DECISION MAKING FOR THE SURGICAL MANAGEMENT OF AORTIC COARCTATION ASSOCIATED WITH VENTRICULAR SEPTAL DEFECT

Coarctation of the aorta and associated ventricular septal defect may be repaired simultaneously or by initial coarctation repair with or without banding of the pulmonary artery. The question is whether specific preoperative criteria can enable the surgeon to choose the optimal surgical management. Between 1980 and 1993, 80 infants younger than 3 months with coarctation and ventricular septal defect were treated surgically. In 64 infants (multistage group), simple coarctation repair was performed through a posterolateral approach, with concomitant banding of the pulmonary artery in 10 infants. Twenty ventricular septal defects were closed as a secondary procedure and four were closed as a tertiary procedure. Sixteen infants (single-stage group) underwent one-stage repair through an anterior midline approach. The total in-hospital mortality rate was 7.5%. Freedom from recoarctation after 5 years was 91.3% in the multistage group versus 60.0% in the single-stage group ($p = 0.018$). Freedom from secondary ventricular septal defect treatment in the multistage group after 5 years was 40.7%, versus 100% in the single-stage group ($p = 0.016$). Thirty-seven ventricular septal defects (47.8%) closed spontaneously. In particular, the preoperative left-to-right shunt and extension of the perimembranous VSD into the inlet or outlet were risk factors for the need for eventual surgical ventricular septal defect closure after initial coarctation repair. On the basis of these two risk factors, the probability of the need for eventual surgical treatment of ventricular septal defect after initial coarctation repair can be calculated. This policy offers a well-considered choice between single-stage and multistage repair, weighing the risk of secondary ventricular septal defect treatment versus the risk of recoarctation. Finally, the number of surgical procedures per infant will be as low as possible. (J THORAC CARDIOVASC SURG 1996;111:168-75)

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The optimal surgical management of aortic coarctation (CoA) with associated ventricular septal defect (VSD) in infants with cardiac failure remains

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a matter of debate. These infants may be treated by different therapeutic strategies; single-stage total repair, initial CoA repair with concomitant pulmonary artery banding (PAB), or CoA repair alone with initial conservative treatment of the VSD.¹⁻⁵

Are there specific preoperative criteria that enable the surgeon to select the optimal surgical management in this pathophysiologic setting? The purpose of this retrospective study was to find such preoperative criteria to optimize surgical decision making in infants with CoA and VSD.

Patients and methods

Between 1980 and 1993, 80 infants younger than 3 months who had CoA associated with a VSD were

operated on at the Divisions of Cardiothoracic Surgery of the University Hospital Groningen and the Dijkzigt University Hospital Rotterdam. The therapeutic strategies in the two hospitals were different, changed with time, and were subject to the personal preference of six different surgeons.

The study included 48 boys (60%) and 32 girls. Mean age at operation was 29 days (standard deviation [SD] $\pm$ 23 days), and mean weight was 3386 gm (SD $\pm$ 646 gm). All infants were treated with diuretics and digitalis; 30 infants (39%) were critically ill and needed prostaglandin E₁ or inotropic medication. The diagnosis of CoA, the morphologic characteristics of the VSD, and the presence of any coexisting anomalies were determined by physical examination, cross-sectional echocardiography, cardiac catheterization, and operative reports. In the last few years, cardiac catheterization was performed only when the echocardiographic diagnosis was not conclusive. Coexisting anomalies were hypoplastic aortic arch in 27 infants (33.8%) and aortic valve stenosis in seven (8.8%). The hemodynamic and oximetric data of those who underwent cardiac catheterization are presented in Table 1.

In 64 infants only the CoA was operated on, with the VSD left to be treated at a later stage if necessary. These infants constituted the multistage group. Through a left posterolateral thoracotomy, the CoA and the isthmus were resected. An end-to-end anastomosis was performed in 30 infants (46.9%), a left subclavian flap angioplasty was performed in 28 (43.8%), a subclavian artery–descending aorta anastomosis was performed in three (4.7%), and a patch angioplasty was performed in three (4.7%). Hypoplastic aortic arches were left undisturbed.⁵

PAB was performed at the time of CoA repair in 10 infants (15.6%).^{6,7} Four of these eventually underwent debanding and closure of the VSD as a secondary procedure; four others required debanding alone because the VSDs had become hemodynamically insignificant. The last two infants are scheduled to undergo debanding without VSD closure for the same reason.

