

## Surgical outcome of ventricular septal defect repair in Songklanagarind Hospital

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### Abstract:

**Surgical outcome of ventricular septal defect repair in Songklanagarind Hospital**

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We conducted a retrospective study of pediatrics with ventricular septal defect (VSD) that were surgically treated for correction between August 2001 and September 2004 in Songklanagarind Hospital, noting age at time of surgery, sex, type of defect, pulmonary arterial pressure, duration of stay in the intensive care unit and hospital, complications, and mortality. There were 93 patients with mean age  $7.7 \pm 4.2$  years. Perimembranous VSD was the most frequent in this study (57%) followed by subarterial VSD (40.9%) and muscular VSD (2.1%). Pulmonary hypertension (PHT) was found in 100% of the patients; it was severe in 7 patients (7.5%), moderate in 58 (62.4%) and mild in 28 (30.1%). The average duration of stay in the ICU

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and hospital were  $1.7\pm 1.7$  and  $6.9\pm 3.7$  days, respectively; both were significantly longer in the severe PHT group compared to the mild and moderate PHT groups ( $P<0.05$ ). Postoperative complications presented in 8 patients (8.6%), with 4 (4.3%) in the severe PHT group, 3 in the moderate group, and 1 in the mild group. Complete AV block was found in one patient in the severe PHT group. The overall mortality was 3.2% (2 in severe PHT, 1 in moderate PHT). We concluded that VSD with severe PHT continues to be a major cause of postoperative morbidity and mortality, and have increased the duration of ICU and hospital stay in these patients.

**Key words:** surgery, ventricular septal defect, pulmonary hypertension

### บทคัดย่อ:

**วัตถุประสงค์:** เพื่อศึกษาผลการผ่าตัดผู้ป่วยโรคหัวใจพิการแต่กำเนิดชนิดผนังหัวใจห้องล่างรั่วที่มีความดันเลือดในปอดสูง

**รูปแบบวิจัย:** พรรณนาแบบย้อนหลัง

**วัสดุและวิธีการ:** รวบรวมข้อมูลจากเวชระเบียนผู้ป่วยเด็กที่เข้ารับการผ่าตัดปิดผนังหัวใจห้องล่างรั่วแต่กำเนิด ตั้งแต่เดือนสิงหาคม พ.ศ. 2544 จนถึงเดือนกันยายน พ.ศ. 2547 โดยรวบรวมข้อมูลเกี่ยวกับลักษณะทางคลินิกของผู้ป่วย, การผ่าตัด, อัตราการเกิดภาวะแทรกซ้อน, อัตราการตาย, ระยะเวลาการอยู่โรงพยาบาลและหอผู้ป่วยหนัก, ผลการผ่าตัดในระยะสั้น

**ผลการศึกษา:** มีผู้ป่วยทั้งหมด 93 ราย เป็นชนิด perimembraneous ร้อยละ 57, ชนิด subarterial ร้อยละ 40, ชนิด muscular ร้อยละ 2.1 ภาวะความดันเลือดในปอดสูง พบว่าร้อยละ 7.5 อยู่ในภาวะรุนแรง, ร้อยละ 62.4 อยู่ในภาวะปานกลาง, ร้อยละ 30.1 อยู่ในภาวะเล็กน้อย ระยะเวลาการรักษาตัวในหอผู้ป่วยหนักและโรงพยาบาลโดยเฉลี่ยคิดเป็น  $1.7\pm 1.7$  วัน และ  $6.9\pm 3.7$  วัน ตามลำดับ ซึ่งกลุ่มที่มีภาวะความดันเลือดในปอดรุนแรงมีระยะเวลาการรักษาตัวในหอผู้ป่วยหนักและโรงพยาบาลยาวนานกว่าผู้ป่วยกลุ่มที่เหลืออย่างมีนัยสำคัญทางสถิติ ภาวะแทรกซ้อนหลังการผ่าตัดคิดเป็นร้อยละ 8.6 โดยร้อยละ 4.3 พบในกลุ่มที่มีความดันเลือดในปอดรุนแรง อัตราตายโดยรวมภายหลังการผ่าตัดคิดเป็นร้อยละ 3.2 (สองรายมีความดันในปอดสูงขั้นรุนแรง, หนึ่งรายขั้นปานกลาง)

