SURGICAL TREATMENT OF SUBAORTIC STENOSIS AFTER BIVENTRICULAR REPAIR OF DOUBLE-OUTLET RIGHT VENTRICLE

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Out of 180 patients who underwent biventricular repair of double-outlet right ventricle between 1980 and 1995, 9 (5%) required reoperation because of subaortic stenosis. Two other patients who initially underwent operation elsewhere underwent reoperation at our institution because of subaortic stenosis. The median age at biventricular repair was 4 months. Repair consisted of tunnel construction from the left ventricle to the aorta in nine patients; the remaining two patients received an arterial switch operation with ventricular septal defect closure. Subaortic stenosis developed with time: the mean postoperative left ventricle–to–aorta gradient after repair was 10 ± 19 mm Hg (range, 0 to 50 mm Hg) and became 84 ± 27 mm Hg (range, 40 to 124 mm Hg) in a mean delay of 45 ± 66 months (range, 1 to 213 months). At reoperation, the obstruction was caused by the protrusion of the inferior rim of the ventricular septal defect into the left ventricular outflow tract associated with subaortic hypertrophied muscle and membrane. The 11 patients underwent 15 reoperations. Surgical technique consisted of an extended septoplasty in 6 reoperations. In this technique an incision was made in the septal patch and was extended into the muscle toward the apex until a large opening of the left ventricular outflow pathway was obtained. A new patch was then secured to streamline the left ventricular outflow tract. None of the patients who underwent extended septoplasty had to undergo reoperation. There were no early or late deaths. At 115 ± 85 months after biventricular repair, all patients were in New York Heart Association functional class I or II and the mean postoperative left ventricle–to–aorta gradient was 20 ± 24 mm Hg (range, 0 to 60 mm Hg). We conclude that after biventricular repair of double-outlet right ventricle, the subaortic region is at risk for the development of stenosis. Surgical treatment adapted to the anatomy of the obstruction can offer good early and midterm results. It seems that an aggressive approach by an extended septoplasty avoids multiple reoperations. (J Thorac Cardiovasc Surg 1996;112:1570-80)

Despite improvements achieved in the surgical management of congenital heart disease, double-outlet right ventricle (DORV) remains a complex anomaly that raises challenging surgical problems. Since the first intracardiac tunnel repair was done in 1957,[1] several anatomic landmarks have been described, and a variety of surgical procedures have been proposed. In the current era, the surgical approach is to connect the left ventricle (LV) to the systemic circulation and the right ventricle to the pulmonary circulation with use of an intraventricular baffle either alone or in association with extracardiac procedures such as the arterial switch operation (ASO) or the insertion of extracardiac conduits. Although operative techniques and indications have become more accurate, the progressive development of subaortic obstruction, even in the presence of a large, nonrestrictive ventricular septal defect (VSD), has been noted. This report presents our experience with left ventricular outflow tract

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(LVOT) obstruction after biventricular repair of this type of anomaly.

Material and methods

Anatomic considerations. The term *double outlet* is used to describe a specific ventriculoarterial connection rather than to describe conotruncal morphologic features.[2] DORV was diagnosed when the lumen of both great arteries arose for more than 50% from the right ventricle over a malalignment form of VSD with a normal atrial arrangement (situs solitus) and a concordant atrioventricular connection. The absence of bilateral complete infundibular structures (bilateral conus) was not an exclusion criteria.[3] Patients in whom the infundibular morphologic features were those of tetralogy of Fallot (subaortic VSD and anterior deviation of the outlet septum with subpulmonary stenosis) were included in the DORV group according to the rule of 50% when the aorta was not in its normal position (posterior and right)[4] or when the aortic-mitral fibrous continuity was not complete.[5] Other patients with subaortic VSD and pulmonary infundibular stenosis were not included in the study unless the aorta arose entirely from the right ventricle.[6] The Taussig-Bing type of DORV was diagnosed when the aorta arose entirely from the right ventricle and the pulmonary artery for more than 50% over a subpulmonary VSD.[7, 8]

Patient population. Between January 1980 and December 1995, out of 180 patients who underwent biventricular repair of DORV (Table I), 9 (5%) required reoperation because of subaortic stenosis. Two other patients who initially underwent operation elsewhere underwent reoperation at our institution because of subaortic stenosis and were included in the study. Only one patient had subaortic stenosis before the first operation. At initial presentation, associated lesions were present in eight patients (73%) (Table II). Five patients (36%) had undergone previous palliative procedures: two pulmonary artery banding procedures, one of which was associated with coarctation repair, and three modified Blalock-Taussig shunt procedures. The median age at biventricular repair was 4 months and ranged from 18 days to 18 years.

