Fate of the pericardial monocusp pulmonary valve for right ventricular outflow tract reconstruction

Early function, late failure without obstruction

Sudden change from a pressure-loaded to a volume-loaded right ventricle as a result of transannular patch reconstruction of the right ventricular outflow tract may result in early hemodynamic compromise. Trileaflet porcine valves in conduits function well early but typically create late obstruction. We studied the fate of a pericardial monocusp valve constructed during transannular patch reconstruction for right ventricular outflow tract obstruction in 19 patients, 2 weeks to 27 years of age (mean age 61 months). Patients had the monocusp constructed of autologous ($n = 16$) or bovine pericardium ($n = 3$) when the former was not available during transannular patch reconstruction associated with repair of tetralogy of Fallot ($n = 12$), pulmonary stenosis/atresia ($n = 4$), and truncus arteriosus ($n = 3$). Function of the monocusp was assessed by presence of a split-second heart sound, echocardiographic assessment of right ventricular dilatation, monocusp competence, and fluoroscopic evaluation of monocusp motion. Functional assessments were accomplished immediately after the operation and at 2, 6, 12, and 24 months after the operation. There were no operative deaths, but there was one late hospital death. Sixteen of nineteen patients (84 %) had competent pulmonary monocusp valves immediately after the operation, but, by 24 months, only one of seven patients (14 %) had a competent valve. No patient had monocusp stenosis. We conclude that a pericardial monocusp valve for right ventricular outflow tract reconstruction provides excellent early hemodynamic function but that these effects are limited in duration. Because late stenosis has not been seen, this inexpensive and easily constructed valve can be used as an excellent short-term adjunct to right ventricular outflow tract reconstruction. (J THORAC CARDIOVASC SURG 1994;107:908-13)

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The use of a transannular patch (TAP) to reconstruct the right ventricular (RV) outflow tract (OT) for patients

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in whom the pulmonary anulus is deemed inadequate to handle full cardiac output has been used since the origins of congenital heart operations.^{1,2} Nevertheless, the immediate change from a pressure-loaded to a volume-loaded RV with a TAP, in concert with a ventriculotomy, may result in significant hemodynamic compromise.^{1,3-8} A competent pulmonary valve mechanism is one means by which early compromise of the RV may be reduced, thus avoiding the clinical morbidity and potential mortality associated with TAP.^{7,8} Some surgeons choose to leave an imperfect pulmonary valve and anulus with

Table I. Patient profiles

Patient No.	Age (mo)	Preoperative diagnosis	Date of operation	Type of operation	Status
1	12	TOF with RVOT obstruction and PFO	1977	C	Alive
2	207	TOF s/p Blalock Shunt	1980	B	Alive
3	27	TOF with RVOT obstruction	1981	B	Alive
4	93	TOF with RVOT obstruction	1981	B	Alive
5	312	TOF with aorto-pulmonary collaterals	1985	B	Dead
6	54	TOF with RVOT obstruction s/p Blalock shunt	1989	B	Alive
7	9	Pulmonary valve stenosis	1989	A	Alive
8	0.5	Pulmonary atresia with persistent ductus arteriosus	1989	D	Alive
9	2	Pulmonary valve stenosis	1989	A	Alive
10	13	TOF with pulmonary valve atresia; s/p Blalock shunt	1990	B	Alive
11	44	TOF with absent pulmonary valve	1991	B	Alive
12	30	TOF with RVOT obstruction	1991	B	Alive
13	16	TOF with RVOT obstruction	1991	B	Alive
14	3	Truncus arteriosus type I	1991	A	Alive
15	6	TOF with absent pulmonary valve	1992	B	Alive
16	1	Truncus arteriosus type I	1992	A	Alive
17	204	TOF w/RVOT obstruction s/p Waterson shunt	1993	B	Alive
18	24	Pulmonary atresia with intact ventricular septum	1993	A	Alive
19	1	Truncus arteriosus type II	1993	A	Alive

C, Tetralogy of Fallot repair with RVOT reconstruction with a monocusp and closure of patent foramen ovali; s/p, status post; B, tetralogy of Fallot repair with RV outflow tract reconstruction with a monocusp; A, RVOT reconstruction with a monocusp or direct pulmonary artery -RV connection with monocusp; D, ligation of persistent ductus arteriosus and RVOT reconstruction with a monocusp.

associated residual outflow obstruction because of the recognized early morbidity associated with TAP, whereas others implant a valved homograft or conduit.^{4,8-16} Unfortunately, homografts are scarce and expensive. Heterograft valved conduits are also expensive and invariably lead to late obstruction requiring reoperation. With the use of generous monocusp of autologous or bovine pericardium as part of a TAP repair, early hemodynamic benefits might also be realized, but the early and late function of these valves is unknown. The purpose of this study is to present our experience with pericardial monocusp valves incorporated into TAP repairs of congenital heart defects, outline some technical considerations, and evaluate potential advantages of this modification in technique.