Twenty-three infants could not be weaned from the ventilator after simple CoA repair as a result of persistent pulmonary overflow: 20 of these underwent subsequent closure of the VSD and the other three underwent PAB as a secondary procedure. Two other infants, who were weaned from the ventilator successfully, required PAB as a secondary procedure at 3 and 7 days after extubation because of persistent dyspnea. Of the five infants who underwent PAB as a secondary procedure, three finally underwent debanding and VSD closure; the other two needed debanding alone, without closure of the VSD. In total, 34 of the 64 patients with VSD (53.1%) needed surgical treatment of the VSD, whether secondary or tertiary closure of the VSD or PAB as a primary or secondary procedure (Fig. 1).

The other group of 16 infants were operated on through an anterior midline approach, with extracorporeal circulation, deep hypothermic circulatory arrest, and crystalloid cardioplegia, to correct the CoA and the VSD in a single-stage procedure. The CoA was resected, and an end-to-end anastomosis was performed in 15 infants (93.7%) and a patch angioplasty was performed in one.

Table 1. Preoperative hemodynamic profile in infants undergoing repair of CoA associated with a VSD

	Pressure (mm Hg)	O ₂ saturation (%)
Left atrium	11 $\pm$ 4	94 $\pm$ 5
Left ventricle (systolic)	91 $\pm$ 19	94 $\pm$ 4
Left ventricle (diastolic)	9 $\pm$ 5	
Right atrium	6 $\pm$ 3	76 $\pm$ 10
Right ventricle (systolic)	82 $\pm$ 19	83 $\pm$ 10
Right ventricle (diastolic)	8 $\pm$ 13	
Pulmonary artery (systolic)	81 $\pm$ 20	85 $\pm$ 8
Pulmonary artery (diastolic)	32 $\pm$ 9	
Ascending aorta		91 $\pm$ 8
Descending aorta		91 $\pm$ 6

Data are mean $\pm$ SD.

All 16 VSDs were closed through the right atrium. These infants constituted the single-stage group.

Follow-up. To follow up on the infants, we saw them 2 weeks after discharge from the hospital, 3 months later, and then yearly. No patient was unavailable for follow up. Mean follow-up duration was 6.0 years (SD 4.3, range 1 day to 14.3 years). Special attention was given to systolic blood pressure in arms and legs and to femoral pulsations. Radiographs of the chest were obtained and electrocardiography was performed to exclude left ventricular hypertrophy or signs of pulmonary overflow. Cross-sectional echocardiography was performed to exclude recoarctation and to evaluate the hemodynamic importance, if any, of the VSD. If these data suggested a recoarctation, cardiac catheterization was performed. Recoarctation was defined by an arm-to-leg systolic pressure gradient exceeding 20 mm Hg across the repaired area.

Statistical analysis. All data were summarized in contingency tables, and 70% confidence limits (CL 70%) of binomial distributions were calculated. One-way analysis of variance was performed. To determine incremental risk factors, multivariate stepwise logistic regression analysis were performed (see appendix). All *p* values less than 0.05 were considered significant. Time-related analysis were performed according to the Kaplan-Meier method.

Results

Mortality and morbidity. In total, eight infants (10%) died in our series. The in-hospital (<30 days) mortality comprised six infants (7.5%), three in the multistage group (4.7%, CL 70% 2.1% to 9.2%) and three in the single-stage group (18.8%, CL 70% 8.4% to 34.2%). This difference in in-hospital mortality rate was not statistically significant. None of these six infants could be weaned from the ventilator and all died of heart failure.