In nine patients the type of anatomic repair consisted of the placement of an intraventricular baffle connecting the LV to the aorta through a transventricular approach: three patients needed concomitant anterocaudal VSD enlargement, two had wide excision of the conal septum, and one had both VSD enlargement and conal excision. Two patients with a subpulmonary VSD (Taussig-Bing anomaly) had VSD closure connecting the LV to the pulmonary artery followed by an ASO. Four patients had right ventricular outflow tract obstruction. The right ventricular outflow tract was enlarged by infundibular resection, pulmonary valvotomy, and placement of a transannular patch in two patients. Two others had an REV (réparation à l'étage ventriculaire) operation.[9] One patient who had undergone a previous banding operation needed right venticular outflow patch enlargement after the construction of a large intracardiac tunnel. Two patients had abnormal attachment of the tricuspid valve chordae on the conal septum. These chordae were detached for baffle reconstruction and then fixed to the posterosuperior edge of the Dacron patch. At discharge from the hospital after biventricular repair, mean LV-to-aorta gradient was 10 ± 19 mm Hg (median, 0; range, 0 to 50 mm Hg). Patients were categorized according to the position of the VSD and its relationship to the great arteries (subaortic, subpulmonary, doubly committed, and noncommitted) and according to the relation of the aorta to the pulmonary trunk ("more-or-less" normal, side by side, and aorta anterior to pulmonary trunk) (Table II).[4, 10]

Table I. *Biventricular repair of DORV at Marie Lannelongue Hospital from 1980 to 1995 (n = 180)*

	n	%
Biventricular repair (median age, 21 [0.3-290] mo; median weight, 9.5 [2.8-61] kg)	180	100
Morphologic considerations		
Great arteries		
Normal	79	44
Side-by-side	41	23
Anterior aorta	51	28
L-Malposition	7	4
Situs inversus	2	1
Total	180	100
VSD		
Subaortic	106	59
Subpulmonary (TBA)	37	21
Doubly committed	17	9
Noncommitted	20	11
Total	180	100
Palliative procedures	83	46
BT shunt	39	22
Pulmonary banding	44	24
Type of operation		
Intracardiac repair	150	83
With VSD enlargement	35	23
ASO	28	16
Senning operation	2	1
Total	180	100

TBA, Taussig-Bing anomaly; *BT,* Blalock-Taussig.

The mean interval before reoperation because of subaortic stenosis was 45 ± 66 months (range, 1 to 213 months). Reoperation was indicated when the LV-to-aorta peak systolic gradient was more than 60 mm Hg at hemodynamic evaluation or in the presence of symptoms. Three patients needed a second reoperation, respectively 5, 35, and 120 months after the first operation, and two of these needed a third reoperation because of restenosis (Table II).

Operative technique. Operations were done with the use of moderate hypothermia. Myocardial protection was achieved by single-dose crystalloid cardioplegia until 1990 and thereafter by multidose antegrade cold blood cardioplegia with topical cooling.

In all cases, a transverse aortotomy was done. The aortic valve, the anulus, and the LVOT were first explored through this approach. The conal septum, the subaortic

Table II. *Patient characteristics and surgical data*

Patient No.	*Great arteries*	*VSD*	*Bilateral conus*	*Associated lesions*	*Palliation*	*Age (mo) at biventricular repair*	*Surgical technique at repair*
1	Anterior aorta	Noncommitted	−	RVOTO	BT shunt	12 (conal resection)	REV
2	Side-by-side	Subpulmonary	+	—	PAB	4 (conal resection)	IR
3	Side-by-side	Subpulmonary	+	Coarctation	PAB and CR	3	ASO
4	Side-by-side	Noncommitted	+	Hypoplastic pulmonary arteries	—	1.5 (VSD surgically enlarged; conal resection)	IR
5	Normal	Subaortic	−	—	—	3*	IR
6	Side-by-side	Noncommitted	+	Noncompressive pulmonary sling	—	4 (VSD surgically enlarged)	IR
7	Side-by-side	Subpulmonary	+	—	—	0.7	ASO
8	Normal	Subaortic	−	IAA and subaortic stenosis	—	0.5*	IR
9	Anterior aorta	Subaortic	+	RVOTO	BT shunt	216	IR
10	Anterior aorta	Subaortic	+	RVOTO	BT shunt	58 (VSD surgically enlarged)	IR
11	Anterior aorta	Noncommitted	+	RVOTO	—	4 (VSD surgically enlarged)	REV

RVOTO, Right ventricular outflow tract obstruction; *BT,* Blalock-Taussig; *REV,* réparation à l'étage ventriculaire; *FR,* fibromuscular resection; *PAB,* pulmonary artery banding; *IR,* intracardiac repair; *SPE,* septal patch enlargement; *CR,* coarctation repair; *ES,* extended septoplasty; *MVR,* mitral valve replacement; *AVR,* aortic valve replacement; *IAA,* interrupted aortic arch.

* Operation done elsewhere.

† Reoperation because of residual VSD.

‡ Reoperation because of residual VSD and tricuspid regurgitation.

conus (if present), the VSD patch, and attachments of the mitral valve were systematically inspected. The obstruction was located at two levels. The first obstruction was at the entry of the tunnel: in all cases protrusion of the inferior rim of the tunnel into the LVOT was present. In addition, according to the morphologic type, a narrow ring of subaortic stenosis composed of fibrous material beneath the aortic valve and a bulge of the underlying septal or subaortic conal muscle were hemodynamically the main causes of the obstruction in the majority of the patients. Obstruction as a result of accessory mitral valve tissue was not observed in this series.

The surgical techniques that were done can be divided into three categories (Fig. 2).

