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Clinical and Hemodynamic Follow-up of Left Ventricular to Aortic Conduits in Patients With Aortic Stenosis

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To assess the long-term results of left ventricular outflow tract reconstruction utilizing an apical left ventricular to aortic valved (porcine) conduit the clinical and hemodynamic data were reviewed from 24 patients who had placement of an apico-aortic conduit. Eighteen of the patients are asymptomatic and taking no cardiac medications. Three patients were reoperated on, one patient 1.5 years after his original operation for subacute bacterial endocarditis and two patients 3 to 4 years after their original operation for severe conduit valve insufficiency. None of the patients is taking anticoagulants and no thromboembolic events have occurred. Postoperative catheterization has been performed 1 to 1.5 years (mean 1.2) after repair in 15 of 21 patients. The rest left ventricular outflow tract gradient has decreased from 102.5 ± 20 mm Hg preoperatively to 14.8 ± 9.9 mm Hg postoperatively (probability [p] < 0.001). Some degree of conduit obstruction was demonstrated by catheter passage in 11 of the 15 patients. In these 11 patients, the obstruction occurred at three distant sites: at the egress of the left ventricle in 9, at the porcine valve in 5 and at the aortic to conduit junction in 1. Isometric exercise in five and supine bicycle exercise in six patients increased the left ventricular outflow tract gradient by 2.5 ± 1.1 and 20.8 ± 11.8 mm Hg, respectively, despite an increase in cardiac index of 1 ± 0.3 and 3.7 ± 0.4 liters/min per m^2, respectively. The data suggest that a left ventricular to aortic conduit is an effective form of therapy for severe left ventricular outflow tract obstruction.

The use of a valved conduit to connect the apex of the left ventricle to the aorta has proved a valuable alternative for the treatment of left ventricular outflow tract obstruction not readily amenable to conventional surgery (1–8). There is limited information on the long-term follow-up of patients with a left ventricular to aortic conduit (1,2,9,10). Stansel et al. (2) reported the late development of peripheral emobli in three of four children who received such a conduit to relieve their aortic valve disease. Other reported sequelae have included bacterial endocarditis of the porcine valve (1,2) and pseudoaneurysm formation of the left ventricular apex graft anastomosis (11). In addition, there is only limited information regarding late postoperative hemodynamics (1,2) and no information on the hemodynamic response to exercise in patients with an apico-aortic conduit. In this report we review the clinical course and rest and exercise hemodynamic data of 24 patients who have had an apico-aortic conduit.

Methods

Study patients (Tables 1 and 2). Between December 1976 and January 1982, 24 patients had their left ventricular outflow tract reconstructed using a porcine valve composite conduit to connect the apex of the left ventricle to the aorta. All 24 patients were symptomatic (syncope in 6, angina in 19 and/or severe dyspnea on exertion in 24); all had a left ventricular outflow tract that was not readily amenable to repair by conventional methods (resection of subaortic membrane or aortic valve replacement). Severe anular aortic stenosis was present in 9 patients (Cases 1, 3, 5, 7, 11, 12, 14, 17, 18), severe long-segment fibromuscular subaortic stenosis in 14 patients (Cases 4, 6, 8, 9, 10, 13, 15, 16, 19 to 24) and Patient 2 had the combination of severe valvular aortic stenosis and severe coarctation of the thoracic aorta. Twenty-one of the 24 patients had had one or two previous aortic valvotomies or left ventricular outflow tract operations, or both.

Surgical procedure. All surgery was performed at either the University of Michigan or the University of Indiana Medical Center.

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Table 1. Clinical and Laboratory Data in 15 Patients ≥ 1.5 Years Postplacement of a Left Ventricular to Aortic Conduit