Late mortality comprised two infants of the multistage group; they died 42 days and 1 year after CoA repair of right ventricle failure and lung embolism, respectively. Paraplegia and other neuro-

Fig. 1. Diagram showing all the procedures for VSD after the initial CoA repair.

logic complications were not seen in our series. Permanent heart block was seen in one infant.⁸⁻¹⁰

Hemodynamic characteristics of the VSD. Fifty infants underwent complete cardiac catheterization before CoA repair. In another three infants, only arch angiography was done. To quantify the hemodynamic characteristics of the VSD, the total left-to-right shunt as a percentage of total pulmonary flow and the ratio between the right ventricular peak systolic pressure and the left ventricular peak systolic pressure (RV/LV) as a parameter for pressure loading between the ventricles were calculated in 50 infants. The mean left-to-right shunt in these 50 infants was 60.4% (SD $\pm$ 16.2%, range 22% to 89%), and the mean RV/LV ratio was 0.94 (SD $\pm$ 0.165, range 0.37 to 1.4). In 40 of these 50 infants, the VSD was large; the left-to-right shunts were more than 50% and the right ventricular peak systolic pressures approached or even exceeded the left ventricular peak systolic pressure. In seven infants the VSD was of moderate size; the left-to-right shunt was almost 50%, the right ventricular peak systolic pressure was raised to approximately half the left ventricular pressure. In three infants, the VSD was small; the left-to-right shunt was less than 30% and there was no rise in right ventricular peak systolic pressure. The mean left-to-right shunt of the multistage and single-stage groups were 58% (SD $\pm$ 16%) and 68% (SD $\pm$ 14%), respectively

($p = 0.108$). The mean RV/LV ratios were 0.91 (SD $\pm$ 0.18) and 0.97 (SD $\pm$ 0.92, $p = 0.07$).

Twenty-seven infants underwent two-dimensional echocardiography and Doppler flow mapping. The modified Bernoulli equation was used to calculate a pressure drop across the VSD between the left and right ventricle (pLVRV): $pLVRV = 4(V_2^2 - V_1^2)$, where V_1 is the flow (in meters per second) in front of the VSD and V_2 is the flow velocity in the jet behind the VSD. The mean pLVRV was 7.0 mm Hg (SD 15, range 0 to 81 mm Hg). Twenty of these 27 infants (74%) had pLVRVs of less than 10 mm Hg, and their VSDs were considered to be nonrestrictive. The mean pLVRV of the multistage group was significantly higher (10.5 ± 18 mm Hg) than that of the single-stage group (5 ± 8 mm Hg, $p = 0.027$).

Finally, 13 of the 80 infants in our series underwent two-dimensional echocardiography and cardiac catheterization. In this group the pLVRV, the RV/LV ratio, and the left-to-right shunt could all be calculated. Correlation analysis between the RV/LV ratio and the pLVRV yielded a coefficient of 0.93 ($p < 0.001$). The linear regression equation was as follows: RV/LV ratio = $1.02731 - (0.008221 \times pLVRV)$. Correlation analysis between the left-to-right shunt and the pLVRV yielded a coefficient of 0.93 ($p = 0.038$). The linear regression equation was as follows: left-to-right shunt = $74.20604 - (0.381676 \times pLVRV)$.

VSD morphology, surgery, and follow-up. Of the 80 VSDs, 48 were perimembranous defects (60%): 28 were simple, 10 extended into the outlet septum, seven extended into the inlet septum, and three extended into both inlet and outlet septa. Twenty-one defects were muscular defects, eight were multiple, and three were malaligned and situated in the outlet septum.

In the multi-stage group, 21 of the 37 perimembranous defects (56%, CL 70% 46% to 66%) required surgical closure after the initial CoA repair. Of the 27 muscular VSDs, six defects required surgical closure (22%, CL 70% 13% to 33%, $p = 0.005$).

Of the 37 perimembranous VSDs, 23 defects (68%, CL 70% 52% to 71%) were described as simple. Ten of these 23 VSDs eventually required surgical closure. Of the other 14 VSDs that extended into the inlet or outlet septum, 11 defects (78%, CL 70% 61% to 90%) required eventual surgical closure ($p = 0.036$).

In total, 43 defects of the 80 (53.8%) were closed surgically. The other 37 VSDs (47.8%) closed spontaneously or became small and hemodynamically insignificant.