Fibromuscular resection. In the first six reoperations, surgical management of the subaortic obstruction was done through the aorta by fibrous resection and myectomy between the superior edge of the patch and the aortic anulus. During these operations, the protrusion of the interventricular septum at the level of the inferior edge of the VSD was noted but the size of the LV outflow tunnel was judged acceptable after testing with Hegar's dilators. Two of the patients treated with this technique required a second reoperation.

Limited tunnel enlargement. This technique was used at the beginning of our experience. Three patients had limited tunnel enlargement. In two a fibromuscular resection beneath the aortic anulus was associated with the procedure. One patient had previously undergone reoperation and was treated with a subaortic fibromuscular resection. This technique consisted of isolated enlargement of the VSD patch. After the aortotomy and the analysis of the LVOT, a right ventriculotomy was done at the level of the previous one. The septal patch was incised parallel to the long axis of the ventricle and the tunnel was enlarged by insertion of a new Dacron patch. Recurrent subaortic stenosis developed in two patients treated with this technique, necessitating another reoperation.

Extended septoplasty. After exploration of the valvular and subvalvular area through an oblique aortotomy, a right ventriculotomy was done and the septal patch was incised centrally parallel to the long axis of the interventricular septum. A Hegar's dilator was then introduced into the LV through the aortic valve and the septal incision was extended beyond the inferior rim of the patch, in the muscle, toward the apex until a large opening of the LVOT was obtained. The incised area was the upper part of the trabecular septum and thus injury to the conduction system and major septal coronary arteries was avoided. The absence of mitral papillary muscles, known as "septophobic," was determined by preoperative echocardiography. However, those (or, the papillary muscles) were not in jeopardy because the incision started at the level of the patch and was extended in a step-by-step fashion, under direct vision. The patch incision was then extended toward the aorta, into the conus if any, and stopped at the fibrous edge of the aortic anulus (Fig. 1, *A*). Care was taken not to damage the aortic cusps. Septal or subaortic conal myectomy and resection of fibrous components were associated to the procedure to obtain a harmonious outflow tract. A new patch was then secured and molded around Hegar's dilators passed through the

First reoperation delay (mo)/technique	*Second/third reoperation delay (mo)/ technique*	*Actual gradient (peak, mm Hg)*
130/FR	—	None
1/SPE	—	20
11/FR, mitral repair	5/ES, MVR 46/AVR, MVR	18
29/FR	—	60
33/*†	180/ES and FR	45
20/FR	—	None
18/FR	—	15
7/FR	35/SPE and FR 26/ES and FR	60
30/ES	10/‡	None
24/SPE and FR	120/ES	None
10/ES	—	None

aortic valve to align the LVOT (Fig. 1, *B*). This technique had been previously done by DeLeon and coworkers[11] through a right atriotomy in three patients with previous VSD closure.

We performed this technique in five instances: in three children requiring a first reoperation and in one at a second reoperation. One other patient received extended septoplasty and fibromuscular resection at a third reoperation. It was possible to enlarge the VSD by muscular resection through the aortotomy at the entry of the tunnel in one patient who underwent septal patch enlargement associated with fibromuscular resection at a first reoperation. None of the patients who underwent an extended septoplasty required further reoperation.

A summary of the operative data for each patient is shown in Table II. Values are expressed as mean plus or minus the standard deviation.

Results

There were no early or late deaths. The mean postoperative LV-to-aorta gradient after the first reoperation for subaortic stenosis was 23 ± 23 mm Hg (median, 15; range, 0 to 60 mm Hg). After the second reoperation ($n = 3$) the mean gradient was 71 ± 37 mm Hg (range, 0 to 98 mm Hg) and the two patients who underwent a third reoperation had gradients of 18 mm Hg (patient No. 3) and 40 mm Hg (patient No. 8) at hospital discharge (Table II).

Follow-up was available in all patients. Patient No. 9 underwent a second reoperation after an extended septoplasty because of residual VSD (pulmonic-to-systemic flow ratio >1.5) and moderate tricuspid regurgitation. Another patient (No. 3) with Taussig-Bing anomaly and moderate mitral regurgitation in whom an ASO and VSD closure were initially done needed mitral valve repair because of severe mitral regurgitation associated to fibromuscular resection in a first reoperation at age 15 months. This patient underwent a second reoperation 5 months later and received extended septoplasty because of residual transventricular gradient (45 mm Hg), aortic (native pulmonary) valve repair because of mild regurgitation, and mitral valve replacement (18 mm, mechanical valve) because of residual stenosis and regurgitation. After this operation he had atrioventricular block, which necessitated permanent pacemaker implantation. The patient remained in good functional state for 4 years and recently underwent a third reoperation because of aortic valve disease and dysfunction of the mitral valve prothesis in which a combined aortic valve replacement (16 mm, mechanical valve) and mitral valve rereplacement with a larger size valve (21 mm, mechanical valve) was done.

LV end-diastolic internal diameter values on preoperative and postoperative (at hospital discharge) echocardiograms at the time of anatomic repair were reviewed. In patients in whom values were obtained the mean decrease in LV end-diastolic internal diameter was 17% ± 7% (95% confidence limits) after operation.