Case	Diagnosis	Symptoms Preconduit	Conduit—Stent and Valve Size (mm); Site	Prior Cardiac Operations	Age/Weight (yr/kg) at Operation	Follow-up (yr/kg)	Symptoms Postconduit	U/A	Renal Scan	Renal Angio	Conduit Valve Insufficiency
1	AS (Val), AL (T)	Angina, DOE	18/20; Des Ao	Ao valvotomy	15/65	4.5/70.2	None	NL	NL	NL	None
2	AS (Val), coart, MI (M)	PND, CHF, DOE, angina	20/20; Des Ao	None	30/82.3	1 2/84	1.2 yr SOB, CHF fever	NL	—	—	None
3	AS (Val), AI (M)	Angina, DOE	118; Abd Ao	Ao valvotomy	12.5/36.8	5/45	None	NL	NL	NL	2+ (3 yr postop)
4	Sub AS, AI (M), MI (M)	Angina, DOE	120; Abd Ao	Sub Ao resection	16/69.4	4.1/72.1	DOE, angina, 3 yr postop	NL	NL	NL	4+ (3 yr postop)
5	AS (Val), AI (M)	DOE, angina	122; Des Ao	Ao valvotomy	14.3/50.7	3.5/60.2	None	NL	NL	NL	None
6	Sub AS, AI (M)	DOE, angina	118; Des Ao	Sub Ao resection	8.5/27.2	4.5/40	DOE, syncope 4 yr postop	NL	—	—	4+ (3.9 yr postop)
7	AS (Val), AI (M)	Angina	116; Des Ao	Ao valvotomy	11/43.7	3 5/56.5	None	NL	NL	NL	None
8	Sub AS, VSD, DCRV + 60, AI (mod)	Angina, DOE	118; As Ao	None	8/20.1	5.5/47.2	None	NL	—	NL	None
9	Sub AS, AI	Angina, DOE, syncope	18/20; As Ao	Sub Ao resection	12.2/30.1	4.5/54	None	NL	NL	NL	None
10	Sub AS	Angina, DOE, palpitation	16/18; Des Ao	Sub Ao resection (× 2)	11/28.2	4.5/62.1	None	NL	NL	NL	None
11	AS (Val)	DOE	14/16; Des Ao	Ao valvotomy	7/16.1	3.3/35	None	NL	—	—	None
12	AS (Val), AI (M)	DOE	18/20; Des Ao	Ao valvotomy	17/75.2	3/74	None	NL	—	—	None
13	AS (Val & subval), AI (M)	DOE	18/20; Des Ao	Ao valvotomy, sub Ao resection	19/65.3	2 5/66	None	NL	—	—	None
14	AS (Val), PS (Val), PI (M), AI (M)	Syncope, angina	14/16; Abd Ao	Ao valvotomy, pulmonary valvotomy (× 2)	5.5/14.2	2/16.2	None	NL	NL	NL	None
15	Sub AS, AI (M)	Syncope, angina, VT	18/20; Des Ao	Sub Ao resection (× 2)	19/73	1.5/75 3	None	NL	NL	—	None

Abd = abdominal, AI = aortic insufficiency; Angio = angiography, Ao = aortic; AS = aortic stenosis, As = ascending, CHF = congestive heart failure, Coarct = coarction of the aorta, DCRV = double chambered right ventricle, Des = descending, DOE = dyspnea on exertion, M = mild, MI = mitral insufficiency; NL = normal, PI = pulmonary insufficiency, PND = paroxysmal nocturnal dyspnea, postop = postoperative, PS = pulmonary stenosis, S = severe, SOB = shortness of breath, Sub Ao = subaortic, Sub AS = subaortic stenosis, subval = subvalvular, T = trivial, U/A = urine analysis, Val = valvular, VSD = ventricular septal defect, VT = ventricular tachycardia.

There were only three deaths in the group, all occurring in the immediate postoperative period: one due to unrecognized cortriatratium, one due to pseudomonas sepsis and pneumonia and one due to disseminated intravascular coagulation and low cardiac output (Table 2). The 15 patients who have been followed up from 1.5 to 5.5 years (mean ± standard deviation 3.4 ± 0.3) after operation form the basis of this report. The surgical repair was performed using the techniques described by Brown and co-workers (4–7).

A woven Teflon tube graft containing a glutaraldehyde sterilized porcine valve (Hancock) of appropriate size was first sutured to the aorta utilizing a partial occlusion clamp and multiple mattress sutures buttressed with pledgets. The site of insertion was the descending thoracic aorta at the diaphragm in 16 patients in whom insertion of an apico-aortic conduit was the only procedure planned (Tables 1 and 2). A median sternotomy was used when an additional cardiac abnormality was repaired, in which case the site of anastomosis was the abdominal aorta (four patients) or ascending aorta (four patients) (Table 3). On cardiopulmonary bypass, a cloth-covered, rigid, angled connector (Hancock) was inserted into a hole cut in the left ventricular apex with a circular knife. This was secured with deep mattress sutures supported by pledgets. Care was taken to excise a circle of endocardium in making the ventriculotomy and to position the connector so that it projected

Table 2. Clinical and Laboratory Data in Six Patients More Than 1.5 Years Postplacement of a Left Ventricular to Aortic Conduit and in Three Patients Who Died