**สรุป:** จากประสบการณ์การผ่าตัดในโรงพยาบาลสงขลานครินทร์ พบว่าโรคผนังหัวใจห้องล่างรั่วแต่กำเนิดที่มีความดันเลือดในปอดสูงมีความเสี่ยงต่อการผ่าตัด อีกทั้งระดับความรุนแรงของความดันเลือดในปอดที่สูงขึ้นเป็นสาเหตุสำคัญอย่างหนึ่งต่ออัตราการเสียชีวิตภายหลังการผ่าตัด

**คำสำคัญ:** ผลการผ่าตัด, ผนังหัวใจห้องล่างรั่ว, ภาวะความดันเลือดในปอดสูง

### Introduction

Ventricular septal defect (VSD) is the most common congenital heart defects, accounting for 25% to 30% of all children born with structural heart disease.<sup>1,3</sup> Such defects occur both in isolation and as part of most complex forms of congenital heart disease. Isolated VSD occurs in approximately 2 out of every 1,000 live births.<sup>2</sup> In the VSDs that need closure, surgical treatment is standard and aimed at prevention of pulmonary hypertension (PHT), heart failure, endocarditis, or, in some instances, progressive aortic valve insufficiency (AI).<sup>3</sup> Kirklin and colleagues described VSD closure using

extracorporeal circulation in 1957<sup>4</sup>, although it still entails both morbidity and mortality risks. The purpose of this study was to review the results of surgical closure of isolated VSD with pulmonary hypertension in patients under 15 years of age in Songklanagarind Hospital.

### Materials and methods

The study was conducted in a tertiary hospital serving a population of approximately 10 million people in southern Thailand. Approval for retrospective chart review was obtained

from the hospital's medical records division. Between August 2001 and September 2004, 104 patients were operated upon for closure of a congenital VSD. This study was restricted to patients under 15 years of age with isolated VSD (N= 93).

#### Preoperative evaluation

All patients had standard preoperative evaluation according to the requirements of the surgical procedure and based on institutional policy regarding preoperative diagnostic work up requiring consensus between the cardiac surgeons and pediatric cardiologists. The diagnostic evaluation consisted of complete two-dimensional and color Doppler echocardiography in all patients. Echocardiography for tricuspid or pulmonary regurgitation velocity measurements and pulsed Doppler were performed to determine the presence of pulmonary hypertension. Patients were classified into 3 groups: (i) severe PHT—systolic pulmonary artery pressure of 80% or more of the systolic systemic arterial pressure (Pp/Ps > 0.8), (ii) moderate PHT—Pp/Ps = 0.5–0.8, and, (iii) mild PHT—Pp/Ps = 0.3–0.49. Based on these data, the team of surgeons and cardiologists evaluated each case on the need for preoperative cardiac catheterization (e.g. severe pulmonary hypertension and/or indefinite diagnosis). Surgery was indicated in patients whose pulmonary/systemic blood flow (Qp/Qs) was 1.5 or greater.<sup>2-3</sup>

#### Surgical protocol

All patients were operated on using conventional cardiac surgical techniques. Under general anesthesia and via a median sternotomy, standard ascending aortic and bicaval venous cannulation were performed. Cardiopulmonary bypass with systemic hypothermia to 30°C was used. Myocardial protection was achieved by using antegrade cold blood /or crystalloid cardioplegia. In cases with AI, the aorta was opened for aortic valve repair and additional cardioplegia was delivered directly into the coronary ostia to supplement the initial arresting dose. The VSD was closed with a synthetic patch (Dacron®) using a transatrial approach for a perimembranous, muscular type, and using a transpulmonary artery approach for a subpulmonary type. After surgery, all patients were managed in ICU. Postoperative echocardiographic evaluation was performed in all patients.

#### Outcomes analysis

Outcome variables for this study were the following: (1) 30-day mortality rate, (2) duration of hospital stay, (3) duration of ICU stay, (4) postoperative ventilator time, and (5) postoperative echocardiographic result and complications. The SPSS for Windows statistical package (release 10.0, SPSS Inc., Chicago, IL, USA) was used to analyze the results. A significant difference was considered to be a probability value smaller than 0.05.

### Results

#### Patient data

The average age at the time of operation was 7.7±4.2 years (range 1–15 years) and mean body weight was 23.2±13.0 kg (range 6–70 kg). The incidence of types of ventricular septal defect were: 53 patients (57%) with perimembranous VSD, with subarterial VSD 38 (40.9%), and with muscular VSD 2 (2.2%). The most common associated defects were aortic valve insufficiency in 27 patients (29.1%); mild AI in 16, moderate AI in 4 and severe AI in 7. A subaortic ridge was found and excised in 12 patients (13%). Patients with moderate to severe AI underwent aortic valvoplasty in addition to closure of the defect. Patients' characteristics and operative data in each degree of pulmonary hypertension are described in Table 1. Table 2 demonstrates postoperative data, complications, mortality rate, and residual shunt in each group.

