JACC Vol. 19, No. 1
January 1992:142–8

Bulboventricular Foramen Size in Infants With Double-Inlet Left Ventricle or Tricuspid Atresia With Transposed Great Arteries: Influence on Initial Palliative Operation and Rate of Growth

ABRAHAM MATTIAU, MD, TAL GEVA, MD, STEVEN D. COLAN, MD, FACC, THIERRY SLUYSMANS, MD, IRA A. PARNESS, MD, PHILIP J. SPEVAK, MD, MARY VAN DER VELDE, MD, JOHN E. MAYER, JR., MD, FACC, STEPHEN P. SANDERS, MD

Boston, Massachusetts

Bulboventricular foramen obstruction may complicate the management of patients with single left ventricle. Bulboventricular foramen size was measured in 28 neonates and infants >5 months old and followed up for 2 to 5 years in those patients whose only systemic outflow was through the foramen. The bulboventricular foramen was measured in two planes by two-dimensional echocardiography, its area calculated and indexed to body surface area. One patient died before surgical treatment. The mean initial bulboventricular foramen area index was 0.94 cm^2/m^2 in 12 patients (Group A) in whom the foramen was bypassed as the first procedure in early infancy. The remaining 15 patients underwent other palliative operations but the bulboventricular foramen continued to serve as the systemic outflow tract. There was one surgical death. Six (Group B) of the 14 survivors developed bulboventricular foramen obstruction during follow-up (mean initial bulboventricular foramen area index 1.75 cm^2/m^2). The remaining eight patients (Group C) did not develop obstruction during follow-up and had an initial bulboventricular foramen larger than that in the other two groups (mean initial bulboventricular foramen area index 3.95 cm^2/m^2).

All patients with an initial bulboventricular foramen area index <2 cm^2/m^2 who did not undergo early bulboventricular foramen bypass developed late obstruction. Although the bulboventricular foramen area increased slightly with growth, when indexed to body surface area it decreased with time.

An excellent correlation was found between antemortem echocardiographic measurements and the bulboventricular foramen dimensions measured in 11 heart specimens (2 from the patients who died before or during operation, 5 from patients who died late postoperatively and 4 additional specimens from patients who were not followed up longitudinally).

It is concluded that 1) [illegible] two-dimensional echocardiography is reliable for determining bulboventricular foramen size; 2) bulboventricular foramen size in the neonate is an important predictor of late obstruction; and 3) although the bulboventricular foramen appears to grow, its growth does not keep pace with somatic growth in most patients.

(J Am Coll Cardiol 1992;19:142–8)

Obstruction of the bulboventricular foramen or "ventricular septal defect," the communication between the left ventricle and the outflow chamber, may complicate the course of patients with double-inlet left ventricle or tricuspid atresia and transposition of the great arteries. The development of bulboventricular foramen obstruction is associated with pressure overload, hypertrophy and fibrosis of the left ventricle (1). In addition, pulmonary blood flow may be increased because of the excess resistance to systemic outflow.

Surgical options for infants in whom the bulboventricular foramen is obstructed include 1) a pulmonary artery to aorta anastomosis (transection of the main pulmonary artery with anastomosis of the proximal pulmonary artery to the ascending aorta and construction of a systemic arterial to pulmonary artery shunt); 2) enlargement of the bulboventricular foramen by resection of surrounding muscular septum; and 3) interposition of a conduit from the left ventricle to the descending or ascending aorta. These operations have carried significant morbidity and mortality and it appears that the larger the gradient before surgery, the worse the prognosis (1–5).

It may be difficult to determine if the bulboventricular foramen is obstructed in neonates. Although decisions are sometimes based on the pressure gradient across the foramen, this criterion is frequently not reliable in neonates because of patency of the ductus arteriosus. Consequently, it is important to assess the size of the bulboventricular foramen directly.

From the Department of Cardiology, The Department of Pathology, and the Division of Cardiovascular Surgery, The Children's Hospital and The Departments of Pediatrics, Pathology and Surgery, Harvard Medical School, Boston, Massachusetts.

