart diseases may explain that atrial arrhythmias occurred in 23% of patients at follow-up. Atrial fibrillation (10%) was the most common atrial arrhythmia and was more common than in previous reports of surgically (7) and medically (2) managed VSD patients.

Atrioventricular block was uncommon and permanent pacing was rarely required in our series as well as in others (6).

Residual VSD. The prevalence of a residual VSD was 19.5% in the NHS-2 study (1), but reached 34% in other series (6). We did not find that residual VSDs were related to the early surgical experience, as previously reported (6). On the contrary, better imaging may have allowed better detection in recent years. Most defects were small and hemodynamically insignificant. Only 1 patient underwent closure of the residual defect and it was not the primary indication for the surgery. However, 11% of patients presented to us with a residual VSD requiring closure. Surgical indications were pulmonary hypertension in 4 patients, mitral regurgitation in 2 patients, and symptoms in 1 patient. The Q_p/Q_s ratio was above 2 to 1 in 1 patient who had a 40-mm defect. The others had defects between 5 and 10 mm, but 3 of them had an elevated pulmonary artery pressure or pulmonary vascular resistance. Of interest, a majority of our patients with a residual VSD at late follow-up had suture closure of the VSD between 1964 and 2007. This finding thus supports the use of patch closure. Late residual VSDs were less common in patients who had an IOTEE, supporting its routine use.

Valvular regurgitation and valve repair. The prevalence of AR (82%) was higher in our patients at follow-up than in Bol Raap et al. (7) series but similar to Otterstad et al. (6) series. A higher prevalence of STS type 1 VSDs in our series (26%) may explain this finding. Other potential associations with moderate or more AR at late follow-up include aortic valve or sinus of Valsalva repair, a dilated proximal aorta at follow-up, and lack of use of IOTEE. No patient had a residual VSD or a bicuspid aortic valve. Our limited data do not suggest that the severity of pre-operative AR, the closure technique, and the VSD size predict failure of the repair and development of moderate AR at follow-up, as found in larger series (17,18). In their study, van Son et al. (19) identified the presence of a subarterial VSD and a sinus of Valsalva to right ventricle shunt as risk factors for AR after repair of a ruptured sinus of Valsalva aneurysm.

TR was almost universal. Potential associations with more-than-moderate late TR include an STS type 2 VSD, the presence of a residual VSD, previous tricuspid valve repair, and lack of use of IOTEE. We observe no relation of moderate or more severe TR with the use of a patch for VSD closure. Peak TR velocities in patients with greater-than-moderate late TR were between 2.5 and 3.7 m/s. Tricuspid valve repair (11 patients) was performed exclusively in patients with a STS type 2 VSD. At late follow-up, TR decreased from severe to moderate in 2 patients; however, it progressed from moderate to severe in 1 patient despite repair. Late TR velocity, closure technique, and the presence of a residual VSD are not associated with failure of tricuspid valve repair in our series.

Ventricular size and function. The mean LV end-diastolic diameter at late follow-up in our cohort is within normal limits in adults (<54 mm) (14) as reported in a similar series (7). The mean early and late post-operative LVEF in our cohort are near normal and comparable to previous reports (7,20). In patients who had a VSD closed in childhood, LVEF is lower when compared with control subjects (20) and fractional shortening is abnormal in 22% (21). Potential causes of ventricular dysfunction after VSD closure include less advanced myocardial protection during the early surgical experience and long-standing volume loading when the defect is repaired as an adult (16,20).

Study limitations. Our study is retrospective and limited to a tertiary care center with expertise in adult congenital cardiology and surgery. Thus, our results may not be directly applicable to all practices. Follow-up after the surgical hospitalization was unavailable in 15% of patients. As it is the case in other series (5), heterogeneity is introduced in our study population by the fact that concomitant congenital or acquired structural heart lesions were the primary motivation for consideration of repair in 18 patients (39%). Most patients did not have 24-h Holter monitoring performed during follow-up, thus the occurrence of arrhythmias may be underestimated. Despite our best efforts, we cannot exclude that the 2 patients who died during late follow-up suffered sudden cardiac death. There were few patients who did not have IOTEE guidance during VSD closure, which limits the assessment of its incremental value. Patients operated on before major progress in cardiac surgery, such as cold potassium blood cardioplegia, are also under-represented, limiting meaningful comparisons between our early and contemporary experience.

Clinical implications. In our practice, common indications for VSD closure included AR, pulmonary hypertension, and associated defects, especially aortic valve cusp prolapse. This is in contrast to practice guidelines (4) that emphasize LV volume overload. Surgical closure of a VSD in adults can be accomplished with very low short- and long-term mortality and morbidity. Clinically significant ventricular arrhythmias were less prevalent in our series than previously reported and atrial fibrillation was more common. Aortic and tricuspid valve regurgitation were very common at follow-up. Significant AR occurred predominantly in patients who had closure of an STS type 1 VSD and repair of the aortic valve or sinus of Valsalva. The VSD patch closure appears to be the technique of choice given that residual VSDs occurred

more often after suture closure. We support the routine use of IOTEE guidance because it may lead to less late AR, TR, and residual VSDs. These data emphasize important residua and the need for life-long informed cardiology follow-up.

