

PLEUROPERITONEAL SHUNTS AS TREATMENT FOR REFRACTORY CHYLOTHORAX FOLLOWING SURGERY FOR CONGENITAL HEART DISEASE

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Persistent chylothorax is a recognized complication of surgery for congenital heart disease (CHD), and may occur secondary to trauma to the thoracic duct, elevated central venous pressure or caval obstruction. Medical therapy (tube thoracostomy, low fat diet) has a high failure rate (>75%) and may be accompanied by respiratory compromise, lymphopenia and protein deficiency. Since 1983, we have utilized pleuroperitoneal shunts as surgical treatment for refractory chylothorax. Twelve children, aged 2 weeks-10 years, had persistent chylothorax following surgery for CHD (Senning=4 pts, Mustard=1 pt, Fontan=2 pts, closure VSD=1 pt, repair AV canal=1 pt, repair ToF=1 pt, central shunt=1 pt, subclavian flap angioplasty=1 pt). All patients were treated with medical therapy (chest tube, low-fat diet or intravenous alimentation) for 10-64 days (mean, 29 days). Chylothorax resolved with medical therapy in 2 patients, and 10 patients underwent placement of a pleuroperitoneal shunt with resolution of effusion in 9/10 patients (90%). The only patient whose effusion failed to resolve expired 1 week after shunt insertion with low cardiac output. The 9 surviving patients were discharged from the hospital 6-49 days (mean, 20 days) following shunt placement. Complete resolution of the chylothorax occurred in all 9 patients, and the shunt was subsequently removed in 2.5-10 months (mean, 6.5 months).

When aggressive medical therapy of chylothorax following surgery for CHD fails, early placement of a pleuroperitoneal shunt offers an excellent alternative to thoracic duct ligation or pleurodesis, and allows for immediate improvement in respiratory symptoms, and nutritional and immunologic state.

BLADE AND BALLOON ATRIAL SEPTOSTOMY: RESULTS AND FOLLOW-UP IN 131 PATIENTS

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Blade and balloon atrial septostomy (BBAS) is a useful technique for acute palliation of patients (pts) with certain types of congenital heart disease. The purpose of this study was to assess the long term adequacy of the atrial septal defect (ASD) created by BBAS. We performed 136 BBAS procedures in 131 patients from 1978-1990. Ages ranged from 1 day to 30 yrs (median = 5 mo.) and weights from 2.5 to 66.6 kg (median = 5.4 kg). Primary diagnoses included transposition of the great arteries (TGA, n=45), tricuspid and/or pulmonary atresia (TPA, n=21), left AV valve stenosis or atresia (LAV, n=34), total anomalous pulmonary venous connection (TAPVC, n=24), and pulmonary vascular obstructive disease (PVOD, n=12). Transatrial gradient (TAG) improved from 6.6 ± 7.0 mm Hg to 1.5 ± 2.5 mm Hg ($p < 0.001$) with BBAS. Arterial oxygen saturations improved from $65.3 \pm 14.1\%$ to $75.5 \pm 8.2\%$ ($p < 0.001$) in pts with TGA and decreased from $89 \pm 8.8\%$ to $78.3 \pm 9.5\%$ ($p < 0.01$) in pts with PVOD. Repeat diagnostic cath was performed on 50 pts 34.8 ± 25.2 mo after BBAS, with no significant change in the TAG (1.6 ± 2.7 mm Hg, $p = NS$) or in arterial saturations (77% vs 78% , $p = NS$) when compared with the immediate post-BBAS data. Five pts required repeat BBAS because of persistent restriction of the ASD. Four of the five had LAV, 1 had TGA. Seven pts had Blalock-Hanlon atrial septectomy performed after BBAS, but only 1 since 1985; 5/7 had LAV. Complications included stroke in 4 pts and death in 3. Difficulty in retracting the blade ($n=3$) was resolved by pulling the partially closed blade into the sheath. We conclude that BBAS is an effective method for establishing an adequate ASD, facilitating long term survival in the absence of or until the time of corrective surgery. Pts with left AV valve obstruction required repeat procedures more often than the other groups.

AUGMENTED ATRIAL NATRIURETIC FACTOR RESPONSE TO EXERCISE AFTER COARCTATION REPAIR

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Since resting or exercise hypertension may occur following repair of coarctation of the aorta, the response of atrial natriuretic factor (ANF) to exercise after coarctectomy was evaluated. Thirty-one pts aged 8 to 40 (median 16.8) yrs underwent maximal bicycle stress tests and were compared to 16 age matched normal controls. Pts were studied 4-25 (median 10.4) years after repair and were asymptomatic. ANF samples were drawn pre, at peak, and 15 min post exercise. 22% of pts were hypertensive at rest and 29% at the end of exercise. Pts had a significant increase in ANF from pre to peak exercise (48 ± 30 to 62 ± 48 pg/ml, $p < 0.01$) and these levels remained elevated at 15 min post exercise, while controls showed no significant change in ANF from baseline through recovery. At 15 min of recovery from exercise, pts' ANF (65.5 ± 48 pg/ml) were significantly greater than controls (36.6 ± 19.6 pg/ml) by Wilcoxon rank sum test, ($p < 0.05$). Multiple regression analysis indicated that in pts reaching maximal exercise, peak exercise ANF was closely predicted by baseline systolic blood pressure, peak diastolic blood pressure, and peak heart rate, (multiple $r=0.85$, $r^2=0.72$, $p < 0.001$).

Conclusions: Post coarctectomy pts have an augmented ANF output with exercise compared to normal controls. This is related mainly to resting systolic pressure, peak exercise diastolic pressure and heart rate and may reflect atrial hemodynamic changes associated with rest or exercise induced hypertension.

RISK OF LATE ENDOCARDITIS FOLLOWING CONGENITAL HEART REPAIR: 30 YEAR FOLLOW-UP IN 610 PATIENTS

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We report the long-term risk of infective endocarditis (IE) in 610 patients who survived at least 3 months following surgical repair of congenital heart disease, during the years 1955-1960. The median follow-up was 27.7 years, range <1-33 years. All patients were followed for 30 years unless death or endocarditis occurred first.

	N	Patient		Cases/100,000		95% C.I.
		Years	Cases	Patient yrs		
ASD	119	2,819	1	36	0.9-198	
VSD	145	3,723	2	54	6.5-194	
PS	183	4,080	0	0	0- 90	
TET	163	4,315	0	0	0- 86	
ALL	610	14,937	3	20	0.04- 59	
Olmssted	-	-	-	4	3.7 - 6.1	

[atrial septal defect (ASD), ventricular septal defect (VSD), pulmonary stenosis (PS) and tetralogy of Fallot (TET)]. Compared to the reported rate of IE, [4/100,000 patient years in an unselected population (Olmssted County)], the relative rate of IE was 5 times higher overall in postoperative CHD patients and 13 times higher in patients status post VSD repair.

Univariate and multivariate analysis demonstrated no statistical variables predictive of an increased risk of late endocarditis.

Conclusion: Late endocarditis following successful repair of congenital heart defects is uncommon but occurs more frequently than in a control population.