#### Postoperative complications

Eight patients (8.6%) had postoperative complications. One patient with severe PHT had complete heart block (1.1%). Two patients required reoperation within 7 days because of severe aortic valve regurgitation after VSD closure with aortic valve repair (moderate PHT case), another with mild PHT patient was operated on because of right ventricular outflow tract obstruction from missing of diagnostic defect. Respiratory tract infection (pneumonia) was noted in 2 cases with severe PHT, and in 1 case with moderate PHT. Postoperative cardiac arrhythmia (premature ventricular contraction) was found in 2 cases (1 in moderate PHT, 1 in severe PHT) and disappeared before discharge.

**Table 1 Comparison of preoperative characteristics and operative data between patients with mild, moderate, and severe pulmonary hypertension (total =93 cases)**

Variables	Mild PHT* (N=28)	Moderate PHT (N=58)	Severe PHT (N=7)	P-value***
<b>Demographic data</b>				
Age (years)	8.1 ± 4.2	7.9 ± 4.3	4.0 ± 2.9	P > 0.05
Sex (male:female)	20:8	33:25	3:4	
Body weight (kg)	24.9 ± 12.5	24.1 ± 13.1	9.6 ± 2.3	P < 0.05
Height (cm)	124.0 ± 24.1	121.8 ± 25.9	84.3 ± 13.9	P < 0.05
<b>Type of VSD (cases)</b>				
Perimembraneous type	12	36	5	
Subarterial type	15	22	1	
Muscular type	1	0	1	
<b>Associated defect (cases)</b>				
AI**	9	16	0	
Subaortic ridge	4	6	0	
AI & Subaortic ridge	1	1	0	
<b>Operative data</b>				
Total operative time (min)	168.7 ± 34.5	163.2 ± 36.5	162.1 ± 42.9	P > 0.05
Cardiopulmonary bypass time (min)	72.7 ± 24.0	73.6 ± 25.0	71.1 ± 7.6	P > 0.05
Aortic cross-clamp time (min)	50.9 ± 19.0	47.6 ± 19.4	56.2 ± 19.3	P > 0.05

\*PHT, Pulmonary hypertension;

\*\*AI, Aortic valve insufficiency;

\*\*\*P-value, compared between severe PHT group and mild-moderate PHT group

**Table 2 Comparison of the postoperative variables between patients with mild, moderate, and severe pulmonary hypertension (total = 93 cases)**

Variables	Mild PHT (N=28)	Moderate PHT (N=58)	Severe PHT (N=7)	P-value*
<b>Postoperative data</b>				
Ventilator time (hours)	4.1 ± 3.9	10.0 ± 25.3	38.4 ± 27.3	P < 0.05
ICU stay (days)	1.3 ± 0.9	1.5 ± 1.3	5.0 ± 3.5	P < 0.05
Postoperative hospital stay (days)	5.9 ± 1.9	7.1 ± 3.6	10.2 ± 8.2	P < 0.05
Complications (cases)	1	3	4	
30-day mortality rate (cases)	0	1	2	
<b>Echocardiographic data</b>				
Residual VSD (cases)	8	7	2	
Ejection fraction (%)	60.1 ± 9.0	58.6 ± 12.5	57.5 ± 15.4	P > 0.05

\*P-value, compared between severe PHT group and mild-moderate PHT group

### Mortality rate

The overall in-hospital mortality rate for these 93 patients was 3.2%. One patient with severe PHT died 4 days after surgery from pulmonary hypertensive crisis and low cardiac output, and another died suddenly from cardiac arrhythmia on postoperative day 6. One patient with moderate PHT died 7 days after surgery from cardiac arrhythmia. All 90 hospital survivors were followed up for  $11.7 \pm 9.4$  months on average (range, 2–33 months) by physical examination, ECG, chest x-ray and echocardiography. All of the children gained weight and improved functional class. In this follow-up period, no patients died.

### Operative results

Echocardiography showed a residual shunt in 17 patients (18.3%), which was not hemodynamically important and required no reoperation. They had a trivial residual shunt across the septum ( $Qp/Qs < 1.5$ ). The postoperative echocardiographic assessment of cardiac function (ejection fraction) was  $59.0 \pm 11.6$  % (range, 21–77%).