Case	Diagnosis	Symptoms Preconduit	Conduit Stent and Valve Size (mm) and Site	Prior Cardiac Operation	Age at Operation (yr)	Follow-up (yr)	Symptoms Postconduit	U/A	Renal Scan	Renal Angio	Conduit Valve Insufficiency
16	Sub AS, AI (mod)	Angina, CHF	18/20; Des Ao	None	12	1	None	NL	—	—	None
17	AS (supra-valvar and Val)	Angina, DOE	12/14; Des Ao	Patch repair of Ao root	5	1.2	None	NL	—	—	None
18	AS (Val), AI (S)	DOE, angina	18/22; As Ao	Ao valvotomy 2	19	1.2	None	NL	—	—	None
19	Sub AS, (AI, mild)	DOE, angina	18/22; Des Ao	Subvalvular mem /1	14	1	None	NL	—	—	None
20	Sub AS	DOE, angina	16/18; Des Ao	Sub Ao resection /1	13	1.1	None	NL	—	—	None
21	Sub AS (mild)	DOE, angina	18/20; Des Ao	Sub Ao resection /1	17	0.5	None	NL	—	—	None
22	Sub AS, ASD, cor triatriatum	DOE	12/14; Abd Ao	None	4	Died 12 h postop: cor triatriatum, unrecognized					
23	Sub AS, AI (S)	CHF, DOE, angina	16/20; As Ao	Sub AS/1, coarct/1	17	Died 8 days postop: DIC, low output CVA					
24	Single V, interrupted Ao arch, Sub AS	CHF	10/12, Des Ao	Repair Ao arch	0.3	Died 5 wk postop: pseudomonas sepsis and pneumonia					

CVA = cerebral vascular accident; DIC = disseminated intravascular coagulation; mem = membrane; V = ventricle. Other abbreviations as in Table 1.

into the left ventricular cavity several millimeters beyond the endocardium. Finally, the two prostheses were trimmed to appropriate lengths and sutured together.

Follow-up and cardiac catheterization. The hospital records, operative reports, catheterization data and angiograms were thoroughly reviewed, specific attention being paid to clinical status, chest X-ray films, electrocardiograms, urine analysis, renal scans and postoperative cardiac catheterization data. The 15 long-term survivors have had right and left heart catheterization, left ventricular angiography and descending or ascending aortic angiography, or both, 0.7 to 1.5 years (mean 1.0) after placement of the apico-aortic conduit. An additional abdominal aortogram was performed to visualize the renal arteries in 8 of 15 patients.

The following hemodynamic measurements were made during the catheterization: 1) right atrial, right ventricular, pulmonary artery, left ventricular and aortic pressures and oxygen saturations were measured in all 15 patients; 2) cardiac output was measured by thermodilution in all 15 patients; 3) sites of potential conduit obstruction were localized by pressure tracing during catheter passage through the conduit in 11 of the patients; and 4) supine exercise was performed by 6 patients and isometric exercise was performed by 5 patients. Supine exercise was performed using a variable resistance bicycle ergometer at a work load designed to raise the heart rate to between 140 and 160 beats/min. Isometric handgrip exercise was performed using a hand dynometer of 50% of maximal voluntary capacity.

Statistical analysis. Data are expressed as mean ± standard error of the mean. Significance was evaluated by either the Student *t* test or the chi-square test.

Results

Clinical course. Tables 1 and 2 summarize the clinical course of the 24 patients who had placement of a porcine valve composite conduit connecting the apex of the left ventricle to the aorta. There was initial symptomatic improvement in all 21 surviving patients. However, three patients (Cases 2, 4, 6) became symptomatic (1.1, 3.5 and 3.9 years, respectively) after surgical placement of the apico-aortic conduit (Tables 4 and 5).

Patient 2 developed fever, shortness of breath, paroxysmal nocturnal dyspnea and three pillow orthopnea 14 months after surgery. Although blood cultures were negative, he received two 6 week courses of systemic antibiotic agents. Because of persistent congestive heart failure, a second postoperative cardiac catheterization was performed (Table 5). In addition to a 55 mm Hg left

Table 3. Additional Cardiac Lesions Repaired at the Time of Left Ventricular Outflow Tract Reconstruction Using an Apico-Aortic Conduit

Case	Additional Cardiac Lesion Repaired
3	Attempted aortic valvotomy
4	Attempted aortic valvotomy
8	Resection of anomalous muscle bundle in right ventricle, ventricular septal defect closure and aortic valve plication
9	Aortic valve plication
14	Pulmonary valvotomy and right ventricular outflow tract patch
18	Aortic valve replacement with 15 mm Ionescu-Shiley valve
22	Atrial septal defect repaired
23	Aortic valve replacement with 15 mm Ionescu-Shiley valve

ventricular outflow tract gradient, the left ventricular angiogram demonstrated both a large extracardiac space and a change in angulation of the conduit with the left ventricle. At reoperation, it was found that the apical stent had partially detached from the left ventricle and had produced a large pseudoaneurysm cavity that occupied the lower third of the left hemithorax. Because the pseudoaneurysm of the apical anastomotic site was thought to be the result of endocarditis, the conduit and pseudoaneurysm cavity were removed. The conduit was transected about 6 cm from the aorta and oversewn and the left ventricle apex closed. The aortic anulus was then extensively enlarged and a 9A 1260 Starr-Edwards prosthesis inserted. The patient had an uneventful postoperative course and has remained asymptomatic for the past 2 years.