No iatrogenic aortic valve injuries occurred during the 17 reoperations, and permanent atrioventricular block developed in only one patient. At 115 ± 85 months after biventricular repair and 26 ± 22 months after most recent reoperation, all patients were in New York Heart Association functional class I or II and the mean gradient between the LV and the aorta was 20 ± 24 mm Hg (median, 15; range, 0 to 60 mm Hg) (Table II).

Discussion

Construction of an intracardiac baffle to tunnel the LV to the aorta with use of the VSD as the egress of the LV is the preferred surgical approach for repair of the majority of the forms of DORV and also of dextro-transposition of great arteries with a VSD and pulmonary stenosis. The same principle is applied in Taussig-Bing anomaly when an ASO is indicated: the VSD is tunnelized to the pulmonary artery, then repair is completed by an ASO. On the other hand, reconstruction of the right ventricular outflow tract, if necessary, requires a variety of surgical techniques, including the Lecompte maneuver for REV (réparation à l'étage ventriculaire) operation, infundibular resection with outflow patch

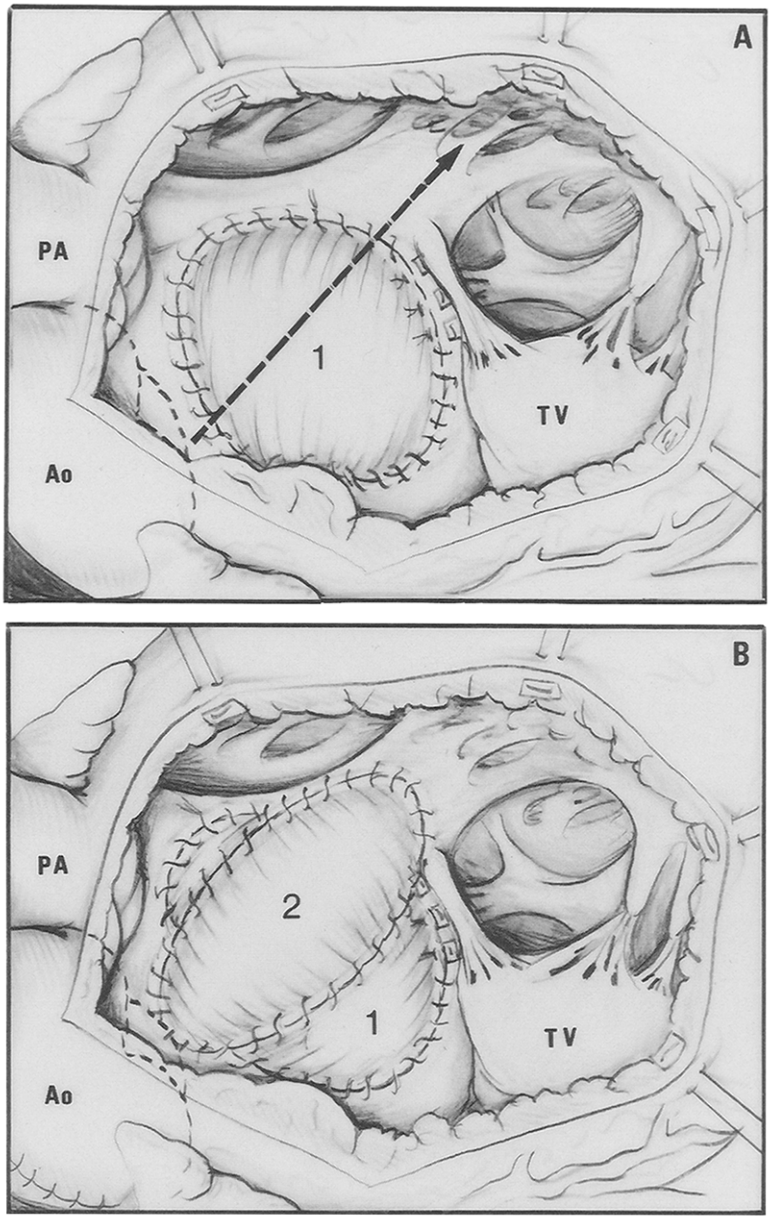

Fig. 1. **A** and **B,** Extended septoplasty viewed through right ventriculotomy. *Arrow* is oriented toward apex of heart. *Ao,* Aorta; *PA,* pulmonary artery; *TV,* tricuspid valve; *1,* previous tunnel patch; *2,* new patch.

enlargement, and extracardiac conduit insertion with use of a homograft or synthetic valved conduit.

During baffle repair, adequate construction of the LV outflow pathway is possible in the presence of a large or surgically enlarged VSD and with use of a patch that is positioned as straight as possible. Subaortic obstruction may be present before surgical repair because of a restrictive VSD,[12-14] subaortic conal muscle, and hypertrophy of the conal septum,[15] especially after pulmonary artery banding in patients without pulmonary stenosis.[16] Because the obstruction may lie at different levels, anatomic biventricular repair, with VSD enlargement if necessary, should take into account the location and the anatomy of the VSD and the spatial arrangement of the atrioventricular valves and great vessels to obtain an unobstructed tunnel.[17] In our series, no patient required reoperation because of subaortic stenosis after intracardiac baffle repair of transposition of the great arteries with pulmonary stenosis or pulmonary atresia.