### Discussion

Ventricular septal defect is by far the most common congenital heart defect performed in pediatric cardiac surgery, and surgical closure of a VSD is the most common open-heart procedure.<sup>1–3</sup> The main objective of the present study was to evaluate management of children with isolated VSD, particularly the result of surgery in relation to the degree of pulmonary hypertension. Surgical repair normally provides excellent results with a low mortality rate, however the operative risk may be higher in patients with severe pulmonary hypertension.<sup>2–3, 5–6, 12</sup> The pathophysiology of VSD is related to the size of the defect and the pulmonary vascular resistance. In our study, patients with severe PHT were younger than other groups and their defects were large VSDs, so they developed heart failure and pulmonary hypertension earlier. This study demonstrates postoperative results and complications after VSD closure in patients with severe pulmonary hypertension compared to mild-moderate pulmonary hypertension. This study

shows that patients with severe pulmonary PHT have longer duration ICU and hospital stays than do patients in the mild-moderate PHT group. 4.3% of all patients who had postoperative complications occurred in the severe PHT group. Our study confirms that a higher degree of pulmonary hypertension increases morbidity.

Pulmonary hypertension is an important determinant of morbidity and mortality in many diseases, including congenital heart disease (left to right shunt) and respiratory disease.<sup>2–3, 5–6, 9–12</sup> The most crucial factor in determining late outcome is the age at which repair is carried out.<sup>13</sup> Most children operated upon by 9 months of age have a normal pulmonary vascular resistance one year after repair. After 2 years of age, resistance may fall, but not to a normal level. To avoid this situation, early closure within 1–2 years of birth has been recommended.<sup>3, 9, 12</sup> In our institute the treatment of pulmonary hypertension during intra- and postoperative pediatric surgery is based on the use of high inspired oxygen concentration (100%) with hyperventilation, induced alkalosis, inotropic support, adequate sedation, and the use of vasodilators. Previous clinical studies showed that nitric oxide plays an important role in the management of the postoperative pulmonary hypertensive crisis,<sup>9–12</sup> but inhaled nitric oxide is not available in our institute. Day et al.<sup>11</sup> reported that nitric oxide did not substantially improve pulmonary hemodynamics and gas exchange immediately after operation for congenital heart disease. Miller and colleagues<sup>10</sup> demonstrated that routine use of inhaled nitric oxide after congenital heart surgery in patients with pulmonary hypertension can lessen the risk of pulmonary hypertensive crisis and shorten the postoperative course, with no toxic effect. Because the patient with severe PHT must be on adequate sedation and ventilation support, the duration of mechanical ventilation and ICU stay may be longer than in patients with mild or moderate PHT. Use of nitric oxide will support postoperative care and decrease the duration of ICU stay in our institute.

In nearly all cases, accurate repair of a perimembranous VSD can be achieved through a right atrial approach, across the tricuspid valve. This approach has been used almost exclusively in our institute because the incidence of complete

heart block is lower than that seen with the transventricular approach.<sup>2-3</sup> Because a subarterial VSD may be difficult to close via the right atrium, this defect is closed by a transpulmonary artery approach with retraction of its leaflets. In our series, aortic valve insufficiency was found in 27 cases and required aortic valve repair in 11 cases. We advocate closure of VSDs, regardless of the type, size, or degree of aortic valve prolapse, when any degree of AI is identified.<sup>7-8</sup> We also close all subarterial VSD associated with aortic valve prolapse, even in the absence of AI, because the likelihood of spontaneous VSD closure is low.<sup>3-7</sup>

With echocardiography, small residual shunts are frequently noted after repair of a VSD.<sup>13-14</sup> In our study, we found an incidence of 18.3% in this regard. However, the great majority of these are hemodynamically small ( $Q_p/Q_s < 1.5:1$ ), and often close spontaneously. Bol-Raap et al showed that the median time of echocardiographically-proven spontaneous closure was 3.9 years and trivial residual shunting was expected to disappear spontaneously.<sup>13</sup> Reoperation for a residual VSD was only 0.7-2%. In our study, none of the patients had significant residual leakage across the repaired defect. Reoperation because of patch leakage was not performed in this study, which was similar to report in other studies because they were not significant hemodynamic. This patient with residual VSD should receive long-term annual evaluation. Conduction disturbance and/or arrhythmia of cardiac rhythm are frequent after repair of VSDs; however, most are benign. The incidence of complete heart block ranged from 0.2-2.3% in various studies.<sup>2, 5, 13</sup> In this study, we found complete heart block in one case (1.1%), consistent with those other studies.