Patients and methods

We retrospectively analyzed 19 patients undergoing operation involving monocusp insertion between 1972 and 1993. There were six male patients and 13 female patients between 11 days and 27 years of age (mean, 61 months) (Table I). The experience includes eight infants less than 1 year of age, and one adult over 18 years of age. Five patients had received a previous palliative operation, including three Blalock-Taussig operations,

one Waterson operation, and one polytetrafluoroethylene central shunt.* All nineteen patients had transannular patching with a monocusp in conjunction with definitive repair of tetralogy of Fallot ($n = 12$), pulmonary stenosis/atresia ($n = 4$), or truncus arteriosus ($n = 3$), whenever pulmonary vascular resistance or imperfect pulmonary arterial anatomy suggested that a valve in the pulmonary circuit would be useful (Table I). The monocusp was constructed of autologous pericardium ($n = 16$) or bovine ($n = 3$) pericardium when the former was unavailable. The pericardial "leaflet" was sewn into the free space of the right ventriculotomy before construction of the TAP. The site of intended coaptation of the monocusp with the back wall of the pulmonary anulus was measured, and the monocusp was cut into a redundant triangle and sewn to the edge of the right ventriculotomy so that the leading edge of the monocusp would rest against the posterior pulmonary anulus during diastole and billow fully open against the overlying TAP during ventricular systole. This natural motion was aided by placing four to six small titanium clips along the redundant leading edge of the monocusp.

After insertion of the monocusp, a TAP of autologous or bovine pericardium was used to roof over the right ventriculotomy and pulmonary arteriotomy. After the operation, monocusp function was assessed by auscultating for the presence of

*Gore-Tex shunt, registered trademark of W.L. Gore & Associates, Inc., Newark, Del.

Table II. Presence of pulmonary incompetency by echocardiography, catheterization, or clinical exam

	No. of patients	Trace-to-mild	Mild-to-moderate	Moderate-to-severe
Perioperative period	19	16	2	1
2 mo	14	7	6	1
6 mo	10	0	9	1
1 year	10	0	6	4
18 mo	9	0	6	3
2 years	7	1	2	4

a split-second heart sound, auscultation of pulmonary insufficiency, echocardiographic evaluation of RV dilatation and monocusp competence, fluoroscopic observation of valve motion immediately after the operation and at 2, 6, 12, and 24 months after the operation. A gross estimate of hemodynamic stability is reflected in the number of inotropic agents required after the operation.

Results

There were no intraoperative or immediately postoperative deaths. There was one late hospital death in a 27-year-old patient who required reopening of the ventricular septal defect for postoperative pulmonary hypertension. No inotropic drugs were required in 8 of 19 patients (42%). An additional 6 of 19 (31%) were given only 2 to 3 $\mu\text{g/kg}$ per minute of dopamine during the postoperative period according to one surgeon's preference. Postoperative stay ranged from 3 to 18 days (mean 9.0 days). There have been no late deaths. Follow-up was complete except for one child who returned to China immediately after the perioperative period. Follow-up ranged from 1 month to 16 years (mean 3 years). Only four of eighteen patients (21%) were receiving medications: one patient was receiving digoxin and three were receiving diuretics at the time this article was written. Immediately after the operation, 13 of 19 (78%) patients had a split second heart sound, whereas only one of seven (14%) patients had a split second heart sound at 24 months (Table II). Of the 19 patients assessed with echocardiography, auscultation, or both immediately after operation, 16 (84%) had no appreciable pulmonic incompetence, but, by 6 months, regurgitation invariably occurred. At 24 months, only one of seven (14%) patients continued to have a competent pulmonary valve mechanism. Two of nineteen (10%) patients had pulmonary incompetence within days of the operation, and, by 24 months, two patients had mild-to-moderate incompetence of the monocusp, and four patients had moderate-to-severe incompetence of the monocusp. During follow-up, for up to 16 years, no patient had a gradient at the monocusp site or calcification at the monocusp site.

Discussion

A significant component of tetralogy of Fallot is pulmonary valve stenosis or atresia.² Although clinical severity of the RVOT obstruction varies, severe hypoplasia or atresia of the pulmonary valve anulus almost always requires TAP reconstruction.

