

Results: Indications for PVR were valvular regurgitation (n=6), obstruction (n=3), and both (n=1). Valve types were St. Jude (n=7), Starr-Edwards (n=2), and Bjork-Shiley (n=1). In 5 pts, the mechanical PVR was placed within a conduit. Concomitant mechanical tricuspid valve replacement (St. Jude) was performed in 6 pts. Postoperatively, all but 1 were anticoagulated with warfarin. During late follow-up (8.3 +/- 7.7 yrs, max 25 yrs), 1 pt required repeat PVR for outgrowth of prosthesis 25 yrs postoperatively, and 1 pt with normal prosthetic pulmonary valve function underwent heart transplantation for severe biventricular failure 3.5 yrs postoperatively. The average mean gradient across the mechanical PVR by echocardiography in the remaining 8 pts was 11 +/- 7 mmHg. There were no perivalvular leaks or vegetations, and no evidence of pannus formation or prosthesis dysfunction. There were 3 late minor bleeding events (epistaxis in 2 pts and menorrhagia in 1). There was 1 sudden late death 15 yrs after PVR, and 1 death from unknown causes in the aforementioned pt with outgrowth of the mechanical PVR, 5.5 yrs post re-PVR with a bioprosthesis. There were no cases of pulmonary emboli or PVR thrombosis.

Conclusions: Mechanical PVR appears to provide excellent durability and hemodynamic results without mechanical valve failure or valve thrombosis in this small series. Mechanical prostheses should be considered for PVR in selected pts, particularly those who require chronic warfarin anticoagulation for other reasons.

11:45 a.m.

**840-6 Coarctation Long-Term Assessment (COALA-Study) Incidence of Restenosis and Hypertension After Surgical Repair**

Alfred Hager, Simone Kanz, Harald Kaemmerer, Christian Schreiber, John Hess, Deutsches Herzzentrum München, TUM, München, Germany

Background: to assess the incidence of restenosis and arterial hypertension in patients after surgical repair of aortic coarctation in a cross sectional study.

Patients and Methods: From 1974 to now 405 patients born before 1.1.1965 underwent surgery for isolated aortic coarctation in our hospital. From those 383 who are still alive 26 moved to remote or unknown areas and 83 denied a follow up examination at our institution. The study group of the remaining 274 patients (16 - 73 years old, 90 female, 184 male, 72% of those that are still alive), 18.4 ± 5.9 years (range 1 - 27 years) after surgery, underwent a structured clinical investigation with a Doppler sonographic measurement of the blood pressure at all limbs, exercise test, and ambulatory blood pressure measurement.

Results: 29 patients (11%) already underwent surgery for restenosis, another 20 patients (7%) had a leg-arm gradient of more than 20 mmHg suggesting restenosis now. Risk factors were young age at the first repair and first repair without end-to-end anastomosis.

67 Patients (24%) were already on antihypertensive drug therapy, another 48 patients (18%) had a mean systolic blood pressure of 134 mmHg or higher in ambulatory blood pressure measurement and should therefore also be classified hypertensive. Furthermore, another 28 patients (10%) showed a blood pressure during exercise exceeding 2 SD of reference values. All in all, only 131 patients (48%) had a normal blood pressure reaction. The only independent risk factors for an abnormal blood pressure was a repair without end-to-end anastomosis. Other risk factors like age at repair, age at investigation, and body weight at investigation were not significant in a multiple regression analysis anymore.

Conclusions: In the long-term follow up 18% of patients after surgery for aortic coarctation show restenosis. An arterial hypertensive blood pressure reaction is much more common in long term follow up with an incidence of 52%. In only few patients hypertension is due to restenosis. Best results were achieved in patients in whom an end-to-end anastomosis could be performed and in whom repair was not performed at a very young age.

POSTER SESSION

**1152 Heart Failure, Exercise, and Risk Factors in Congenital Heart Disease**

Tuesday, March 09, 2004, Noon-2:00 p.m.  
 Morial Convention Center, Hall G  
 Presentation Hour: 1:00 p.m.-2:00 p.m.

**1152-199 Thromboembolic Events Among Children With Cardiomyopathy: Results From the National Australian Childhood Cardiomyopathy Study**

Robert G. Weintraub, Patty Chondros, Alan Nugent, John Carlin, Piers E.F Daubeney, National Australian Childhood Cardiomyopathy Study, Melbourne, Australia

Background: Subjects with cardiomyopathy (CM) are at risk from thromboembolism. There is little information about predisposing factors and the magnitude of the risk in children.

Methods: The National Australian Childhood Cardiomyopathy Study is a population-based study, including all children in Australia with primary CM who presented at 0-10 years of age. Cases were classified according to WHO criteria. A thromboembolic event was defined as the finding of intracardiac thrombus, or onset of organ dysfunction related to an embolic event. Risk factors examined included CM type, congestive heart failure (CHF) at diagnosis, duration of inotropic therapy, mechanical ventilation, ICU and hospital stay at presentation. Children not surviving 24 hours from presentation were excluded

from survival analysis. Study end-points were death or transplantation.