Incremental risk factors in the multistage group. To find incremental risk factors for the necessity of surgical treatment of the VSD in the multistage group, age and weight at operation, body surface area, left-to-right shunt, RV/LV ratio, pLVRV, and postoperative ventilation time were entered into a multivariate stepwise logistic regression analysis. The analysis found the left-to-right shunt to be the only incremental risk factor for the necessity of surgical closure of the VSD after the initial CoA repair (Fig. 2, A). The influences of the morphologic characteristics of the VSD, in particular the perimembranous VSD with extension into the inlet or outlet septum, are illustrated in Fig. 2 (B). At a left-to-right shunt of 50%, the necessity of surgical closure of the VSD after initial CoA repair in the perimembranous VSD with extension into the inlet or outlet septum is more than double that seen with a simple perimembranous VSD.

Freedom from recoarctation and secondary surgical VSD treatment. Recoarctation was found in 13 of the 80 patients (16.3%, CL 70% 12% to 22%), nine times in the multistage group and four times in the single-stage group. It was found in six of the 45 infants after resection and end-to-end anastomosis (13.3%, CL 70% 8% to 21%), five of the 28 infants after subclavian flap angioplasty (17.9%, CL 70%

10% to 28%), and two of the four infants after patch angioplasty (50%, CL 70% 18% to 82%). The differences in the prevalence of recoarctation between resection and end-to-end anastomosis and subclavian flap angioplasty were not significant. Percutaneous balloon angioplasty was performed 10 times for recoarctation, and reoperation was performed three times.

The freedom from recoarctation in the multistage group after 5 years was 91.3%, whereas the freedom from recoarctation in the single-stage group for the same period was significantly lower (60.0%, $p = 0.018$; Fig. 3). The freedom from secondary treatment with respect to the VSD (including secondary VSD closure, secondary PAB, and debanding and surgical closure of the VSD) after 5 years in the multistage group was 40.7%, versus 100% in the single-stage group ($p = 0.016$; Fig. 4).

In the multistage group ($n = 64$), 111 procedures in total were performed: 54 CoA repairs and 10 CoA repairs with concomitant PAB. Thirty-three infants required a secondary surgical procedure (52%, CL 70% 44% to 59%): 20 secondary surgical closures of the VSD, five secondary PABs, four secondary VSD surgical closures with debanding, and four secondary debandings. Five infants required a tertiary surgical procedure (8%, CL 70% 4% to 13%): three debandings with VSD surgical closure, and two debandings. Six infants underwent percutaneous balloon dilation angioplasty and three underwent reoperation for recoarctation. Of the 64 infants 17 (26.5%) underwent only one procedure. Five infants in this group died, so at the latest follow-up, 59 infants (92%) are well after 1.73 procedures per infant.

In the single-stage group, 21 procedures were performed: 16 CoA repairs with concomitant VSD closure and one implantation of a ventricular inhibited pacemaker as a secondary procedure because of a permanent heart block. Four percutaneous balloon dilation angioplasty procedures for recoarctation were performed. Three patients died in this group, so 13 infants (81%) are well after 1.31 surgical procedures per infant. There was no statistical difference between the number of procedures per infant in the multistage and that in single-stage group ($p = 0.11$).

Discussion

It is considered an established fact that the CoA, which is judged to be the dominant lesion in the morphologic setting of CoA with associated VSD,

Fig. 2. A, Nomogram of the multivariate equation for the incremental risk factors for surgical VSD treatment (including VSD closure and PAB and debanding with VSD closure) after CoA repair according to the left-to-right shunt in the total group (intercept -3.2704 , logarithmic coefficient 0.0521 , $p = 0.0027$). **B,** Nomogram of the multivariate equation for the incremental risk factors for surgical VSD treatment after CoA repair with respect to the left-to-right shunt and the morphologic type of the VSD. Note the influence of the inlet or outlet perimembranous VSD. *peri*, Perimembranous.

should be repaired promptly. The discussion now centers on the question of whether it is reasonable to repair the CoA alone and then follow the post-operative course or perform CoA repair and VSD closure as a single-stage procedure.¹¹⁻²¹ It would be ideal if one could predict which VSDs require immediate surgical closure and which VSDs can be treated conservatively. In our series, multivariate

logistic regression analysis showed the preoperative left-to-right shunt to be the only hemodynamic risk factor for the necessity of eventual surgical treatment of the VSD (including secondary closure of the VSD, PAB, and debanding with closure of the VSD (Fig. 2, A).