Manuscript received February 20, 1991; revised manuscript received June 11, 1991, accepted July 2, 1991.

Address for reprints: Stephen P. Sanders, MD, Department of Cardiology, The Children's Hospital, 300 Longwood Avenue, Boston, Massachusetts 02115.

0735-1097/92/$5.50

In some patients a bulboventricular foramen that is not restrictive in the neonatal period becomes obstructed with time. However, the relation between the size of the bulboventricular foramen in infancy and later in life is unknown. If the rate of change in bulboventricular foramen size could be predicted it might be possible to identify infants at risk for developing late obstruction.

Therefore, we undertook this study to 1) assess the accuracy of biplane two-dimensional echocardiography in estimating the size of the bulboventricular foramen; 2) determine the relation between this size and the choice of the initial palliative surgical procedure; 3) measure the change in size of the bulboventricular foramen with somatic growth; 4) determine the relation between the initial size of the foramen and the subsequent development of obstruction.

Methods

Study patients. All neonates and infants with double-inlet left ventricle or tricuspid atresia and transposition of the great arteries who presented to Boston Children's Hospital between 1982 and 1987 before 5 months of age and who had not had prior cardiac surgery were identified. Patients with double-inlet left ventricle who had normally related great arteries or both great arteries arising from the same outflow chamber were excluded from the study. This calendar period was chosen because high quality echocardiograms were available and all survivors had been followed up for ≥2 years. The medical records of patients were reviewed to determine the type of surgical procedure performed, the outcome of the procedure and the clinical course of the patient, especially regarding obstruction of the bulboventricular foramen. Obstruction of the bulboventricular foramen was defined (after infancy) as a systolic gradient ≥10 mm Hg by Doppler echocardiography or cardiac catheterization.

We also identified the patients with double-inlet left ventricle or tricuspid atresia with transposition of the great arteries who died during the preceding 6 years and who had a recent antemortem echocardiogram and a postmortem examination. The heart specimens were examined and measurements made for comparison with the echocardiographic measurements.

Echocardiographic analysis. The echocardiograms were reviewed and views selected that displayed the maximal diameter of the bulboventricular foramen in the short-axis plane of the heart (usually a parasternal short-axis or subxiphoid short-axis view, measuring the transverse dimension of the bulboventricular foramen) and in a long-axis plane of the heart (usually a parasternal long-axis or apical view, measuring the apex to base dimension of the bulboventricular foramen) (Fig. 1). Printed copies of selected still frames were obtained with a video page printer. The transverse (short-axis view) and apex to base (long-axis view) dimensions of the bulboventricular foramen were then measured with a computer and digitizing tablet. The cross-sectional

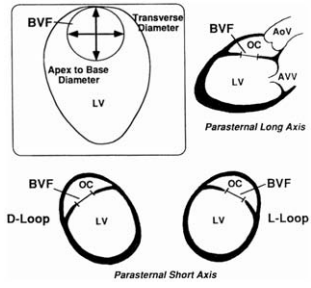


Figure 1. Schematic drawing of the bulboventricular foramen diameters and the measurement technique in long- and short-axis parasternal views. AoV = aortic valve; AVV = atrioventricular valve; BVF = bulboventricular foramen; LV = left ventricle; OC = outflow chamber.

area of the bulboventricular foramen (BVFA) was calculated by using the formula for a regular ellipse:

$$\text{BVFA} = A/2 \times B/2 \times 3.14,$$

where A = transverse diameter and B = apex to base diameter. Bulboventricular foramen area was then indexed to body surface area as bulboventricular foramen area index = bulboventricular foramen area divided by body surface area. The bulboventricular foramen area and area index were calculated for the initial and subsequent examinations.

Pathologic studies. Transverse and apex to base diameters were measured in a fashion similar to the echocardiographic measurements in the postmortem heart specimens, which had been fixed in a buffered formalin solution, and the bulboventricular foramen area was calculated as an ellipse. The bulboventricular foramen dimensions and area obtained in the specimens were compared with those obtained from the most recent antemortem echocardiogram.