Results: There were 314 patients diagnosed with CM during the study period, of which 299 were included in survival analysis. Of these, 19 (6.4%) of these developed thromboembolic complications, including 14/172 (8.1%) with dilated CM, 3/39 (7.7%) with unclassified CM, 1/12 (8.5%) with restrictive CM and 1/80 (1.2%) with hypertrophic CM (p=0.02 compared to other cases). 6 of 19 (31.6%) of thromboembolic events occurred <2 days of presentation and 14 (73.7%) occurred <30 days. CHF at presentation was related to risk of thromboembolism (hazard ratio 5.0; 95% CI: 1.6-21.8). The one and 5-year survival free from thromboembolic events were 93% (95% CI: 89-96%) and 91% (95% CI 84-95%), respectively, for children with dilated CM, and 92% (95% CI: 76-97%) at both time points for children with unclassified CM. Embolic phenomena included a cerebrovascular event in 5 cases and a pulmonary embolus in 1. The risk of death or transplantation was significantly increased for all study patients with thromboembolism (hazard ratio 4.2; 95%CI: 1.6-11.2) as well as for those with dilated CM alone (hazard ratio 4.6; 95% CI: 1.45-14.6).

Conclusions: Thromboembolism occurs in up to 9% of children with CM within one year of diagnosis and is related to early CHF and CM type. Thromboembolic complications occur early and are associated with an increased risk of death or transplantation.

**1152-200 N-Terminal Pro-B-Type Natriuretic Peptide Differentiates Lung From Heart Disease in Infants With Respiratory Distress**

Shlomo Cohen, Chaim Springer, Zeev Perles, Azaria JJT Rein, Avraham Avital, Zvi Argaman, Amiram Nir, Hadassah University Hospital, Jerusalem, Israel, Shaarei Zedek Medical Center, Jerusalem, Israel

Background: Respiratory distress (RD) is a common symptom in infants. RD is usually caused by lung disease, but can also be a result heart disease. It is often difficult to determine the cause of RD. N-terminal pro-B-type natriuretic peptide (N-BNP) is co-secreted along with BNP from cardiac myocytes. Like BNP, N-BNP is a marker for cardiac volume and pressure overload.

Aim: To determine whether N-BNP levels can differentiate between lung disease and heart disease in infant with RD.

Methods: Infants (age 1-36m) who presented at Hadassah University Hospital with RD underwent physical examination, chest X-ray, arterial blood gases and echocardiogram. Control N-BNP values were obtained from age-matched babies with no acute illness and no history or signs for heart or lung disease. Plasma N-BNP levels were measured by Electrochemoluminescence immunoassay (Roche, Germany).

Results: There were 18 infants with lung disease (laryngitis, pneumonia and RSV bronchiolitis), 19 infants with heart disease (myocarditis, dilated cardiomyopathy, atrio-ventricular canal, hypertrophic cardiomyopathy and ventricular septal defect) and 16 healthy infants. Infants with respiratory distress due to heart disease had significantly higher plasma N-BNP than infants with respiratory distress due to lung disease or control (table).

\* p<0.001 vs lung and vs control; # p<0.05 vs control.

Conclusions: Plasma N-BNP can differentiate infants with RD due to heart disease from infant with RD due to lung disease.

Group	Number	Age-months mean(±sd)	Respiratory rate mean(±sd)	O <sub>2</sub> saturation mean(±sd)	N-BNP (pg/ml) median
Control	16	14.3(5.4)	36(7.8)	97(1.4)	164
Lung	18	9.2(5.9)	62(16.1)#	89.4(3.7)#	311
Heart	19	11.9(12.3)	68(15.7)#	88.6(4.7)#	15403*

**1152-201 Brain Natriuretic Peptide Correlates With Myocardial Performance Index in Congenital Heart Disease Patients**

Alice A. Perlowski, John S. Child, Robert S. Ross, Pamela D. Miner, David Geffen School of Medicine-UCLA Medical Center, Los Angeles, CA

Background: The myocardial performance index (MPI) is an echocardiographic Doppler-derived measure of ventricular function that has been previously validated in congenital heart disease(CHD) patients. It may be preferred over conventional non-invasive measures of ventricular function in patients with complex anatomy, since it is neither dependent on geometric shape nor heart rate. Although Brain Natriuretic Peptide (BNP) is well described as a predictor of systolic and diastolic dysfunction in anatomically correct hearts, it is unclear how it relates to MPI in those with CHD.

Methods: We prospectively evaluated 54 adult patients with a broad range of both cyanotic and noncyanotic heart disease. Included were both surgically repaired and unrepaired pts. Levels of BNP were measured in all subjects using standardized assays. Doppler echocardiography was performed on the study subjects within six months of the BNP assay. No subject had a change in functional clinical status during this interval. Echo images were evaluated by an experienced observer blinded to BNP results and clinical status. The MPI was calculated as the interval from AV valve closure to opening minus ventricular ejection time, divided by ventricular ejection time. EF was calculated with standard methodology by the same observer.

Results: Of a total of 54 patients with measurable left ventricular (LV) or univentricular (UV) EFs, 34 had adequate data to calculate LV or UV MPIs. Of 30 pts with measurable right ventricular (RV) EFs, 23 had adequate data to calculate RV MPIs. BNP was found to be significantly correlated with LV/UV MPI (r= 0.461 with p= 0.006)and RV MPI (r= 0.748 with p < 0.0001) while LV/UV EF and RV EF had no significant correlation with BNP (r= - 0.189, p= 0.172; r= -0.066, p= 0.729, respectively) using a Pearson's correlation coefficient test.

Conclusion: In patients with CHD, BNP correlates significantly with MPI but not with left, right, or univentricular EF. This appears to be particularly true in the case of geometrically