Patient 4 developed a new murmur of conduit valve (porcine) insufficiency 3 years after placement of the apico-aortic conduit. Over the next 6 months, he developed shortness of breath and angina. Because of increasing symptoms he underwent a second postoperative cardiac catheterization (Table 5). At reoperation, the conduit was cross clamped and divided and the porcine valve removed. Examination of the valve revealed complete degeneration of the leaflets (no calcification was noted). A new 20 mm porcine valve conduit was then sutured into position. The patient had an uneventful postoperative course and has remained asymptomatic for the past 3 months.

Patient 6 was asymptomatic for 3.9 years after placement of the apico-aortic conduit, when a new murmur of conduit (porcine) valve insufficiency was noted. Over the next 3 months she developed dyspnea on exertion and syncope. At a second postoperative catheterization performed at another institution severe conduit valve insufficiency and an 82 mm Hg left ventricular outflow tract gradient were noted. Left ventricular angiography at both postoperative catheterizations demonstrated that the apical prosthesis was improperly positioned with the orifice of the stent directed toward the interventricular septum. During systole, the apical portion of the left ventricle nearly obliterated the orifice of the apical stent. She subsequently underwent reoperation at the other institution. At reoperation, the apico-aortic conduit was ligated; an aortoventriculoplasty was performed (the aortic anulus and ventricular septum were incised and a 12 × 3.5 cm Cooley-Meadox Dacron patch was anchored to the ventricular septum and aorta) and a 21 mm Björk-Shiley prosthesis was inserted into the enlarged aortic valve anulus. The patient was reported to have had an uneventful postoperative course and was recently discharged from the hospital.

The remaining 18 patients are asymptomatic and are taking no cardiac medications or anticoagulants. They have a grade 2–3/6 systolic ejection murmur at the apex and a normal heart size on chest X-ray film with no calcifications of the porcine aortic valve or conduit. Only 1 of these 18 patients (Case 3) has developed a murmur of conduit valve insufficiency (3 years after repair). In all 18 patients, the rest electrocardiogram still demonstrates a pattern of left ventricular hypertrophy and strain.

None of the 21 surviving patients had received any anticoagulant therapy after placement of the apico-aortic conduit. There has been no clinical or laboratory evidence of peripheral emboli in any of the patients as documented by: physical examination and normal urine analysis in all; normal renal scan in nine and normal renal angiography in nine patients who underwent these examinations. None of the patients has any clinical or laboratory evidence of hemolysis.

Postoperative cardiac catheterization. This procedure was performed 0.7 to 1.5 years (mean 1.0) after placement of the apico-aortic conduit in 15 of the 21 surviving patients (Table 3). All 15 patients exhibited a decrease in peak left ventricular outflow tract gradient from 102.5 ± 20.5 mm Hg before operation to 14.8 ± 9.9 mm Hg after operation ($p < 0.001$) (Fig. 1). The site of residual left ventricular outflow tract obstruction was identified by catheter pullback in 11 of the 15 patients. Obstruction in these patients tended to be in three distinct areas: the proximal end of the conduit (junction of the apical stent with the left ventricle) in nine patients, the porcine aortic valve in five and the distal end of the conduit (junction of the conduit with the aorta) in one. More than one site of obstruction was present in five patients (Fig. 2).

In addition to a reduction in rest left ventricular outflow tract gradient, placement of the apico-aortic conduit resulted in a significant decrease in left ventricular end-diastolic pressure (from 13.5 ± 2.8 to 11.8 ± 7.8 mm Hg) ($p < 0.05$) and a small increase in cardiac index (from 3.59 ± 1.08 to 3.83 ± 1.17 liters/min per m^2) ($p < 0.05$).

The hemodynamic response to exercise was evaluated in 8 of the 15 patients, supine bicycle exercise in 6 patients and isometric handgrip exercise in 5 patients (3 patients had both types of exercise) (Tables 4 and 6). In seven of the eight patients, exercise caused only a small increase in left ventricular outflow tract obstruction despite an increase in cardiac index of 5 to 140% (Fig. 3). In Patient 7, supine exercise increased the left ventricular outflow tract gradient from 31 to 103 mm Hg. In this patient, as in Patient 6, left ventricular angiography demonstrated that the left ventricular apical prosthesis was improperly positioned with the

Table 4. Postoperative Hemodynamic Data in 15 Patients With Left Ventricular Apico-Aortic Conduit