Statistics. The echocardiographic and postmortem measurements were compared with use of linear regression analysis. Analysis of variance was used to test the differences in bulboventricular foramen area and bulboventricular foramen area index among patient groups. Because of the small number of subjects a Wilcoxon rank-sum test was used to test the significance of the difference in bulboventricular foramen area index between patients with and without coarctation and between patients with pulmonary artery banding who did and did not develop bulboventricular foramen obstruction. The slopes of the regression lines for bulboventricular foramen area and bulboventricular foramen area index versus age were determined and averaged as an index of the rate of growth of the bulboventricular foramen.

JACC Vol. 19, No. 1
January 1992:142–8

Table 1. Clinical Course of 28 Patients

Patient	Initial BVFAI	Segment	Diagnosis	Surgical Procedure				Alive
				1	2	3	4	
Group A								
A1	0.68	SDD	TGA, DILV, CoA	PAA, CO	G			+
A2	0.89	SLL	TGA, DILV, CoA	PAA, CO	CO	G	F	+
A3	1.10	SLL	TGA, DILV, CoA	PAA, CO	CO			–
A4	0.38	SLL	TGA, DILV, CoA	PAA, CO				–
A5	0.89	SLL	TGA, DILV, CoA	PAA, CO	G	F		+
A6	0.96	SDD	TGA, DILV, CoA	PAA, CO	G			+
A7	0.82	SDD	TGA, TA, CoA	PAA, CO	F			+
A8	2.39	SLL	TGA, DILV, CoA	PAA, CO	G	SH	F	+
A9	0.96	SLL	TGA, DILV, CoA	PAA, CO	SHN	PD		–
A10	0.34	SDD	TGA, DILV, CoA	PAA, CO	SH	F	W	+
A11	0.43	SDD	TGA, DILV, CoA	PAA, CO				–
A12	1.74	SDD	TGA, DILV, CoA	PAA, CO				–
A13	0.51	SLL	TGA, DILV, CoA					–
A14	1.02	SLL	TGA, DILV, CoA	B, CO				–
Group B								
B1	1.35	SLL	TGA, DILV	F	LVAO			–
B2	2.64	SDD	TGA, DILV	B	PAA	G		–
B3	1.67	SLL	TGA, DILV, CoA	B, CO	PAA	REV	F	+
B4	1.24	SDL	TGA, TA	B	PAA, F	W		+
B5	1.86	SDD	TGA, TA	B	B	PAA	F	+
B6	1.76	SDD	TGA, DILV	B	PAA	F		+
Group C								
C1	4.64	SLL	TGA, DILV	B	F			+
C2	4.36	SDA	TGA, DILV	B	F	G		–
C3	2.05	SLL	TGA, DILV, CoA	B, CO	G	F	TR	–
C4	6.12	SLL	TGA, DILV	SH	F			+
C5	6.31	SDD	TGA, DILV	B	SH	F	G	+
C6	2.34	SLL	TGA, DILV	SH	A.SP	SHN	F	–
C7	3.42	SLL	TGA, DILV	SH	F			+
C8	2.34	SDD	TGA, TA	B	F			+

A.SP = atrial septectomy; B = pulmonary artery band; BVFAI = bulboventricular foramen area indexed to body surface area; CO = coarctation repair; CoA = coarctation of the aorta; DILV = double-inlet left ventricle; F = Fontan procedure; G = Glenn procedure; Group A = patients in whom the bulboventricular foramen was by passed when they were neonates. Group B = patients in whom the bulboventricular foramen provided the only systemic outflow and who developed bulboventricular foramen obstruction during follow-up. Group C = patients in whom the bulboventricular foramen provided the only systemic outflow and who did not develop bulboventricular foramen obstruction during follow-up. LVAO = Left ventricle to aorta conduit; PAA = pulmonary artery to aorta anastomosis; PD = plication of diaphragm; REV = revision of pulmonary artery to aorta anastomosis; SH = systemic to pulmonary artery shunt; SHN = shunt narrowing; TA = tricuspid atresia; TGA = transposition of the great arteries; TR = tricuspid valve replacement; W = pericardial window; + = alive at the end of follow-up and after the last surgical procedure was performed; – = dead.