REFERENCES

1. Kidd L, Driscoll DJ, Gersony WM, et al. Second Natural History Study of congenital heart defects. Results of treatment of patients with ventricular septal defects. Circulation 1993;87:I38–51.
2. Neumayer U, Stone S, Somerville J. Small ventricular septal defects in adults. Eur Heart J 1998;19:1573–82.
3. Gabriel HM, Heger M, Innerhofer P, et al. Long-term outcome of patients with ventricular septal defect considered not to require surgical closure during childhood. J Am Coll Cardiol 2002;39:1066–71.
4. Warnes CA, Williams RG, Bashore TM, et al., on behalf of American Society of Echocardiography, Heart Rhythm Society, International Society for Adult Congenital Heart Disease, Society for Cardiovascular Angiography and Interventions, Society of Thoracic Surgeons. ACC/AHA 2008 guidelines for the management of adults with congenital heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Develop Guidelines on the Management of Adults With Congenital Heart Disease). J Am Coll Cardiol 2008;52:e1–121.
5. Mattila S, Kostiainen S, Kyllonen KE, Tala P. Repair of ventricular septal defect in adults. Scand J Thorac Cardiovasc Surg 1985;19:29–31.
6. Otterstad JE, Froysaker T, Erikssen J, Simonsen S. Long-term results in isolated ventricular septal defect surgically repaired after age 10. Comparison with the natural course in similarly-aged patients. Scand J Thorac Cardiovasc Surg 1985;19:221–9.
7. Bol Raap G, Meijboom FJ, Kappetein AP, Galema TW, Yap SC, Bogers AJ. Long-term follow-up and quality of life after closure of ventricular septal defect in adults. Eur J Cardiothorac Surg 2007;32:215–9.
8. Jacobs JP, Burke RP, Quintessenza JA, Mavroudis C. Congenital Heart Surgery Nomenclature and Database Project: ventricular septal defect. Ann Thorac Surg 2000;69:S25–35.
9. Warnes CA, Somerville J. Tricuspid atresia in adolescents and adults: current state and late complications. Br Heart J 1986;56:535–43.
10. Tajik AJ, Seward JB, Hagler DJ, Mair DD, Lie JT. Two-dimensional real-time ultrasonic imaging of the heart and great vessels. Technique, image orientation, structure identification, and validation. Mayo Clin Proc 1978;53:271–303.
11. Nishimura RA, Miller FA Jr., Callahan MJ, Benassi RC, Seward JB, Tajik AJ. Doppler echocardiography: theory, instrumentation, technique, and application. Mayo Clin Proc 1985;60:321–43.
12. Quinones MA, Waggoner AD, Reduto LA, et al. A new, simplified and accurate method for determining ejection fraction with two-dimensional echocardiography. Circulation 1981;64:744–53.
13. Rich S, Sheikh A, Gallastegui J, Kondos GT, Mason T, Lam W. Determination of left ventricular ejection fraction by visual estimation during real-time two-dimensional echocardiography. Am Heart J 1982;104:603–6.
14. Lang RM, Bierig M, Devereux RB, et al. Recommendations for chamber quantification. Eur J Echocardiogr 2006;7:79–108.
15. Yock PG, Popp RL. Noninvasive estimation of right ventricular systolic pressure by Doppler ultrasound in patients with tricuspid regurgitation. Circulation 1984;70:657–62.
16. Ammash NM, Warnes CA. Ventricular septal defects in adults. Ann Intern Med 2001;135:812–24.
17. Chiu SN, Wang JK, Lin MT, et al. Progression of aortic regurgitation after surgical repair of outlet-type ventricular septal defects. Am Heart J 2007;153:336–42.
18. Cheung YF, Chiu CS, Yung TC, Chau AK. Impact of preoperative aortic cusp prolapse on long-term outcome after surgical closure of subarterial ventricular septal defect. Ann Thorac Surg 2002;73:622–7.
19. van Son JA, Danielson GK, Schaff HV, Orszulak TA, Edwards WD, Seward JB. Long-term outcome of surgical repair of ruptured sinus of Valsalva aneurysm. Circulation 1994;90:II20–9.
20. Jablonsky G, Hilton JD, Liu PP, et al. Rest and exercise ventricular function in adults with congenital ventricular septal defects. Am J Cardiol 1983;51:293–8.
21. Meijboom F, Szatmari A, Utens E, et al. Long-term follow-up after surgical closure of ventricular septal defect in infancy and childhood. J Am Coll Cardiol 1994;24:1358–64.

Key Words: ventricular septal defect ■ adult congenital heart disease ■ cardiac surgery.