●121 ISCHEMIA IN THE DEVELOPING RABBIT MYOCARDIUM. Stanton B. Perry and Joanne S. Ingwall. Spon. by James E. Lock. Harvard Med School NMR Lab, Boston MA.

The relationship between maturation and response of the myocardium to ischemic injury remains controversial. Therefore, we studied isovolumic, buffer-perfused hearts(n=3-10/group) from 3 age groups of New Zealand white rabbits: 3 day(3d), 18 day (18d), and adult. Experiments (control (CON), 30 min no-flow ischemia(ISCH) and 30 min reflow(REF)) were performed at 37° C with performance measured as rate-pressure product (RPP). P31-NMR yielded ATP, creatine phosphate(CP), and inorganic phosphate(Pi) contents (μmol/gdw) and intracellular pH(pHi) at 4-6 min intervals. Na23-NMR with shift reagent (Dy(TTHA)3-) yielded intra-(Nai) and extracellular(Nao) sodium at 4 min intervals in 3d and 18d hearts.

| | | CON | END ISCH | END REF-%control | | During ISCH the rate of change in ATP, CP,Pi and pHi did not differ among groups. [ATP] differed among groups only at END-REF and was lower in adults (41 vs 65%CON for 3d and 18d). [CP] increased 4-fold with development. Recovery of CP and Pi (%CON) was lower in adult than 18d hearts. pHi was ≈7.1 during CON and REF and ≈6 at END-ISCH in each group. Nai reversibly increased ≈ 2.5-fold during ISCH for both 3d and 18d hearts. Nao increased 120% in 18d vs 103% in 3d during REF. Recovery of RPP was best in 18d hearts and worst adult hearts. |
|---|---|---|---|---|---|---|
| 3d | CP | 8.3 | 0.3 | 7.6 | (91) | |
| | Pi | 11.6 | 52.2 | 11.4 | (98) | |
| | RPP | 15200 | 0 | 9700 | (70) | |
| 18d | CP | 23 | 0.4 | 23.9 | (104) | |
| | Pi | 8.4 | 63.6 | 10.8 | (128) | |
| | RPP | 20700 | 0 | 18200 | (91) | |
| Adult | CP | 32.8 | 0.2 | 27.6 | (84) | |
| | Pi | 5.4 | 63.3 | 8.7 | (161) | |
| | RPP | 13400 | 0 | 8000 | (63) | |

Thus, tolerance to ischemia in the rabbit myocardium initially increases and then decreases during development. However, the results do not demonstrate a simple relationship between any metabolite or ion measured and developmental differences in functional recovery.

†122 TRANSMISSION OF AORTIC ACCELERATION FORCES TO INTRACRANIAL ARTERIES IN NEWBORN INFANTS: A PULSED DOPPLER (PD) STUDY. Tonse Raju, Shin Kim, Vivek Ghai. (Spon.by D.Vidyasagar) Univerisity of Illinois Medical Center, Department of Pediatrics, Chicago, Illinois.

Recent work suggests absence of elastic elements and lack of reticular fibers in major intracranial (IC) arteries of premature infants, (J. Neurosurg.'85 Hegedus). To study the hemodynamic counterpart of this structural immaturity, we measured the fractional transmission of aortic acceleration (ACC) forces to the IC arteries. Using standard cardiac Doppler techniques and a real time PD device (HP 770200) we measured cardiac out put (CO), maximum velocity (V max), ACC (dVmax/dt), and other time intervals in the aorta & IC vessels. Scanning via the temporal bone, (Transcranial Doppler. Raju JUM.'86) we studied the middle cerebral artery, (MCA) and via the fontanelle, the basilar and anterior cerebral arteires (ACA) in real time with flow/Doppler angle of <5°. Results in 4 term and 4 stable preterms (Wt. 1.2 Kg, GA 3.3 wks) were: 1) Higher aortic V max and ACC in term Vs preterm. 2) But a significantly larger % of aortic ACC transmission to all IC arteries in preterms as compared to the term. We conclude that transmission of high acceleration forces to preterm infants' brain vessels lacking elastic elements could render them vulnerable for intracranial hemorrhage. (*Preterm Vs term. p<0.05, ANOVA and Tukey's test)

| | Vmax(cm/s) Term | Preterm | Sys.ACC(cm/s2) Term | Preterm | % Aortic ACC Term | Preterm |
|---|---|---|---|---|---|---|
| Aorta | 109 | 52.3* | 1970 | 1009* | | |
| Basilar | 41.1 | 37.4 | 560 | 631* | 28.4% | 62.5% * |
| MCA | 40 | 30 | 562 | 500* | 28.5% | 50%* |
| ACA | 20.4 | 22 | 353 | 279* | 18% | 27.6%* |