Results

Twenty-eight neonates and infants, 16 male and 12 female, were identified who met the selection criteria for this study (Table 1) (6,7). All patients had an outflow chamber from which the aorta originated and which communicated with the left ventricle through a bulboventricular foramen.

Echocardiographic-pathologic correlations. Twelve of the 28 patients died. Seven of the 12 and 4 additional patients (who presented after prior surgical treatment at another hospital and thus were not included among the 28 study patients) had a recent antemortem echocardiographic and postmortem examination.

The transverse and apex to base dimensions of the bulboventricular foramen measured from the most recent antemortem echocardiogram and from the fixed specimens were highly correlated ($r = 0.99$ and 0.92, $p < 0.001$) with a small SE. The measurements from the postmortem specimens were consistently about 10% to 12% smaller than the echocardiographic measurements. Bulboventricular foramen area calculated from the heart specimens was highly correlated with that obtained from the antemortem echocardiograms ($r = 0.99$ $p < 0.001$) (Fig. 2).

Surgical palliation. One patient (Patient A13, Table 1) died before any surgical procedure was performed.

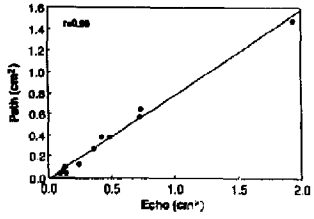


Figure 2. Regression plot of calculated bulboventricular foramen area from pathologic specimens (Path) and from a recent antemortem echocardiogram (Echo).

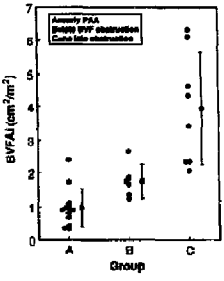


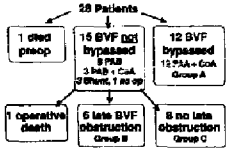
Figure 4. Initial bulboventricular foramen (BVF) area indexed to body surface area (BVFAi) in the three groups. Group A = early pulmonary artery to aorta anastomosis (PAA); Group B = late bulboventricular foramen obstruction; Group C = no bulboventricular foramen obstruction at latest follow-up.

Twelve patients underwent pulmonary artery to aorta anastomosis as neonates (Group A, Fig. 3). All the patients in this group had obstruction of the aortic arch or isthmus, or both, and the operation always included arch repair along with the pulmonary artery to aorta anastomosis. The mean initial bulboventricular foramen area index in this group was 0.94 ± 0.55 cm^2/m^2 (range 0.34 to 2.39) (Fig. 4). Only 2 of the 12 patients had a gradient detected across the bulboventricular foramen at the initial evaluation. In one the ductus was closed and in the other it was restrictive. Of the 10 patients without a gradient, 9 had a patent ductus arteriosus at the initial echocardiographic evaluation.

Eleven patients had been judged subjectively to have a small bulboventricular foramen at the time of the initial echocardiogram or angiogram. In the other patient in this group (Patient A8, Table 1) pulmonary artery to aorta anastomosis was performed because coarctation of the aorta was present even though the bulboventricular foramen was judged to be adequate in size on echocardiography. Eight of the 12 patients survived the initial palliative operation.

In the remaining 15 patients, the bulboventricular foramen was the only systemic outflow tract (Fig. 3). Palliative procedures were performed in 14 patients including pulmonary artery banding in 8, pulmonary artery banding and coarctation repair in 3 and creation of a systemic to pulmonary artery shunt in 3. One patient died after pulmonary artery banding and coarctation repair (Patient A14, Table 1). The remaining 14 survivors had been judged to have an unobstructed bulboventricular foramen at the time of the initial operation on the basis of subjective evaluation of the echocardiogram or angiogram and lack of a gradient across the bulboventricular foramen.