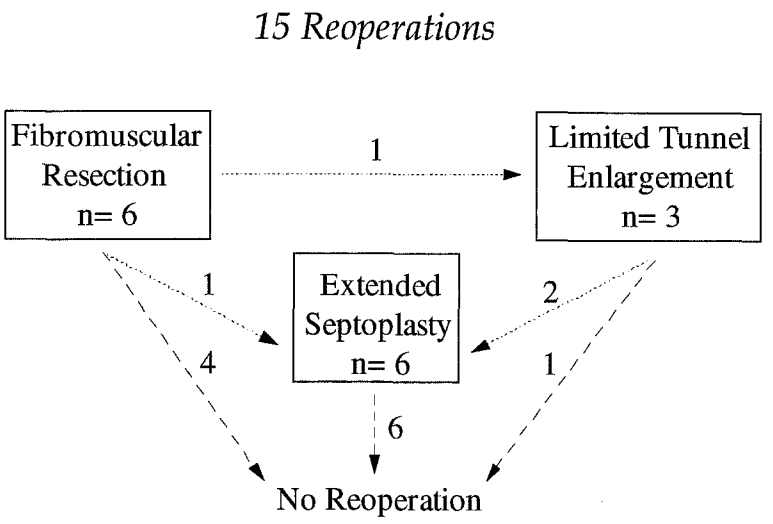

Fig. 2. Diagram representing evolution of cases after subaortic stenosis relief by each of three proposed surgical techniques.

The occurrence of subaortic obstructive lesions after biventricular repair in this group of anomalies has been variably assessed in previously published series. Luber[18] and Kirklin[19] and their associates reported in their series, respectively, two (2/57) and seven (7/127) patients who underwent reoperation because of subaortic obstruction after biventricular repair of DORV. Chaitman and associates[20] published in 1976 the case of a patient who underwent reoperation because of LVOT obstruction 15 years after the intracardiac repair of DORV. Rocchini and colleagues[21] reported for the first time the presence of subaortic obstruction in five patients who underwent operation because of anomalous ventriculoarterial connections. In each case, the obstruction appeared angiographically to be located at the entrance of the LV baffle, although all were deemed to have a nonrestrictive VSD before operation and at operation.

The anatomic features of the LV outflow tunnel after intracardiac baffle repair can be analyzed at three levels (Fig. 3). The VSD is the entry of the tunnel. Because the patch is inserted to the right aspect of the septum, the rims of the VSD can form the narrowest segment of the tunnel. The VSD is occasionally limited posterosuperiorly by the arterial-mitral fibrous continuity or by the left arterial cusp, depending on the degree of overriding of the latter; but, by definition, in the majority of cases the posterosuperior roof is constituted by subaortic (subpulmonary in the case of Taussig-Bing anomaly) conal muscle (Fig. 4).

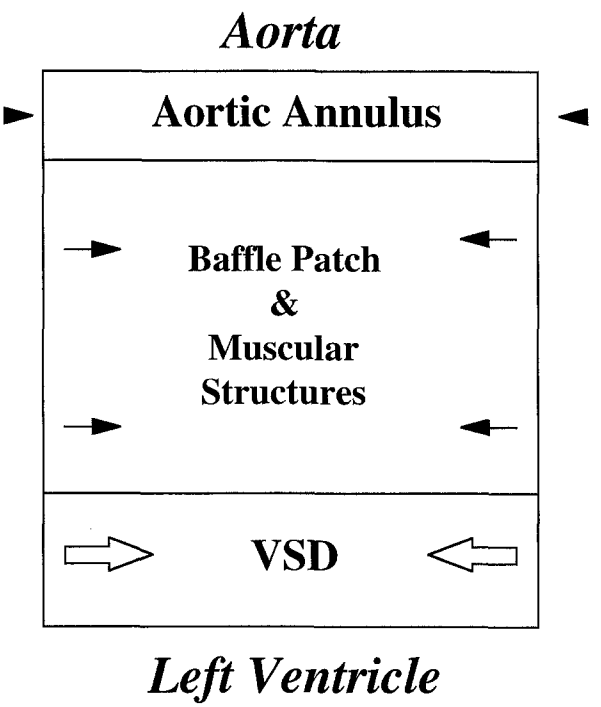

Fig. 3. Diagram representing LVOT after intracardiac baffle repair of DORV. *Arrows* indicate the development of obstruction at three different levels (see text).

The second level is the tunnel itself. After repair, the LVOT at this level is made for its largest part of synthetic material, by the remnant of conal septum, and by the subarterial conal muscle and has a sinuous shape. The shape and the length of the