123 BALLOON PULMONARY VALVULOPLASTY FOR COMPLEX CYANOTIC HEART DEFECTS. P. Syamasundar Rao, King Faisal Spec. Hospital, Dept. of Pediatrics, Riyadh, Saudi Arabia.

Six infants with complex cyanotic heart defects (CCHD) (tetralogy of Fallot (TOF) in two, transposition of the great arteries with ventricular septal defect (VSD) and pulmonary stenosis (PS) in two, dextrocardia, ventricular inversion, VSD and PS in the fifth, and critical PS with hypoplastic right ventricle in the sixth), aged 7 days to six months weighing 2.9 to 7.9 kg, underwent percutaneous balloon pulmonary valvuloplasty (PBPV) as a palliative procedure. The indication for PBPV was complex cardiac defect not amenable to total correction at the age and size at presentation but at the same time requiring palliation of pulmonary oligemia. The pulmonary blood flow index (1.83 ± 0.55 vs. 3.07 ± 0.65 l/min/m$^2$, $p < 0.05$), QP:QS (0.55 ± 0.05 vs. 1.0 ± 0.28, $p < 0.02$), and pulmonary artery pressure (21 ± 6 vs. 38 ± 12 mmHg, $p < 0.5$) increased following PBPV. Arterial oxygen saturation increased immediately in TOF and dextrocardia patients while there was no immediate increase in other three; the latter is presumably related to dynamics of interatrial shunting. Immediate surgical intervention was avoided in all six patients.

On follow-up 3 to 6 months after PBPV, all infants were thriving well with decreased hypoxemia and polycythemia. Follow-up catheterization data are available in 3 patients, 3 to 6 months following PBPV and in all three, the immediate post PBPV improvement persisted. These data suggest that PBPV offers an excellent palliation of pulmonary oligemia in CCHD, thus, avoiding the risks of immediate surgical palliation and paving for a better result of eventual total surgical correction.

●124 EXERCISE STUDIES IN CHILDREN WITH TRANSPOSITION OF THE GREAT ARTERIES(TGA) AFTER MUSTARD REPAIR. J.Reisman;G.Canny;N.Musewe;D.Wilkes; H.Levison & L.Benson. Cardiol. & Chest Div., Hospital for Sick Children, Toronto, Canada.

The cardiopulmonary responses to exercise of 17 pts (mean age 13y,M/F=11/6) with TGA and Mustard repair(mean age at surgery=19.4 mo) were compared with those of 17 age-matched normal subjects.An incremental exercise test was performed to determine work capacity (Wmax). Heart rate (HR), oxygen saturation ($Sa_{O_2}$),oxygen consumption ($\dot{V}O_2$) and minute ventilation ($\dot{V}_E$) were measured during exercise. Cardiac output($\dot{Q}$) and stroke volume (SV) were subsequently computed during steady state exercise at 50% Wmax by the Indirect Fick ($CO_2$) method,and expressed as a % predicted. During the incremental test, the TGA patients, as compared to the normal subjects, achieved lower maximal values for Wmax, HR, and $\dot{V}O_2$, but not for $\dot{V}_E$ (Table).

| | TGA | Control | P |
|---|---|---|---|
| Wmax (watts/kg) | 2.3 ± 0.6 | 3.3 ± 0.7 | < 0.01 |
| HR (bpm) | 172 ± 14 | 185 ± 11 | < 0.01 |
| $\dot{V}O_2$ (ml/kg/min) | 31.7 ± 7.7 | 45.3 ± 10.5 | < 0.01 |
| $\dot{V}_E$ (L/kg/min) | 1.3 ± 0.3 | 1.5 ± 0.5 | NS |

During steady state exercise the mean ± SD values achieved by the TGA patients for $\dot{Q}$ and SV were 10.4 ± 1.7 L/min (92 ± 2 % pred) and 66 ± 14.5 mls (98 ± 16 % pred) respectively. We conclude that the aerobic capacity of TGA patients following Mustard repair is significantly reduced.