In 1955, the TAP was introduced by Kirklin and Lillehei and their associates^{2,23} as a method of relieving RVOT obstruction or of reestablishing RV-pulmonary artery continuity. Yet, since the introduction of this technique, controversy has existed regarding the advisability and efficacy of this procedure in relieving RVOT obstruction at the expense of producing pulmonary insufficiency.^{2,30,31}

Surgical correction that includes a TAP creates a new volume load to the RV. This acute conversion often requires a period of time for RV recovery and adaptation as seen in the clinical findings of Fuster and associates¹ and Poirier, McGoon, and Danielson³ at the Mayo Clinic and the experimental results of Austen,⁸ Bender,⁷ Ellison,⁵ and their associates. In contrast to these acute problems, clinical and experimental chronic pulmonary insufficiency has been found to be well tolerated.^{1,3,5,17-30} Indeed, only 3 of 396 patients with transannular patches who were followed up to a maximum of 22 postoperative years at the Mayo Clinic had morbidity caused by RV dilatation.¹

Because of the acute detrimental effects of a ventriculotomy and TAP patch, there is little question that a pulmonary valved conduit reconstruction of RV-pulmonary artery continuity represents the most anatomically and physiologically complete restoration of the pulmonary circuit currently achievable. However, valved conduits, while facilitating a satisfactory initial result, nevertheless, became the major limitation of a durable repair.^{31,32} Valved conduits almost invariably need replacement because of patient growth or intrinsic deterioration.⁹⁻¹⁷ They are expensive, and some (homografts) are in short supply. Bissett and associates¹² have described a 70% prevalence of conduit failure within 6 years, whereas Jonas and associates⁹ found that by 10 years, all conduits required replacement.

In our group of 19 patients treated with a TAP monocusp, 17 had complete perioperative monocusp competence. The majority of patients had significant pulmonary incompetence by 6 months after the operation. The echocardiogram at that time in most patients showed a nonfunctioning monocusp valve, with the cusp sealed in the open position on the underside of the TAP. Although obstructions are observed with valved conduits, no monocusp obstruction has been observed in late follow-up. Hence, while the monocusp valve does not continue to

function for a long period of time, its failure does not require reoperation. It provides function when it is most critically needed in the early postoperative period, then "gets out of the way." Thus, advantages of the monocusp valve TAP technique include the potential for early support of RV function, with addendant reduction in perioperative morbidity and mortality and, most importantly, reduction in late RVOT obstruction from conduit failure.

The monocusp may allow a surgeon to be more aggressive in the initial complete relief of RVOT obstruction, rather than accept an imperfect pulmonary anulus. In fact, long-term studies of tetralogy of Fallot show that most late morbidity and mortality can be traced to residual RVOT obstruction.^{2,4} Monocusp insertion is easily accomplished and is inexpensive. The monocusp thus appears to have all of the advantages of a valved conduit but without the late obstruction leading to reoperation.

We conclude that a pericardial monocusp valve for RVOT reconstruction provides excellent early hemodynamic function and benefit. These effects are limited, and failure of the monocusp in the form of pulmonary valve incompetence is the rule. However, monocusp failure is not accompanied by OT obstruction, therefore, to date, no reoperation has been required. Thus, appropriate application of a monocusp valve may lessen the perioperative adverse effects associated with transannular patching without resorting to valved conduits with their attendant late morbidity.

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Discussion

Dr. Lorenzo Gonzalez-Lavin (*Huntington, W. Va.*). Dr. Gundry, I wish to commend you on your results with the use of a pericardial monocusp as a pulmonary valve for RVOT reconstruction. I have one question, which is mainly with the use of homografts in patients with pulmonary hypertension who need RVOT reconstruction. Our past experience with aortic and pulmonary homograft valve conduits has been gratifying. My question to you is, in an institution where there is a very active cardiac transplantation program in which homograft valves can be retrieved easily, why not use homografts in the cohort of patients that you presented?

Dr. David N. Campbell (*Denver, Colo.*). You correctly point out that children who require TAPs to relieve significant RVOT obstruction have early volume overload, which, combined with ventriculotomy, often causes temporary dysfunction of the RV. The pericardial monocusp technique you described and other monocusp techniques with the use of a bovine pericardium, described by Ionescu in 1985, pulmonary wall, described by the Denver group in 1986, and others in the past deal with this problem effectively in the early postoperative period. Late outcome in all of those studies have been similar to your experience with chronic pulmonary insufficiency.

However, this paper does raise several issues. The first has to do with the decreasing prevalence of early mortality over this period of time. Increased experience, modification of the surgical technique, changes in myocardial protection, and better postoperative care have reduced operative mortality in these children to an acceptably low level (in the range of 3% to 5%) as described in a recent combined study from Alabama and Boston. In this same study, published in this JOURNAL (1992;103:706-23), transannular patching was not a risk factor in multivariate analysis. However, I realize that the feeling about TAPs and operative risk is still somewhat controversial.