Figure 3. Flow diagram of the study patients, and the different groups. BVF = bulboventricular foramen; CoA = coarctation repair; no op = no palliative operation because of native pulmonary valve stenosis; PAA = pulmonary artery to aorta anastomosis; PAB = pulmonary artery banding; Preop = before operation; Shunt = systemic artery to pulmonary artery shunt.



Among these 14 patients, bulboventricular foramen obstruction developed in 6 (Group B, Fig. 3) during follow-up. Systolic gradients ranging from 16 to 80 mm Hg were measured across the bulboventricular foramen by Doppler examination or at cardiac catheterization. The mean initial bulboventricular foramen area index in these patients was 1.75 ± 0.5 cm^2/m^2 (range 1.24 to 2.64) (Fig. 4). One patient in this group (Patient B2, Table 1) had a straddling tricuspid valve that contributed to the bulboventricular foramen obstruction. The mean initial bulboventricular foramen area index in this patient was 2.64 cm^2/m^2, the largest in this group. All others had an initial index <2 cm^2/m^2.

The remaining eight patients (Group C, Fig. 3) did not develop bulboventricular foramen obstruction during follow-up. The mean initial bulboventricular foramen area index in this group was 3.95 ± 1.7 cm^2/m^2 (range 2.34 to 6.31) (Fig. 4).

The patients in Group C tended to be slightly older at the time of the first echocardiogram (mean age 1.4 months [range 0 to 4.8]) than patients in group A (mean age 0.6 month [range 0 to 4.2]) or group B (mean age 0.3 month [range 0 to 1.4]), but this difference did not achieve statistical significance ($p = 0.54$). Body size did not differ significantly among the three groups; mean body surface area was 0.24 ± 0.04 m^2

JACC Vol. 19, No. 1
January 1992:142–8

in Group C, 0.23 ± 0.03 m^2 in Group B and 0.22 ± 0.02 m^2 in Group A.

The mean initial bulboventricular foramen area index was significantly larger in patients who did not develop bulboventricular foramen obstruction (Group C) than in patients in Groups A and B ($p < 0.0001$). Although bulboventricular foramen area index tended to be larger in patients who developed obstruction late (Group B) than in patients who underwent a pulmonary artery to aorta anastomosis as neonates (Group A), this difference did not achieve statistical significance. All 11 infants judged at the initial evaluation to have a small bulboventricular foramen underwent a pulmonary artery to aorta anastomosis.

Eleven of the 12 infants who appeared to have an "adequate" bulboventricular foramen at the initial evaluation and needed a procedure to limit pulmonary blood flow underwent pulmonary artery banding. In one patient (Patient A8, Table 1) the bulboventricular foramen appeared adequate in size but was obstructed by a straddling tricuspid valve; this patient underwent pulmonary artery to aorta anastomosis. Twelve of 15 patients with aortic arch obstruction underwent a pulmonary artery to aorta anastomosis, and the remaining 3 patients underwent coarctation repair and pulmonary artery banding. Eight patients without arch obstruction had with excessive pulmonary blood flow underwent pulmonary artery banding alone. The size of the bulboventricular foramen assessed subjectively and the presence or absence of aortic arch obstruction appeared to be the major factors influencing the type of initial palliative procedure performed.

Bulboventricular foramen growth. Twelve of 14 patients in Groups B and C had two to four echocardiograms (median three) during the follow-up period. The follow-up period spanned by these serial echocardiographic examinations ranged from 4.2 to 52 months (mean 32.4). The bulboventricular foramen size was not measured after a patient underwent an operation to bypass the foramen because, in the presence of an additional systemic outflow, the size of the foramen no longer had hemodynamic importance. Furthermore, the implications of the presence or absence of bulboventricular foramen growth under the condition of reduced flow are unclear. For that reason the echocardiographic follow-up period was shorter in Group B (mean 20 months) than in Group C (mean 45 months).

During follow-up there was a small mean increase in bulboventricular foramen area (0.008 cm^2/month) (Fig. 5A). The rate of growth was not related to the initial size of the foramen and did not keep pace with somatic growth, resulting in a mean decrease in bulboventricular foramen area index of 0.05 cm^2/m^2 per month (Fig. 5B).