●125 *IS THE PLASMA DIGOXIN IMMUNOREACTIVITY OF PREGNANCY ASSOCIATED WITH DIGITALIS-LIKE (Na+K)ATPase INHIBITION?* Richard Ringel, John Hamlyn, Gerard Pinkas. *(spon by Allen Schwartz) Univ of Maryland, Depts of Pediatrics and Physiology, Baltimore.*

*Digoxin immunoreactivity (DI) has been identified in plasma from women with pregnancy induced hypertension (PIH). Thus, it has been suggested that digitalis-like inhibition of cell Na pumps ((Na+K)ATPase) may play a role in PIH. Digoxin and other cardiac glycosides can be displaced from red blood cells (RBC) by incubation in a Na+ATP solution. By measuring Na pump activity with and without such an incubation, the degree of Na pump inhibition can be calculated.*

*We used this technique to evaluate whether DI is associated with digitalis-like binding to RBC (Na+K)ATPase. DI was detected in all 18 pregnant women, 9 normotensive (.29±.07ng/ml) and 9 with PIH (.22±.06, p > 0.1), but was undetectable in 9 nonpregnant women. Additonally, 7 patients on oral digoxin were studied and had levels of .97±.37ng/ml. RBC (Na+K)ATPase activity increased in all subjects after Na+ATP incubation. Pump activity increased to the same extent in pregnant (23%±6) and nonpregnant (25%±3) women despite the DI present in pregnant subjects. Patients taking digoxin had a twofold greater increase in Na pump activity (52%±6), confirming the ability of this technique to displace digoxin from RBC Na pumps.*

*Digoxin immunoreactivity is present in pregnancy irrespective of blood pressure status and is not associated with digitalis-like inhibition of (Na+K)ATPase.*

126 THE EFFECT OF SALT (Na) INTAKE ON BLOOD PRESSURE (BP) IN OBESITY. A.P. Rocchini, C. Moorehead, D. Bondie, R. Chico, A.R. Snider. University of Michigan.

To study the role of Na intake on hypertension in obesity, 33 obese adolescents (ads) and 15 non-obese ads received, in a random sequence, 2 wks of high Na intake (>250 meq/day) and 2 wks of low Na intake (<20 meq/day). All ads had their BP measured, both on low and high Na diets, in the sitting position every 5 min for 1 hr. Other variables measured include: plasma volume (PV) (Evans blue dye), cardiac output (CO) (2D-Echo-doppler), fasting insulin (INS), and aldosterone (aldo). Compared to non-obese ads when switched from high to low Na diet, obese ads had larger decreases in: mean BP (12.2 ± .7 vs 1.7 ± 2.2 mmHg $p<0.01$), PV (974 ± 114 vs 484 ± 111 ml $p<0.01$), and CO (2.6 ± .3 vs 0.6 ± 0.5 L/min $p<0.05$). Obese ads also had higher INS (27.3 ± 2.4 vs 10.1 ± 0.8 uU/ml $p<0.01$) and aldos (15.2 ± 1.3 vs 11.7 ± 2.5 ng/dl $p<0.01$ (high Na) and 25.2 ± 2.1 vs 18.1 ± 2.9 ng/dl $p<0.05$ (low Na)). When changed from high to low Na diet, 23/33 obese and 1/15 non-obese ads ($p<0.01$) decreased their mean BP by >10 mmHg. Comparing the 23 obese ads whose BP's changed by >10 mmHg with the 10 obese ads whose BP did not change, we observed that the salt-sensitive ads also experienced the largest changes in PV (1048 ± 119 vs 398 ± 108 ml $p<0.05$) and CO (3.1 ± 0.4 vs 1.5 ± 1.3 L/min $p<0.05$) and had the highest INS (30.0 ± 2.9 vs 21.1 ± 3.5 uU/ml $p<0.05$) and aldo (16.3 ± 1.2 vs 11.2 ± 2.4 ng/dl $p<0.05$ (high Na) 27.2 ± 2.5 vs 21.2 ± 3.2 ng/dl $p<0.05$ (low Na)). Thus unlike non-obese ads, obese ads' BP is very dependent on Na intake. It appears that the elevated INS and aldos that occur in many obese ads maybe responsible for the fluid retention and elevated BP associated with a high sodium intake.