The second issue has to do with the "no harm, no foul" strategy; that is, although the monocusp patch seems to function only in the early postoperative period, pulmonary insufficiency does occur chronically in a high number of patients. Is this sufficient to justify its use if operative mortality is not significantly affected? In Denver, we would use a valved allograft conduit for children who we believe have decreased RV function or pulmonary hypertension. In this situation, a functioning pulmonary valve may be required for a longer period of time. The pulmonary allograft is best for long-term use in this situation because failure caused by insufficiency and not by calcification or stenosis, as may occur in the aortic homograft. Our results of pulmonary homografts recently reviewed by Albert show excellent long-term function with a low (6%) reoperation rate.

This is necessary, however, in only a small number of patients. Fuster, in a review in 1980 of results at the Mayo Clinic and Alabama over a span of 22 years, showed only 1% of patients had major problems caused by chronic volume overload, which is nearly the same figure as is found in the review by Kirklin and Castaneda in 1992 (2%).

Therefore, I have four questions: (1) What criteria did you use over those 21 years to place the monocusp pulmonary valve? Is it possible that they were preselected for better outcome? (2) Do you believe that your low mortality is from the patch or better overall preoperative, operative, and postoperative care? (3) Although this study covers 22 years, the mean follow-up is only 3 years. Do you feel this is sufficient time to see obstruction develop from calcification of bovine or human pericardium? We know that, in other situations where human or bovine pericardium is used, this does occur. (4) Finally, how many of the overall patients at your institution who have undergone repair of tetralogy of Fallot or pulmonary atresia/stenosis have required valve replacement for chronic volume overload as described by Greg Mishbach, Ed Bovie and others?

Dr. Gundry. Dr. Gonzalez-Lavin asked why we did not use a homograft in these children instead of the pericardial monocusp valves, seeing as how we are a major transplant center and have access to homografts. As we have demonstrated in the discussion portion of our manuscript, the use of homografts, or other alternative conduits for reestablishing RV-pulmonary artery continuity, though appealing in the perioperative period, has not had beneficial long-term effects. Homografts, unfortunately, do not grow and almost always calcify, with the result that most have to be replaced several years after the operation. Moreover, it is clear that the need for a valve within the pulmonary circulation is in the first few days to weeks after the operation, while the RV adjusts to the ventriculotomy and closure of a ventricular septal defect. Long-term studies suggest that less than 1% of patients with transannular patches and complete pulmonary incontinence have any morbidity from this condition.

In answer to Dr. David Campbell's questions, initially, as mentioned in the manuscript, these monocusp valves were placed in patients who were believed to have elevated pulmonary vascular resistance or had diminished runoff through the pulmonary vascular tree as a result of multiple stenoses or other hypoplastic pulmonary arteries, which would make pulmonary arterial hypertension likely in the postoperative period. As our experience has increased with this monocusp valve, we have been using it almost routinely on repairs of tetralogy of Fallot, truncus arteriosus, or pulmonary atresia. Because the follow-up is too short, these patients are not included in this manuscript. So, in that sense, the patients reported here were actually preselected to have a potentially worse outcome from their less-than-adequate pulmonary arterial tree. In answer to your second question, we believe that the low mortality that we report in this manuscript is directly attributable to the monocusp valve. Indeed, anecdotal side-by-side comparisons of patients who have had a monocusp valve versus an unvalved TAP have convinced us that the monocusp valve results in protection against RV failure of the sort commonly seen in the immediate postop-

erative period with the TAP alone. In answer to your third question, regarding the mean follow-up time of 3 years, we have yet to see calcification of these monocusp valves. Long-term follow-up of TAPs constructed from autologous pericardium would suggest that calcification of this patch is rare, and we would think that this same thing would apply to the monocusp valve. Finally, at our institution we have not had patients with TAPs return for the placement of a valve within the pulmonary circuit for chronic volume overload. We have, however, seen patients with absent pulmonary valve syndrome and tetralogy of Fallot who have returned for placement of a valve within the pulmonary circuit. I think these patients' conditions represent a completely different spectrum of pulmonary incompetence.

I think that the best answer to all of the discussants' questions lies in the fact that homografts are expensive and they are rare. If results comparable with or better than those of the homograft conduit replacement can be achieved with the patient's autologous tissue at no additional charge to the patient, then this is a method that is well worth considering during reconstruction of the RVOT and pulmonary RV connections.