The mean decrease in bulboventricular foramen area index with time in the patients who developed obstruction was 0.05 ± 0.06 cm^2/m^2/month compared with 0.047 ± 0.05 cm^2/m^2/month in those who did not develop obstruction

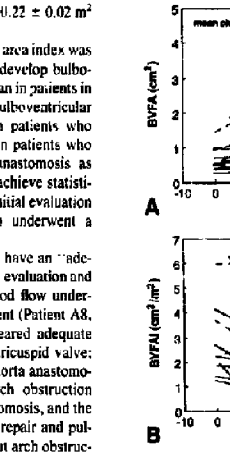


Figure 5. Plot of the regression lines for bulboventricular foramen area (BVFA) (A) and indexed bulboventricular foramen area (BVFAI) (B) versus follow-up time for the 12 patients in whom serial data were available. (The dashed lines indicate patients who underwent systemic to pulmonary artery shunt as the first procedure because of pulmonary valve stenosis.)

($p = 0.85$). Similarly there was no significant difference ($p = 0.71$) in the mean decrease in bulboventricular foramen area index with time in patients who did (0.06 ± 0.07 cm^2/m^2 per month) and did not (0.08 ± 0.06 cm^2/m^2 per month) develop bulboventricular foramen obstruction after pulmonary artery banding.

These findings indicate that the most important determinant of late bulboventricular foramen obstruction is the initial size rather than the rate of growth of the foramen. Of interest, in two of the three patients who underwent creation of a systemic to pulmonary artery shunt the bulboventricular foramen area did increase in size proportional to somatic growth.

Aortic arch obstruction and initial bulboventricular foramen size. Sixteen patients had aortic arch obstruction (coarctation or arch interruption) and 12 patients did not. The mean initial bulboventricular foramen area index was significantly ($p < 0.0001$) smaller in the patients with arch obstruction (1.05 ± 0.6 cm^2/m^2) than in patients without arch obstruction (3.35 ± 1.75 cm^2/m^2, Fig. 6).

Pulmonary artery band and initial bulboventricular foramen size. Eight patients without aortic arch obstruction underwent pulmonary artery banding. Four of these patients developed bulboventricular foramen obstruction during fol-

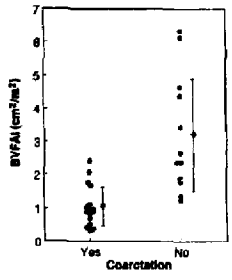


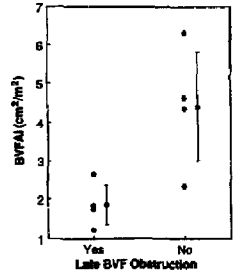
Figure 6. Indexed initial bulboventricular foramen area (BVFAi) in patients with and without coarctation of the aorta. The solid circles and error bars to the right of the data points indicate mean value ± 1 SD.

low-up and four did not. The mean initial bulboventricular foramen area index was significantly smaller ($p = 0.026$) in the group that developed bulboventricular foramen obstruction after pulmonary artery banding (1.87 ± 0.57 cm²/m²) than in the group that did not (4.41 ± 1.62 cm²/m², Fig. 7).

Discussion

Biplane echocardiographic measurements of the bulboventricular foramen were highly correlated with postmortem measurements. The difference in absolute dimensions was 10% to 12%, approximately what would be expected from formalin fixation. This observation indicates that measurement of the bulboventricular foramen area by biplane two-dimensional echocardiography accurately reflects the area of the foramen. Measuring the bulboventricular foramen size in only one plane may be misleading because the foramen is generally elliptic in shape (8). Supporting this notion is the finding that the ratio of the apex to base diameter to the transverse diameter of the bulboventricular foramen in our patients ranged between 0.6 and 1.5. Similar methods have been used successfully by others to assess the size of ventricular septal defects in patients with two ventricles (9).