Case	Conduit Stent and Valve Size (mm); Site	Preop Cath			Postoperative Catheterization																		
					Years Postop at Cath	Rest				Conduit Gradient (mm Hg)				Isometric Exercise				Supine Bicycle Exercise					Other Residua
		CI	LVP	ΔAS		CI	SI	VO_2	LVP	Total	Prox	Val	Dist	CI	SI	LVP	ΔAS	CI	SI	LVP	ΔAS	ΔCI/ΔVO_2	
1	18/20; Des Ao	—	230/23	120	1.0	3.2	42	135	135/17	15	15	0	0	4.9	50	173/17	20	—	—	—	—	—	1+ native AI
2	20/22, Des Ao	2 0	220/40	85	0.9	3 7	45	—	150/13	25	—	—	—	—	—	—	—	—	—	—	—	—	No residual coarct gradient EF = 69%
3	16/18; Abd Ao	3 2	205/18	108	1.5	4 0	46	142	133/13	21	15	6	0	4 2	46	156/16	25	—	—	—	—	—	1+ native AI
4	18/20, Abd Ao	3 4	210/17	120	1 0	4.2	50	116	119/16	27	13	9	5	—	—	—	—	—	—	—	—	—	1+ native AI
5	18/22, Des Ao	3.6	210/18	100	1 1	3 7	47	156	140/16	0	0	0	0	4.2	42	150/25	0	6.6	41	170/20	10	5 7	1+ native AI
6	16/18; Des Ao	5.0	242/16	130	1.2	5.3	53	142	130/14	20	15	5	0	—	—	—	—	—	—	—	—	—	1+ native AI
7	18/16, Des Ao	4 0	225/15	135	1 5	4 2	41	138	145/11	31	23	8	0	5 7	42	163/13	32	6 9	43	232/10	103	6.9	1+ native AI
8	16/18, As Ao	3.0	210/16	110	1 4	4 2	53	141	125/12	15	10	5	0	—	—	—	—	9.3	60	160/11	25	7.2	ΔRVOT = 10, 1+ native AI
9	18/20, As Ao	3 3	200/24	90	1 0	3.8	51	128	124/18	6	6	0	0	—	—	—	—	8.4	58	165/15	15	7 1	
10	16/18, Des Ao	4.2	195/18	105	1 0	4 6	50	121	128/15	7	7	0	0	—	—	—	—	7.9	50	158/15	10	6.5	
11	14/16, Des Ao	3.1	200/15	80	0 7	4.6	—	—	100/10	0	—	—	—	—	—	—	—	—	—	—	—	—	
12	18/20; Des Ao	5 5	200/20	80	0.5	4.1	—	—	125/8	15	—	—	—	—	—	—	—	—	—	—	—	—	1+ native AI
13	18/20, Des Ao	2.1	190/15	70	0.5	3.9	—	—	110/13	10	—	—	—	—	—	—	—	—	—	—	—	—	1+ native AI
14	14/16, Abd Ao	3 6	200/15	70	1 0	4 2	42	140	134/10	22	22	0	0	—	—	—	—	—	—	—	—	—	PSΔ = 20, PI, mod; 1+ native AI
15	18/20, Des Ao	2 5	210/17	90	1 0	3.3	42	118	135/12	0	0	0	0	3 9	44	138/15	0	7.9	53	188/18	25	7.1	1+ native AI

Cath = cardiac catheterization, CI = cardiac index (liters/min per m^2), Dist = distal, EF = ejection fraction, LVP = left ventricular pressure (mm Hg), prox = proximal, RVOT = right ventricular outflow tract, SI = stroke index (ml/min per m^2), Val = valve, VO_2 = indexed oxygen consumption (ml O_2/min per m^2), Δ = change in, ΔAS = change in aortic outflow tract gradient (mm Hg). Other abbreviations as in Table 1

Table 5. Hemodynamic Data in Three Patients With Left Ventricular Apico-Aortic Conduit Who Required Reoperation