The outcomes of surgical corrective procedures to repair ventricular septal defects and their associated abnormalities have significantly improved in recent years. Earlier correction of operable defects can avoid the later development of pulmonary vascular disease.<sup>2, 13-14</sup> Our study reports that closure of a ventricular septal defect (VSD) in children with elevated pulmonary vascular resistance is associated with significant morbidity and mortality, especially in the patient with severe pulmonary hypertension. Pulmonary hypertensive episodes continue to be a major cause of postoperative morbidity and

mortality. Due to improvements in surgical technique, myocardial protection and postoperative care, surgical outcomes have improved markedly. Our study shows that the short-term follow-up was also good and this information will help us improve the quality of surgical care in the future. Until now the most important factor affecting the surgical treatment of ventricular septal defect (VSD) has been pulmonary hypertension.

## Conclusion

**This study supports our recommendation that primary repair should be performed as soon as possible, and before development of severe pulmonary hypertension.**

## References

1. Bernstein D. Epidemiology of congenital heart disease. In: Behrman RE, Kleigman RM, Arvin AM, editors. Nelson Textbook of Pediatrics. 15th ed. Philadelphia: WB Saunders; 1996:1286-7.
2. Tchervenkov CI, Shum-Tim D. Ventricular septal defect. In: Baue A, Geha AS, Hammond GL, Laks H, Naunheim KS, editors. Glenn's Thoracic and Cardiovascular Surgery. Stanford, CT: Appleton & Lange, 1996:1127-36.
3. Castaneda AR, Jonas AR, Mayer JE, Hanley FL. Ventricular septal defect. In: Castaneda AR, editor. Cardiac Surgery of the Neonate and Infant. Philadelphia: WB Saunders; 1994:187-201.
4. Kirklin JW, Harshbarger HG, Donald DE, Edwards JE. Surgical correction of ventricular septal defect: anatomic and technical considerations. J Thorac Cardiovasc Surg 1957;33:45-9.
5. Cartmill TB, DuShane JW, McGoan DC, Kirklin JW. Results of repair of ventricular septal defect. J Thorac Cardiovasc Surg 1966;52:486-50.
6. Clarkson PM, Frye RL, DuShane JW, Burchell HB, Wood EH, Weidman WH. Prognosis for patients with ventricular septal defect and severe pulmonary vascular obstructive disease. Circulation 1968;38:129-35.
7. Yacoub MH, Khan H, Stavri G, Shinebourne E, Radley-

- Smith R. Anatomic correction of the syndrome of prolapsing right coronary aortic cusp, dilatation of sinus of valsalva, and ventricular septal defect. *J Thorac Cardiovasc Surg* 1997;113:253-61.
8. Cheung Y-F, Chiu CSW, Yung T-C, Chau AKT. Impact of preoperative aortic cusp prolapse on long-term outcome after surgical closure of subarterial ventricular-septal defect. *Ann Thorac Surg* 2002;73:622-27.
9. Hopkins RA, Bull C, Haworth SG, de Leval MR, Stark J. Pulmonary hypertensive crises following surgery for congenital heart defects in young children. *Eur J Cardiothorac Surg* 1991;5:628-34.
10. Miller OI, Tang SF, Keech A, Pigott NB, Beller E, Celermajer DS. Inhaled nitric oxide and prevention of pulmonary hypertension after congenital heart surgery: a randomised double-blind study. *Lancet* 2000;356:1464-9.
11. Day RW, Hawkins JA, McGough EC, Crezee KL, Orsmond GS. Randomized controlled study of inhaled-nitric oxide after operation for congenital heart disease. *Ann Thorac Surg* 2000;69:1907-13.
12. Gorenflo M, Nelle M, Schnabe PA, Ullmann MV. Pulmonary hypertension in infancy and childhood. *Cardiol Young* 2003;13:219-27.
13. Bol-Raap G, Weerheim J, Kappetein AP, Withsenburg M, Bogers AJ. Follow-up after surgical closure of congenital ventricular septal defect. *Eur J Cardiothorac Surg* 2003;24:511-15.
14. Roos-Hesselink JW, Meijboom FJ, Spitaels SE, van Domburg R, van Rijen EH, Utens EM, et al. Outcome of patients after surgical closure of ventricular septal defect at young age: longitudinal follow-up of 22-34 years. *Eur Heart J* 2004;25:1057-62.