Figure 7. Indexed initial bulboventricular foramen (BVF) area (BVFAi) in patients without aortic arch obstruction who underwent pulmonary artery banding as infants. The indexed area was significantly smaller in patients who developed bulboventricular foramen obstruction late after banding. Symbols as in Figure 6.



Mechanism of bulboventricular foramen obstruction. Patients may have an excessively small bulboventricular foramen as neonates or obstruction may develop later. Our data suggest that the mechanism of late obstruction in most cases is failure of the bulboventricular foramen to grow in proportion to somatic growth. Subaortic stenosis can also be due to obstruction within the outflow chamber (10) or encroachment of straddling atrioventricular (AV) valve tissue or other fibrous tissue on the bulboventricular foramen (11). In one of our patients straddling AV valve tissue produced moderate obstruction of an intrinsically large bulboventricular foramen. Such instances may not be appreciated by measuring the size of the bulboventricular foramen alone and point out the continued need for Doppler evaluation.

Results of bulboventricular foramen obstruction. Obstruction of the bulboventricular foramen may result in decreased compliance of the left ventricle as a result of hypertrophy and fibrosis (1). Significant left ventricular hypertrophy may necessitate a separate surgical intervention to enlarge or bypass the bulboventricular foramen before a Fontan procedure is attempted (to permit regression of hypertrophy and normalization of chamber compliance). These procedures have been associated with high mortality rates (1–4) although improved survival time has been noted in more recent reports (10,12).

Surgical options. Patients with double-inlet left ventricle who are not treated surgically have a very poor prognosis (13). Most infants with single left ventricle or tricuspid atresia require surgical palliation in infancy before a Fontan operation can be undertaken. It is often difficult to decide which type of palliative procedure to perform in infants without pulmonary stenosis. If bulboventricular foramen obstruction is likely to develop then pulmonary artery to aorta anastomosis is probably the preferred initial palliative operation. However, if bulboventricular foramen obstruction is unlikely to develop, pulmonary artery banding seems preferable because of the lower initial mortality rate.

A valid comparison of the results of pulmonary artery banding versus those of pulmonary artery to aorta anastomosis in the neonate cannot be obtained from this report. First, our study patients represent only a subset of patients with single ventricle who presented to our institution (only those who presented early with an echocardiogram at our institution). Second, all our patients who underwent pulmonary artery to aorta anastomosis also had aortic arch repair, which made the operation more complex. However, our data

JACC Vol. 19, No. 1
January 1992:142–8

do indicate that the initial size of the bulboventricular foramen is an important predictor of late obstruction.

The decision regarding which type of palliation to perform has been based largely on qualitative assessment of the bulboventricular foramen size and the presence or absence of aortic arch obstruction. Previous reports (4,14–16) indicate that arch obstruction is usually associated with a small bulboventricular foramen. This retrospective study indicates that measuring the bulboventricular foramen with biplane two-dimensional echocardiography is a more reliable means of predicting which patients will develop obstruction of the bulboventricular foramen.

Pulmonary artery banding and bulboventricular foramen obstruction. It has been reported previously that even patients without aortic arch obstruction are prone to develop obstruction of the bulboventricular foramen after pulmonary artery banding (17,18) or the Fontan operation (2,4). Hypertrophy induced by the pulmonary artery band has been proposed as the mechanism for the bulboventricular foramen obstruction in patients with banding. However, our study patients who underwent pulmonary artery banding as the initial palliative procedure and then developed bulboventricular foramen obstruction had a significantly smaller initial bulboventricular foramen area index than those who did not develop obstruction. Furthermore, patients with a small initial bulboventricular foramen area index can develop bulboventricular foramen obstruction even without pulmonary artery banding (Patient B1, Table 1). These findings suggest that a small initial bulboventricular foramen may be a more important predictor of late bulboventricular foramen obstruction than having pulmonary artery banding.