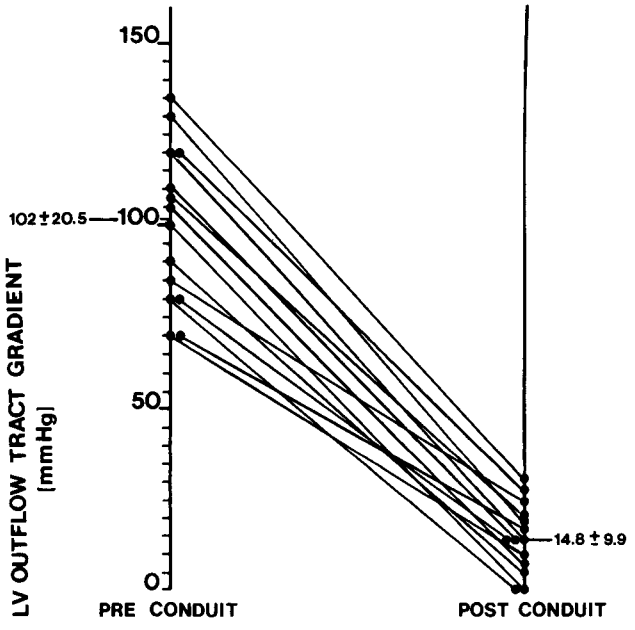
Case	Years Postop	CI	SI	VO_2	LVP	Conduit Gradient (mm Hg) Total	Prox	Val	Dist	Other Residua
2	0.9	3.7	45	—	150/13	25	—	—	—	No residual coarctation
	1.2	2.3	25	—	165/33	55	—	—	—	Pseudoaneurysm of LV apex
4	1.0	4.2	50	116	119/16	27	13	9	5	1+ native AI
		2.6	35	107	132/52	17	—	—	—	4+ conduit valve insufficiency
6	1.2	5.3	53	142	130/14	20	15	5	0	1+ native AI, no conduit AI
	*4.2	3.3	42	125	192/16	82	—	—	—	4+ conduit insufficiency, obliteration of apical portion of conduit during systole

*Catheterization performed at another institution
LV = left ventricular; other abbreviations and units as in Table 4.

orifice of the stent directed toward the interventricular septum and nearly 50% obstructed by left ventricular muscle (Fig. 4).

The exercise factor or relation between the change in oxygen consumption and cardiac index with supine exercise was normal in all six patients (6.7 ± 0.6 liters/min per ml oxygen). With bicycle or handgrip exercise, or both, stroke index increased or remained unchanged in seven of the eight patients. Patient 5, the only patient in whom exercise produced a decrease in stroke index, also experienced the largest exercise-induced increase in left ventricular end-diastolic pressure.

Figure 1. Change in peak systolic left ventricular (LV) outflow tract gradient after placement of the apico-aortic conduit. The preconduit gradient was obtained at the preoperative catheterization and the postconduit gradient at the postoperative catheterization.



Discussion

The long-term clinical follow-up of our 21 surviving patients with an apico-aortic conduit has been good. Eighteen of the 21 patients have experienced marked symptomatic improvement during the 0.5 to 5 years of follow-up; all are at present asymptomatic and receiving no medications or anticoagulant agents.

Complications of apico-aortic conduits. Endocarditis (1,2), thromboembolic events (2), pseudoaneurysm formation of the left ventricular apex graft anastomosis (11) and porcine valve deterioration (12–16) are the four well recognized complications of apico-aortic conduits. Four of our patients have developed one of these complications. In Patient 2, the partial disruption of the apical anastomosis with pseudoaneurysm formation was believed to be due to endocarditis. In the other three patients (Cases 3, 4 and 6), conduit valve insufficiency developed 3 to 4 years after placement of the apico-aortic conduit. In two of these patients (Cases 4 and 6), the porcine valve insufficiency was severe enough to require reoperation, although in the third patient (Case 3), it does not appear to be hemodynamically significant.

We have observed no thromboembolic events in any of our patients and none are receiving any anticoagulant agents. In contrast, Stansel et al. (2) observed clinical and patho-

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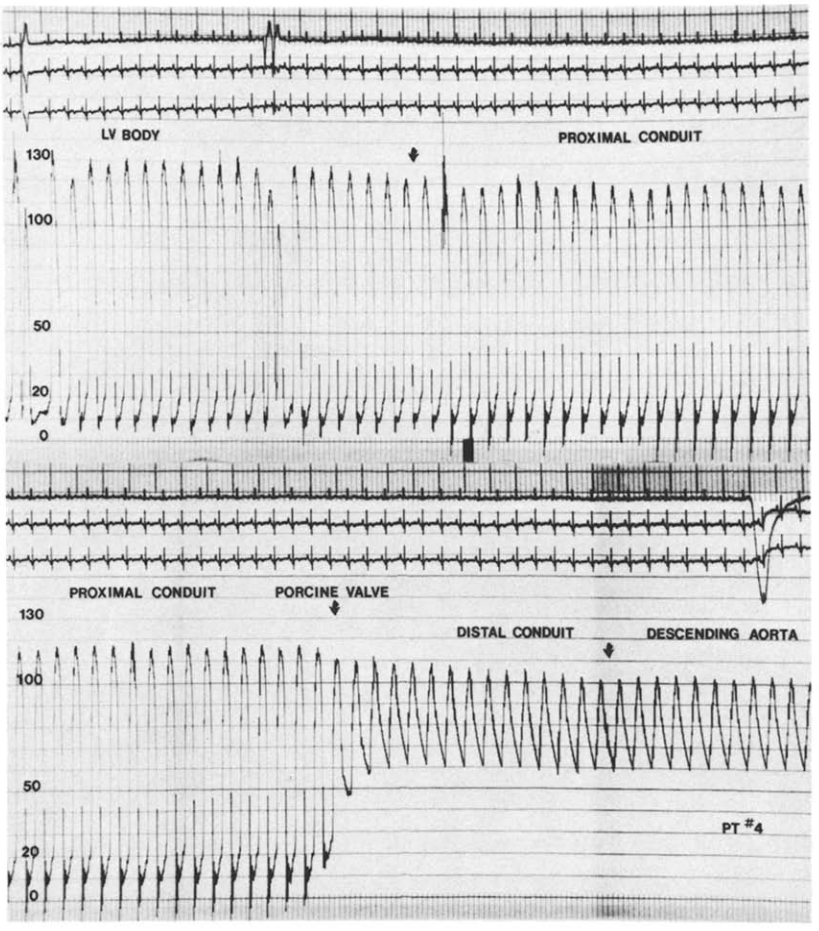