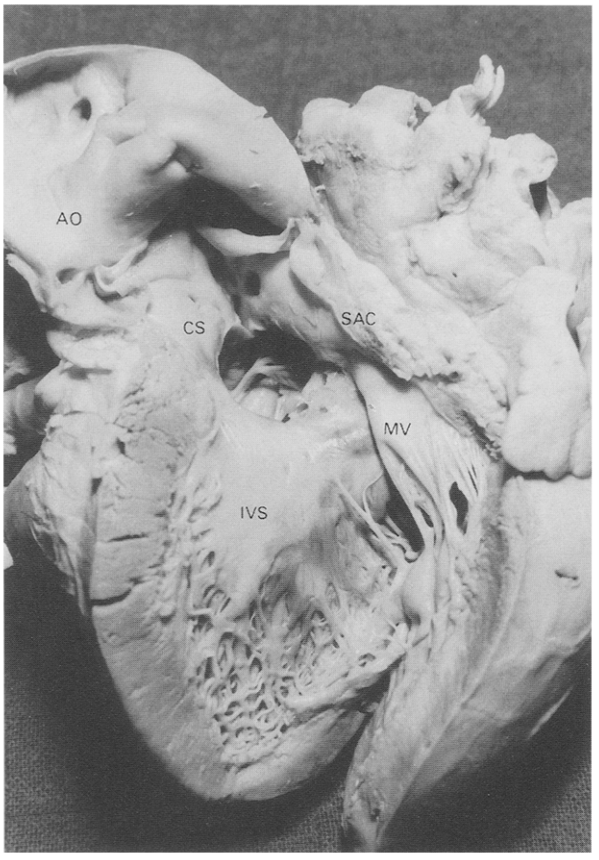

Fig. 4. LV view of LVOT in DORV specimen with subaortic VSD. *AO,* Aorta; *IVS,* interventricular septum; *SAC,* subaortic conus; *CS,* conal septum, *MV,* mitral valve.

tunnel are related to the location of the VSD and to its distance from the aortic valve. If the location of the VSD is noncommitted, the upper part of the trabecular septum (if not resected) can be part of the tunnel construction.

The third level, the end of the tunnel, is the right ventricle–to–aorta junction (the aortic valve "anulus"). It may be possible to fix the growth of the aortic anulus and cause aortic stenosis by inadequate insertion of the upper edge of the patch.

In patients with Taussig-Bing anomaly after an ASO, the anatomic arrangement seems to be different. As previously reported,[22] the interventricular communication is made by two ostia and the tunnel construction from the LV to the pulmonary artery necessitates placement of the patch on the left VSD ostium. In this situation, the LVOT is between the interventricular septum and the left free wall of the conus. The inferior rim of the VSD and the subpulmonary conus can protrude into the LVOT.

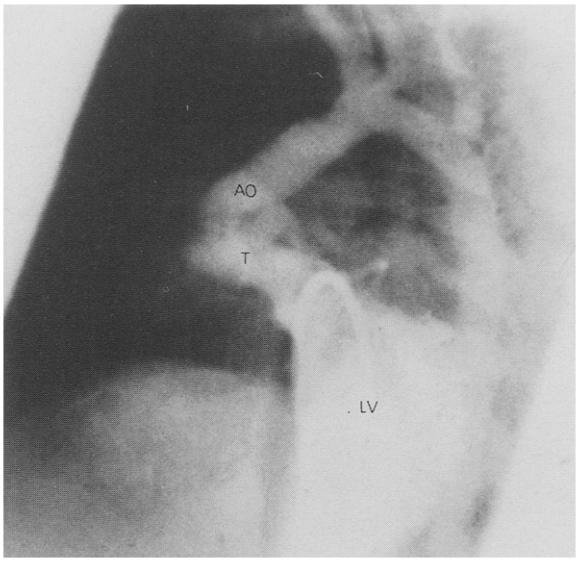

Fig. 5. Angiogram showing LVOT after baffle repair in patient No. 2. *AO,* Aorta; *T,* tunnel.

Rocchini and colleagues[21] theorized that the mechanism of obstruction was related to the decrease in effective VSD size caused by either the baffle itself or the secondary proliferation of fibrous tissue along the rim of the VSD or the baffle, or in both places. They proposed as other possible causes kinking of the baffle, shrinkage of the baffle with time, and spontaneous diminution of the VSD orifice. Rychik and coworkers[23] analyzed the preoperative and postoperative echocardiograms of 24 patients who underwent the Rastelli operation or intraventricular baffle repair for DORV and showed that the LV undergoes geometric changes after these operations in which the VSD is used as the new LV outflow pathway: patients in whom subaortic obstruction subsequently developed had the greatest degree of ventricular contraction and VSD diminution early after operation but the reasons for differential degrees of geometric change remained unclear. Yacoub and Radley-Smith[24] and Serraf and associates[22] both reported the development of subaortic obstruction after biventricular repair of Taussig-Bing anomaly and described surgical landmarks to avoid it.

To our knowledge this is the first reported series of surgical treatment for subaortic stenosis after biventricular repair of DORV. As listed before, the anatomic and clinical features of the 11 patients were variable. However, except for patients No. 2 and No. 8 (Table II), in all cases the obstruction had developed and progressed with time, independent of

Table III. *Individual evolution of LV-to-aorta gradient*

	Patient No.										
	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*
Biventricular repair											
Peak gradient at discharge (mm Hg)	0	50	0	0	0	10	0	45	0	0	10
Delay (mo)	130	1	11	29	213*	20	18	7	30	24	10
First reoperation											
Peak gradient before reoperation (mm Hg)	80	50	100	122	100	75	78	124	60	40	90
Peak gradient at discharge (mm Hg)	0	10	45	60	45	0	30	50	0	15	0
Delay (mo)	—	—	5	—	—	—	—	35	—	120	—
Second reoperation (n = 3)											
Peak gradient before reoperation (mm Hg)	—	—	80	—	—	—	—	143	—	70	—
Peak gradient at discharge (mm Hg)	—	—	45	—	—	—	—	98	—	0	—
Delay (mo)	—	—	46	—	—	—	—	26	—	—	—
Third reoperation (n = 2)											
Peak gradient before reoperation (mm Hg)	—	—	90	—	—	—	—	170	—	—	—
Peak gradient at discharge (mm Hg)	—	—	18	—	—	—	—	40	—	—	—
Delay (mo)	6	88	2	10	8	17	28	21	25	33	23
Actual gradient	0	20	18	60	45	0	15	60	0	0	0