Conclusions. Patients with an initial bulboventricular foramen area index <2 cm^2/m^2 are at high risk of developing bulboventricular foramen obstruction. Because of the relatively short follow-up period (2 to 5 years) in this study, it is possible that some of the patients who have not yet developed obstruction may do so in the future. Consequently, a lower limit for adequate bulboventricular foramen size cannot be established from these data. However, it appears that an initial bulboventricular foramen area index <2 cm^2/m^2 is highly predictive of subsequent obstruction.

References

1. Barber G, Hagler DG, Edwards WD, et al. Surgical repair of univentricular heart (double inlet left ventricle) with obstructed anterior subaortic outlet chamber. J Am Coll Cardiol 1984;4:771–8.
2. Rothman A, Lang P, Lock JE, Jonas RA, Mayer JE, Castaneda AR. Surgical management of subaortic obstruction in single left ventricle and tricuspid atresia. J Am Coll Cardiol 1987;10:421–6.
3. Jonas RA, Castaneda AR, Lang P. Single ventricle (single or double inlet) complicated by subaortic stenosis: surgical options in infancy. Ann Thorac Surg 1985;39:361–6.
4. Penkoske PA, Freedom RM, Williams WG, Trusler GA, Rowe RD. Surgical palliation of subaortic stenosis in the univentricular heart. J Thorac Cardiovasc Surg 1984;87:767–81.
5. Bethea CM, Reynolds LJ. Treatment of bulboventricular stenosis by ventricle-ascending aorta valved conduit bypass. Ann Thorac Surg 1989;47:765–6.
6. Van Praagh R, David I, Van Praagh S. What is a ventricle? The single ventricle trap. Pediatr Cardiol 1982;2:79–84.
7. Van Praagh R, Plett JA, Van Praagh S. Single ventricle: pathology, embryology, terminology and classification. Herz 1979;4:113–30.
8. Freedom RM. The dinosaur and banding of the main pulmonary trunk in the heart with functionally one ventricle and transposition of the great arteries: a saga of evolution and caution. J Am Coll Cardiol 1987;10:427–9.
9. Sharif DS, Huhta JC, Marantz P, Hawkins HK, Yoon GY. Two-dimensional echocardiographic determination of ventricular septal defect size: correlation with autopsy. Am Heart J 1989;117:1333–6.
10. Cheung CH, Lincoln C, Anderson RR, Siew Yen Ho, et al. Options for surgical repair in hearts with univentricular atrioventricular connection and subaortic stenosis. J Thorac Cardiovasc Surg 1990;100:672–81.
11. Freedom RM, Dische MR, Rowe RD. Pathologic anatomy of subaortic stenosis and atresia in the first year of life. Am J Cardiol 1977;39:1031–43.
12. Waldman JD, Lamberti JJ, George L, et al. Experience with Damus procedure. Circulation 1988;78:32–9.
13. Moodie DS, Ritter GD, Tajik AJ, O'Fallon WM. Long-term follow-up in the unoperated univentricular heart. Am J Cardiol 1984;53:1124–8.
14. Freedom RM, Rowe RD. Morphological and topographical variations of the outlet chamber in complex congenital heart diseases: angiocardiographic study. Cathet Cardiovasc Diagn 1978;4:345–71.
15. Freedom RM. The morphologic and therapeutic implication of an obstructive anomaly of the aortic arch in infants with complex congenital heart disease. In: Parenzan L, Crupi GC, Graham G, eds. Congenital Heart Disease in the First Three Months of Life: Medical and Surgical Aspects. Bologna: Patron Editor 1981:341–61.
16. Rudolph AM, Heyman MA, Spitznas U. Hemodynamic consideration in the development of narrowing of the aorta. Am J Cardiol 1972;30:514–25.
17. Freedom RM, Benson LN, Smallhorn JF, Williams WG, Trusler GA, Rowe RD. Subaortic stenosis, the univentricular heart, and banding of the pulmonary artery: an analysis of the course of 43 patients with univentricular heart palliated by pulmonary artery banding. Circulation 1986;73:758–64.
18. Freedom RM, Sondheimer H, Dische R, Rowe RD. Development of "subaortic stenosis" after pulmonary arterial banding for common ventricle. Am J Cardiol 1977;39:78–83.