Figure 2. Case 4. Postoperative pullback pressure tracing demonstrating a 13 mm Hg gradient between the left ventricular (LV) body and the proximal conduit, a 9 mm Hg gradient at the porcine valve and a 5 mm Hg gradient between the distal conduit and descending aorta.

logical evidence of thromboemboli in three of four children with an apico-aortic conduit. The reasons for these two different rates of thromboembolism are unclear. However, because Stansel et al. (2) observed thrombus formation at the suture line between the apical stent and valved corduit, their high rate of embolic events may relate to technical difficulties in suturing the stent to the conduit or to the different operative tecniques used.

Hemodynamic results. All 21 of our long-term surviving patients have initially experienced excellent hemodynamic results. All have had significant decreases in rest left ventricular outflow tract gradient and left ventricular end-diastolic pressure and a small increase in rest cardiac index. One year postoperatively, rest left ventricular outflow tract gradients ranged from 0 to 32 mm Hg (mean 14.8 ± 9.9).

Table 6. Hemodynamic Response of Patients With Left Ventricular Apico-Aortic Conduit to Isometric Handgrip Exercise and Supine Bicycle Exercise

	Isometric (5 patients)	Bicycle (6 patients)
ΔHR (beats/min)	+15 ± 6.2	+71 ± 3.7
ΔCI (liters/min per m^2)	+1 ± 0.3	+3.7 ± 0.4
ΔLVEDP (mm Hg)	+3.5 ± 1.7	+0.2 ± 1.02
ΔLV-Ao gradient (mm Hg)	+2.5 ± 1.1	+20.8 ± 11.8

Values are mean ± standard error of the mean; ΔCI = change in cardiac index, ΔHR = change in heart rate; ΔLV-Ao = change in left ventricular to aortic systolic gradient; ΔLVEDP = change in left ventricular end-diastolic pressure

Conduit obstruction and valve insufficiency. The conduit obstruction was documented by catheter passage in 11 of 15 patients to occur at three sites: at the egress of the left ventricle, at the porcine valve and at the aortic to conduit junction, with obstruction proximally at the egress of the left ventricle the most common site (9 patients). Malorientation of the apical stent with respect to the long axis of the left ventricle in two patients was responsible for this proximal conduit obstruction (Fig. 4). Because only 1 of the 11 patients experienced any distal conduit obstruction (at the aortic anastomotic site), the location of the distal aortic-conduit anastomotic site does not seem to be critical. Finally, five patients exhibited only mild obstruction (5 to 10 mm Hg) at the level of the porcine valve.

Conduit (porcine) valve insufficiency subsequently developed in three patients. Recent reports by Geha (12), Saunders (13), Rocchini (14) and their co-workers and by others (15,16) have documented that children have a high