* Patient underwent a previous reoperation because of residual VSD.

age (Table III). The data did not allow us to determine whether age at initial repair was a risk factor for the development of subsequent LVOT obstruction.

Patient No. 2 had Taussig-Bing anomaly with side-by-side great vessels and type E (Yacoub classification) coronary arteries; 2 weeks after a poorly tolerated pulmonary artery banding procedure done at age 3.5 months, he underwent intracardiac repair with conal septal resection and right ventricular transannular patch placement to enlarge, after removal of the pulmonary artery band, the main pulmonary artery and the right ventricular outflow tract, which was partially occluded by the tunnel itself. Subaortic stenosis appeared immediately after the repair with a 50 mm Hg transventricular gradient at cardiac catheterization and the patient underwent reoperation 4 weeks later and received a septal patch enlargement (Table II). In our opinion, the size of the VSD was overestimated and resection of the conal septum alone was not enough to set up an adequate outflow pathway (Fig. 5).

Patient No. 8 had subaortic stenosis before the first operation he underwent, which was done elsewhere at age 18 days and during which intracardiac baffle repair was associated with repair of interrupted aortic arch. After this operation the LV-to-aorta gradient was 45 mm Hg, and this increased to 125 mm Hg in 7 months, at the time of his first reoperation in our institution. This patient underwent three reoperations (Table II) and has, at age 7 years, a 60 mm Hg gradient at the level of the aortic anulus.

It is known that the natural tendency of VSD is to decrease in size with time.[25] On the other hand, the acute volume unloading of the LV after closure of a VSD increases the wall thickness, decreases LV cavity dimensions, and consequently decreases the VSD size.[26] This phenomenon has major importance after operations in which the VSD is used as the new LV outflow pathway.

The cause of the development of subaortic stenosis is believed to be, first, clinically subtle subaortic constraint at the entry of the tunnel and the sinuous shape of the latter may lead to turbulent flow beneath the aortic valve, resulting in either septal or subarterial conal muscle hypertrophy or the deposition of fibrous material, which may, at a later time, result in hemodynamically significant subaortic obstruction; second, growth of the heart without concomitant increase in the size of the VSD and tunnel; and finally, in some patients, excessive decrease of the LV diameter and the increase in wall thickness after biventricular repair, which also causes the diminution of the VSD orifice and the augmentation of the malalignment. Baffle patch retraction was not observed in this series. Obstruction at the valvular or annular level developed in only one patient (patient No. 8).

In their echocardiography study, Gewillig and colleagues[27] supported the theory that isolated discrete or fibromuscular subaortic stenosis may be the result of a chronic flow disturbance caused by a somewhat narrowed and elongated LVOT: by resecting the subvalvular obstruction only the end

product of a rheologic disturbance is removed but the underlying cause remains unaltered.

In conclusion, after the use of an intracardiac baffle to tunnel the LV to the aorta, the subaortic region is at risk for the development of stenosis. Preoperative detailed echocardiography to define contributing factors to creation of the obstruction is essential. Operation should not only aim at relief of the obstruction, but also at streamlining the LVOT by removal of protrusions located at the three described levels. After the exploration of the LVOT through aortotomy, a right ventriculotomy is done and the outflow is accessed through baffle patch incision extended to the upper trabecular septum. A double approach allows removal of obstructive structures at each level and reconstruction of a harmonious LV outflow pathway by insertion of a new patch molded around Hegar's dilators. It seems that the aggressive approach by an extended septoplasty avoids multiple reoperations.

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Discussion

Dr. Erle H. Austin III *(Louisville, Ky.).* Dr. Belli and the group at Marie Lannelongue Hospital have made many

contributions to the surgical management of DORV. By applying a uniform approach to this complex set of anomalies, Dr. Planché and his colleagues have amassed an impressive series that is remarkable for its excellent results, as well as its significant numbers. I believe that 180 consecutive cases represents the largest single-center experience with DORV ever reported.

Because the chief objective of biventricular repair of DORV is to create an unobstructed pathway from the LV to the aorta, this analysis of those patients who required reoperation because of subaortic stenosis is both important and enlightening. The low prevalence of 5% for the development of subaortic stenosis after primary repair, in the absence of mortality in a challenging reoperative group, serves as a tribute to the technical expertise of Dr. Planché and his colleagues.