Currently, it is unknown whether any specific morphologic type of VSD associated with CoA

Fig. 3. Actuarial freedom from recoarctation stratified into the multistage and single-stage groups. *SE*, Standard error.

Fig. 4. Actuarial freedom from secondary surgical treatment of VSD, secondary PAB, and debanding with surgical closure of the VSD stratified into the multistage and single-stage groups. *SE*, Standard error.

should be considered a surgical risk factor. Anderson, Lenox, and Zuberbuhler²² found that the majority of hearts with both VSD and CoA had a particular form of perimembranous defect which could extend into all parts of the muscular ventricular septum with aortic overriding. Furthermore, this necropsy study suggested that perimembranous defects undergo spontaneous closure in most cases, confirmed by others.^{22, 23} In contrast, we found that the incidence of surgical VSD closure after initial

CoA repair was highest in the “perimembranous” group. Furthermore, the influence of the morphologic category was even more marked when we stratified the perimembranous VSDs into different locations; the nomogram shows that the probability of eventual surgical treatment of a simple perimembranous VSD with a left-to-right shunt of 50% is 18%, whereas a perimembranous VSD with extension into the inlet or outlet septum has a probability of 42% for the same left-to-right shunt (Fig. 2, B). It

therefore seems that the perimembranous VSD with extension into the inlet or outlet septum in particular should be considered another risk factor for the necessity of surgical VSD closure after initial CoA repair.

Surprisingly, the freedom from recoarctation in the multistage group after 5 years was significantly higher than in the single-stage group (Fig. 3). Age and weight did not differ significantly between the two groups and therefore cannot explain this difference in freedom from recoarctation.²⁴ A possible explanation for this phenomenon might be the relationship between recoarctation and the presence of residual ductal tissue in the aorta after the resection of the CoA. In a histologic examination, Russell and associates²⁵ found that one or two tongue-like prolongations of ductal tissue extend distally from the coarctic shelf and occupy constant positions in the aortic wall. To eradicate the risk of recoarctation, the CoA should be resected widely. Even after apparently adequate and deliberately radical resection of the CoA through a posterolateral approach, however, ductal tissue can still be left behind.²⁵ In our series, the risk for recoarctation in the single-stage group was high. An anterior midline approach for this morphologic entity may impose an extra risk factor for recoarctation in a group of infants already likely to have recoarctation because of their young age and low weight.²⁴⁻²⁶ Nevertheless, this may be taken as a calculated risk under the argument that balloon dilation of recoarctation is to be preferred to the risk of an extra operative procedure.^{27, 28}

These clinical findings are of utmost importance because they offer the surgeon new perspectives in surgical decision making for CoA with associated VSD. Currently, two-dimensional echocardiography provides the surgeon the precise description of the anatomy and estimates the pressure drop (pLVRV) across the VSD. Because the correlation between the pLVRV and the left-to-right shunt was almost perfect in this study, it is possible to estimate the left-to-right shunt without further cardiac catheterization. When the morphologic class of the VSD and the preoperative total left-to-right shunt are known, the probability of necessity for eventual surgical treatment of the VSD after the initial CoA repair can then be calculated. This policy offers the surgeon different advantages: first, a well-considered choice between single-stage and multistage repair; second, weighing the risks of secondary or tertiary surgery on the VSD versus the risk of recoarctation;

and third, ensuring that the number of procedures per infant will be as low as possible.

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Appendix

Probability estimates were obtained from the logistic regression equation according to the following formulas:

$$p = \frac{1}{1 + e^{-z}}$$

$$z = b_0 + b_1 + \dots + b_k x_k$$

where e is the base of the natural logarithm, b_0 is the intercept of the logistic equation, and b_1 and b_k are the logistic regression coefficients associated with the values for the incremental risk factors x_1 and x_k .¹²