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with a longer LOS (r=0.72, P=0.0017), C-section vs. vaginal delivery (P≈0.0007), and positive Group B Strep cervical culture (P≈0.036).

Conclusions: The perinatal period is associated with a higher percentage of Pts with cTnT elevations than other ages in pediatrics. Although clinically occult, very high levels of markers of myocardial injury can occur. The relationship with Apgar scores suggests ischemic injury, and relationships with prematurity and increased hospitalization are plausible. Racial differences are consistnt with cardiomyopathy data in infancy. Further investigation of factors associated with neonatal myocardial injury may lead to reduction of cardiomyopathy in later life.

1094-159

# Echocardiographic Evaluation of Diastolic Function **During Device Closure of Atrial Septal Defects: Effect of** Acute Changes in Load

Carlen A. Gomez, Achi Ludomirsky, Gregory Ensing, Albert P. Rocchini, University of Michigan, Ann Arbor, MI

Color M-mode flow propagation velocity (Vp) and tissue Doppler annular velocity (Ea) are indices of left ventricular (LV) relaxation that have been shown to be relatively load independent. The ratio of the load dependent mitral peak E velocity (E) to Vp and Ea has correlated with LV filling pressure in adults. Device closure of an atrial septal defect (ASD) may uniquely change LV loading conditions without affecting intrinsic diastolic function. The purpose of this study was to more clearly characterize load dependent and independent echocardiographic indicators of diastolic function by studying patients before and immediately after ASD closure.

Methods: Diastolic function indices were measured by transesophageal echocardiogram in 20 patients immediately before and after ASD device closure, including mitral inflow Doppler, pulmonary venous Doppler, mitral annular tissue Doppler, and color M-mode flow propagation. Measurements were compared to changes in shunt calculation and filling pressure.

Results: The study group consisted of 20 patients with a secundum ASD, with an age range of 1.5 years to 56 years (median 6 years). The average Qp:Qs was 2.3:1. After ASD closure, there was an increase in mean LV filling pressure from 9.1 to 10.5 mmHg (p<0.01). Indices of diastolic relaxation demonstrated normal baseline mitral E/A, tissue Doppler Ea/Aa, with a mildly low mean Vp (44.2 cm/sec). There was no significant change in E/A, Ea/Aa, or Vp after ASD closure. However, there was a significant increase in peak E velocity (p<0.03) after ASD closure. The patients with the largest shunts pre-closure (Qp/Qs>2; N=8) had the greatest increase in peak E, with a significant increase in E/Vp after ASD closure (p<0.03), though there was minimal increase in LV filling pressure.

Conclusions: The LV relaxation indices Ea and Vp appear to be load independent, and are unaffected by changes in LV filling hemodynamics following ASD closure. Peak E velocity and E/Vp are more load dependent, and increase with the acute change in LV filling volume after closure of large atrial shunts, even when increase in LV filling pressure is small.

1094-160

## Use of Pacemakers and Implantable Cardioverter Defibrillators in Children With Hypertrophic Cardiomyopathy

Bryan C. Cannon, Randall M. Bryant, Arnold L. Fenrich, Naomi J. Kertesz, Jeffrey A. Towbin, Richard A. Friedman, Baylor College of Medicine/Texas Children's Hospital, Houston, TX, University of Florida Health Science Center, Gainesville, FL

Background: Children with hypertrophic cardiomyopathy (HCM) have varied clinical courses, and there is no consensus concerning the use of pacemakers (PM) and implantable cardioverter-defibrillators (ICD). The purpose of this study was to evaluate the use of PM's and ICD's in children with HCM. Methods: All patients (pts) with HCM undergoing PM or ICD implant at 2 institutions from 1/90 to 12/01 were retrospectively evaluated and divided into: GROUP 1 - pts who received a device as a primary therapy to reduce left ventricular outflow tract obstruction (LVOTO) and GROUP 2-pts who received a device for another indication. Data evaluated: LVOTO by Doppler echo before and after implant, indication for device, symptoms, complications and outcome. Results: GROUP 1: 14 pts, median age at diagnosis: 8.5 yrs (0.9-14.7); at device implant: 12.8 yrs (9.3-23.9). All were DDD paced (mean AV delay 81 ± 21 msec). Mean LVOTO peak velocity before device:  $5.8 \pm 0.8$  m/sec, decreasing to  $4.2 \pm 1.2$  one day (p <0.05),  $3.6 \pm 1.2$  two months and 3.3 ± 1.4 one year after implant. In 6 pts with long-term follow-up (at least 4 yrs), mean was 2.8 ± 0.8. All had symptoms of decreased exercise tolerance, dizziness, or chest pain prior to device which resolved after implant in 7/14 (50%) patients. Sudden death occurred 8 and 11 months after implant in 2 pts with a PM; neither patient had a primary indication for ICD placement. GROUP 2: 11 pts, median age at diagnosis: 12.6 yrs (1.5-16.9); at device implant: 14.0 yrs (1.9-17.5). Indications for ICD: resuscitated sudden death (3), and syncope (3). Indications for PM: AV node ablation for intractable atrial arrhythmias (2), symptomatic sinus bradycardia (2), and AV block after radiofrequency ablation (1). In ICD pts, there were 4 inappropriate and 2 appropriate shocks (mean follow up 2.3 yrs). Conclusions: In this cohort of children with HCM, pacing reduced LVOTO peak velocities with symptomatic improvement in half. Devices may be required with atrial arrhythmias, resuscitated sudden death, syncope or sinus bradycardia. ICD's may be the most appropriate first choice for pacing because of the potential therapy for ventricular arrhythmias. This therapy can be instituted with a low incidence of complications.