Among other things, I was particularly impressed by two aspects of this study. First, it appears that subaortic stenosis can develop somewhat late after biventricular repair even in patients without evidence of an outflow gradient at the time of discharge from the hospital. This finding, of course, emphasizes the importance of close and long-term follow-up after surgical correction of all patients with DORV. The other point that I think the current experience indicates is that when reoperation is required because of subaortic stenosis, a conservative attempt to enlarge the LVOT is unlikely to result in permanent relief. An aggressive approach with transaortic and transventricular exposure and generous septoplasty with extension beyond the inferior rim of the patch into the trabecular septum appears to provide a more satisfactory and lasting result.

I have two questions. First, because the median age at repair in the total group of 180 patients was 21 months and the median age at primary repair in the group requiring reoperation because of subaortic stenosis was only 4 months, should we not interpret early age at repair as a risk factor for the development of subaortic stenosis? Although we recognize the benefits of early complete repair in so many congenital heart lesions, maybe we should be less enthusiastic about early complete repair in infants with anatomic forms of DORV that will require extensive intraventricular muscle resection to create a satisfactory intraventricular tunnel.

Second, has this review of this group of patients who required reoperation because of subaortic stenosis resulted in any insights into the technical details during the initial repair that might completely eliminate any potential for the later development of subaortic stenosis?

Dr. Belli. In our series the prevalence of the development of subaortic stenosis was 5%. Out of 180 patients who underwent biventricular repair, 60 (33%) were younger than age 1 year. Thirty of them, that is, half of them, had an ASO and the remaining 30 had intracardiac baffle repair. Although the median age was smaller in the group with subaortic stenosis, the range was similar to that in the group with biventricular repair.

Eight of 11 patients were younger than 1 year old at biventricular repair. Two of these repairs were ASOs. One patient with an associated interrupted aortic arch had an underdeveloped LVOT and aortic root. He underwent three reoperations and, at the present time, he has a transvalvular gradient of 60 mm Hg. He will probably be a candidate for an aortic root replacement in a few years. One other patient needed early reoperation probably because the patch was undersized. Three of the four remaining young patients had a noncommitted VSD, which was enlarged during the biventricular repair. In conclusion, the clinical and morphologic features of this small series are variable and they do not allow us to reach a conclusion concerning the ideal age at repair. The choice between one-stage versus two-stage repair should depend on the anatomic and clinical characteristics of each particular patient. At initial repair, 23% of the patients had concomitant VSD enlargement to obtain an adequate LV outflow pathway. According to our surgical data, the development of subaortic stenosis is not predictable.

Dr. Dominique R. Metras *(Marseille, France).* These are impressive results. I was particularly interested to see that in the Taussig-Bing anomaly, ASO and closure of the VSD did not avoid completely the occurrence of late subaortic stenosis. I would like to report to you the late results in a different approach in Taussig-Bing syndrome, namely, the Kawashima correction, which is construction of a long intraventricular tunnel between the VSD and aorta. We reported in The Journal of Thoracic and Cardiovascular Surgery more than 10 years ago the case of a 22-month-old baby in whom correction was done this way. At that time this was the youngest patient treated by the Kawashima operation to be reported on. The tunnel was done with a half of an 18 mm Dacron tube cut longitudinally. Twelve years later, at the age of 14, this patient underwent recatheterization and there was absolutely no gradient between the LV and aorta. This does not necessarily mean that this will be so in every Kawashima procedure, but simply indicates that this kind of operation may lead to an excellent late result, and presently we still do some Kawashima operations for Taussig-Bing anomalies.

I would like to ask the authors what their present strategy is in the group of patients with Taussig-Bing syndrome and whether they think that in favorable anatomic conditions the Kawashima operation may still represent a valid alternative to the more fashionable ASO.

Dr. Belli. In our series, out of 180 patients, 37 had Taussig-Bing–type DORV. Eighteen of them had an anterior-posterior relationship of the great arteries. These patients underwent an ASO because in this configuration the LV outflow tunnel would have to run anteriorly, directly under the pulmonary trunk: the result would be an iatrogenic right ventricular outflow tract obstruction. The remaining 19 patients had a side-by-side relationship. In this configuration, although an ASO is always feasible, baffle repair is more attractive inasmuch as it preserves the native aortic valve and avoids coronary dissection. However, to avoid postoperative subaortic stenosis, the anatomy in this particular group of patients was evaluated by preoperative echocardiography. In 10 patients, the distance between the pulmonary and tricuspid anuli was inferior to the aortic valve diameter or abnormal tricuspid attachments were present: the patients underwent an ASO. In the remaining nine patients, the distance was

enough to perform an intracardiac baffle repair. In the latter group, we actually recommend performance of the intracardiac repair in patients older than 1 year.

Dr. Yasunaru Kawashima *(Osaka, Japan).* I have enjoyed this precise analysis of subaortic stenosis after the repair of DORV. I would like to make a comment. Among the various types of DORV, it is generally suspected that subaortic stenosis will often develop after the intraventricular repair of the subpulmonic VSD type because the distance between the VSD and aorta in this type is larger than in the other type and also because the anatomy is complicated between the VSD and aortic valve.

I have reviewed the late results in an initial series of 10 patients operated on 5 to 26 years ago with an intraventricular rerouting technique. Results at most recent study either by catheterization or Doppler echocardiography demonstrated that the pressure gradients between the LV and the aorta were all less than 20 mm Hg. The extreme resection of the infundibular septum between the subaortic root and VSD is mandatory to obtain a good long-term result.