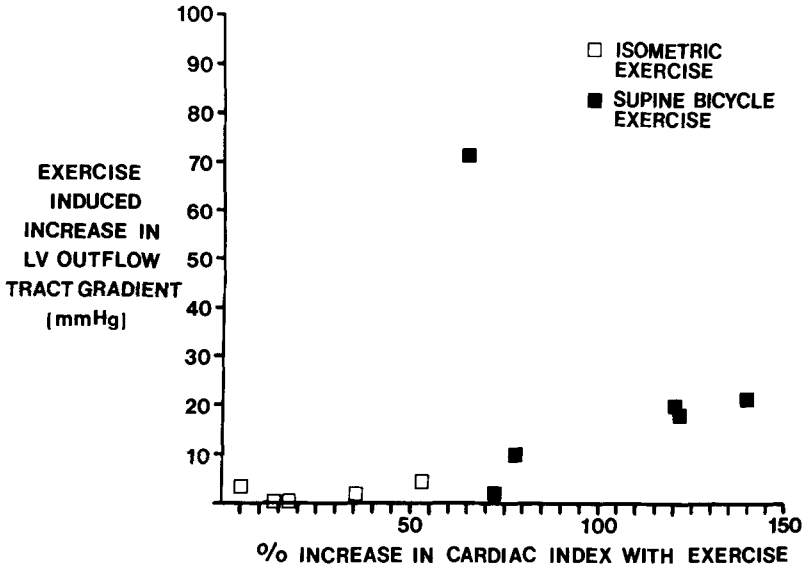


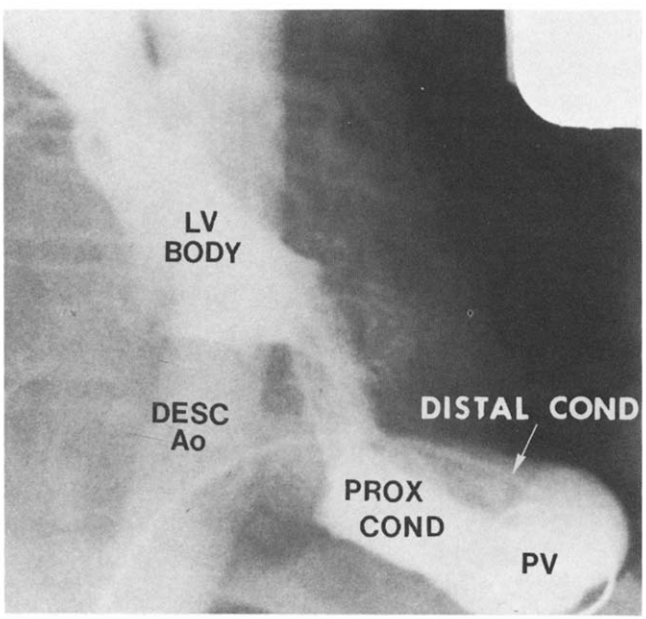
Figure 3. Relation between the exercise-induced increase in left ventricular (LV) outflow tract gradient and the percent increase in cardiac index with exercise. **Open squares** represent values obtained after isometric exercise and **closed squares** represent values obtained during supine bicycle exercise. In all but 1 of the 11 patients (Case 7), exercise produced only a small increase in outflow tract obstruction.

incidence of early porcine xenograft dysfunction. However, in contrast to these reports, the major cause of xenograft dysfunction in our patients with a left ventricular apico-aortic conduit was porcine valve insufficiency, not porcine valve calcification and stenosis.

Exercise testing to detect left ventricular outflow obstruction. Exercise testing at the time of postoperative cardiac catheterization was used in eight patients to evaluate the relation between increasing cardiac output and increasing degree of left ventricular outflow tract obstruction. Despite large increases in cardiac output, only one patient developed a significant increase in left ventricular outflow tract obstruction. In this patient (Case 7), supine bicycle exercise testing demonstrated that improper positioning of the orifice of the left ventricular stent resulted in a dynamic form of severe left ventricular outflow obstruction.

We have no data on which to base recommendations for optimal conduit and stent size, but our findings suggest that a 16 mm stent and 18 mm valved conduit are large enough to provide the adolescent patient adequate relief of left ventricular outflow tract obstruction, both at rest and with exercise.

Figure 4. Case 7. End-systolic frame from the postoperative left ventricular angiogram. There is malorientation of the proximal conduit (PROX COND) in relation to the body of the left ventricle (LV). DESC Ao = descending aorta; PV = porcine valve.



Conclusions and recommendations. Our clinical experience over 5 years with valved apico-aortic conduits for primary or recurrent left ventricular outflow tract obstruction has been very good. Our results have shown that the procedure is well tolerated, relieves signs and symptoms of left ventricular outflow tract obstruction and permits a normal hemodynamic response to exercise. At present, the major limitation of this procedure appears to be the incidence of porcine xenograft dysfunction. As with other operations for left ventricular outflow tract obstruction, the apico-aortic conduit is not a curative procedure. However, unlike other operations, such as an aortoventriculoplasty (Cono operation), the extracardiac location of the valved conduit makes it ideal for reoperation on a malfunctioning prosthetic valve and permits future surgical procedures on the native left ventricular outflow tract. Therefore, despite its limitations, we recommend the placement of an apico-aortic valved conduit as the procedure of choice for any symptomatic patient whose left ventricular outflow tract is not or has never been amenable to repair by conventional surgical methods, such as aortic valve replacement or resection of subaortic stenosis.

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