### Mitral Valve Z-Score Predicts Need to Close Atrial **Shunts at Time of Coarctation Repair**

1094-161

Valerie A. Schroeder Linda Cripe, Jeffery M. Pearl, Peter B. Manning, Robert H. Beekman, III, William Douglas, David P. Nelson, University of Kentucky Medical Center, Lexington, KY, Cincinnati Children's Hospital Medical Center, Cincinnati, OH

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Background: Residual atrial communications (ASD/PFO) following infant coarctation (CoA) repair can lead to significant left to right shunt and congestive heart failure. We performed a retrospective study to evaluate risk factors predicting the need for ASD closure early after infant CoA repair.

Methods: We identified all infants < 3mo undergoing CoA repair (1/97-9/02) who also had a coexisting atrial shunt. Those with moderate or large VSD's were excluded. Echocardiographic variables included, indexed mitral (MV) and aortic (AoV) valve dimensions, left ventricular (LV) volume and relative LV length. Hemodynamic data was derived from catheterization when available. Results: 44 infants were identified (1-83d, 1.6-5.1kg). Group I consisted of 4 infants (9%) who developed severe CHF due to large atrial shunts (Qp/Qs >3 in all 4) and required ASD closure 23-44d post-CoA repair. Group II included the remaining 40 infants (no ASD closure). Both groups showed similar AoV sizes (t-test, Table 1), LV volumes and relative lengths, however, MV Z-scores and areas were significantly smaller in Group I infants. Using logistic regression analysis, the only parameters that significantly correlated with the need for ASD closure were MV Z-scores and area (p=0.01). All patients survived to ICU discharge. Conclusion: The need for ASD closure following CoA repair is rare, however, small mitral valve size may augment atrial shunting and promote CHF. Patients with small MV (Z<-2) should undergo ASD closure during CoA repair.

### Table 1

|                          | Group I | Group II | p-value |
|--------------------------|---------|----------|---------|
| Mitral Z-score           | -5.0    | -1.4     | .019    |
| Mitral valve area cm2/m2 | 2.1     | 4.1      | .0015   |
| Aortic Z-score           | -3.1    | -2.4     | NS      |
| Relative LV length       | 0.92    | 0.91     | NS      |
| LV volume cc/m2          | 30.3    | 34       | NS      |
|                          |         |          |         |

#### 1094-162 Risk Factors for Pulmonary Vein Stenosis

Donna A. Goff, Kimberlee Gauvreau, Steven J. Roth, Pedro J. del Nido, Kathy J. Jenkins, Children's Hospital, Boston, MA

Background: Progressive PVS is due to neoproliferation, but whether this begins as a response to injury or to patient predisposition is currently unknown. A case-control design was used to investigate potential risk factors for progressive PVS.

Methods: We selected all cases (1989-1999) with progressive intraluminal obstruction of >=2 pulmonary veins and surgery with >1 yr follow-up and a random sample of controls without PVS. Records were reviewed to identify demographic, neonatal, anatomic, operative and post-operative variables. Comparisons were made using Fisher's exact test. Because initial findings showed heterotaxy syndrome and/or total anomalous pulmonary venous return (TAPVR) to be highly associated with PVS, a second random control group frequency matched by heterotaxy and TAPVR was selected to further explore the effect of other variables

Results: Twenty-seven patients who met the case definition and 27 controls were identified from surgical databases. Heterotaxy syndrome was present in 9 cases (33%) and TAPVR in 14 (52%) but neither diagnosis was present in any control (p=0.002 and <0.001). Anatomic features associated with heterotaxy such as double outlet right ventricle (DORV), complete common atrioventricular canal and abnormal atrial situs were also more common among cases. In contrast, construncal defects other than DORV were less common among cases than controls (11% vs 41%, p=.03). Mothers of cases were younger (median 22.5 vs 29 yrs, p=.05), and case infants were more likely to have surgery within the first 3 months of life (81% vs 33%, p=.001) with a corresponding younger age (p<.001) and lower weight (p=.002), but control infants were exposed to longer cross clamp times (median 40 vs 57 min, p=.008). In analyses controlling for heterotaxy and TAPVR, the proportion of infants undergoing surgery in the first 3 months of life was no longer different, but control infants were still exposed to longer maximum and total cross clamp (p=.02 & p=.05) and bypass times (p=.04).

Conclusion: Progressive post-operative PVS is associated with specific anatomical defects, suggesting possible genetic mechanisms. Surgical variables were not associated with development of progressive PVS.