169 REPETITIVE RESPONSES DURING VENTRICULAR STIMULATION IN CHILDREN. Ashok Mehta, Grace Wolff, Sharanjeet Singh, Arthur Pickoff, Dolores Tamer, Otto Garcia, Henry Gelband. Univ. of Miami School of Medicine, Dept. of Pediatrics, Miami, Florida.

Repetitive ventricular responses (RVR) to ventricular extrastimulus (VES) can result from either bundle branch reentry (BBR) or intraventricular reentry (IVR). BBR was diagnosed by electrophysiologic studies (EP) when 1) QRS configuration of RVR on the surface ECG resembled that of the paced beat from right ventricular apex (RVA), 2) the reentrant beat had a left bundle branch block pattern, and 3) a His bundle or right bundle deflection preceded the RVR. BBR was initiated by VES during programmed stimulation of RVA in 9 of 98 (9%) children (ages 8 months - 18 yrs) with heart defects. BBR occurred in 5 of 59 (8.4%) preoperative and 4 of 39 (10.2%) postoperative patients (pts). IVR was defined electrophysiologically by 1) QRS configuration of RVR on the surface ECG different from that of the RVA paced beat, 2) right bundle branch block type of reentrant beat and 3) absent His bundle or right bundle deflection preceding the reentrant beat. IVR occurred in 2 of 39 postoperative pts (2-10 yrs after surgery) who did not have ventricular arrhythmias. Sustained ventricular tachycardia was not induced by VES in any of our patients. Thus, BBR is a common mechanism for RVR and may represent a physiologic response of the His-Purkinje system. In contradistinction, RVR are infrequently caused by IVR. The risk of ventricular arrhythmias or sudden death in pts with IVR needs further elucidation.

172 PULMONARY VASCULAR RESPONSE TO DIGOXIN. J.M. Milstein, B.W. Goetzman and S.H. Bennett. Univ. of California, Davis Sch. of Med., Dept. of Pediatrics, Davis, CA.

The effects of digoxin on pulmonary vascular resistance (PVR) were evaluated in 13 normoxemic (N) newborn lambs (0-3 days of age) with normal PVR and in 4 of the 13 with hypoxic (H) induced increase in PVR. Lambs were anesthetized with chloralose and instrumented to enable continuous measurement of cardiac output (CO), pulmonary arterial pressure (PAP), left atrial pressure (LAP) and PVR. Digoxin was injected into a central venous catheter in doses ranging from 10-20 μg/kg. The mean (± SD) increase in PVR was 22.3% (± 21.4) in the N lambs ($p < 0.001$) and only 2% (± 8.5) in the H lambs (NS). The mean duration of the response was 199 sec (± 180). Since the LAP was unchanged, CO increased 7.2% (± 14) in the N lambs and 2.5% (± 6.9) in the H lambs, and the PAP increased 23.1% (± 16.2) in the N lambs and 3.9% (± 2.1) in the H lambs, the change in PVR appears to be due to a direct pulmonary vasoconstrictive response to digoxin. The blunted response in H lambs may have occurred because they were already maximally constricted or because digoxin and H induced vasoconstriction share a common mechanism such as electromechanical coupling with inhibition of calcium influx. Attempts to confirm the latter mechanism, using verapamil, were unsuccessful because even small doses proved lethal to lambs. The transient increase in PVR produced by digoxin may actually be beneficial to infants with patent ductus arteriosus and L→R shunting but could further compound existing pulmonary hypertension in other sick infants. A cautious approach to digitalization of newborns is warranted.

● **170** EARLY CLOSURE OF THE PATENT DUCTUS ARTERIOSUS (PDA): A CONTROLLED STUDY. T. Allen Merritt, J. Peter Harris, and Klaus J. Roghmann (Spon. by Donald L. Shapiro) U. of Roch. School of Med., Strong Mem. Hosp., Dept. of Peds., Roch., NY

A controlled trial of early closure of PDA with indomethacin versus closure of PDA after failure of medical management was performed in 24 very low birthweight infants with severe RDS requiring mechanical ventilation. End points in the study design were freedom from bronchopulmonary dysplasia and 6 month survival. No differences in birthweight, gestational age, Apgar scores, or age of first PDA diagnosis were found between the two groups. Infants undergoing early PDA closure (48.8±16.0 hrs) versus the control infants (167.4±162.7 hrs) had significantly reduced BPD (18.2 vs. 88.9% $p<.0027$) and greater survival ($p<.0014$) than infants undergoing PDA closure because of cardiopulmonary decompensation. Our data suggest that early intervention to eliminate adverse effects of the PDA can have greatest benefit if closure occurs before pulmonary damage, associated with congestive heart failure, pulmonary edema, and their effects on lung function, is beyond a state of physiologic repair. (Supp. by HD-13279).

173 ELECTROCARDIOGRAPHIC-ECHOCARDIOGRAPHIC CORRELATION OF ATRIAL ENLARGEMENT DURING CHILDHOOD Jeffrey Moak, Ehud Krongrad, College of Physicians and Surgeons, Columbia University, N.Y.C., N.Y. 10032

Electrocardiographic(ECG) criteria for left atrial enlargement (LAE) are considered to accurately reflect left atrial dimensions. No data are available correlating left atrial size (LAS) and ECG criteria for LAE in children. ECG and echocardiograms (Echo) of ninety-five patients(pts) (age-1 day to 18y, median 7mos.)were studied to determine if LAE as manifested by four different ECG criteria accurately reflect LAE as determined by LAS and LA/AO ratio on echo, clinical and catheterization data. Four cardiac defects known to cause LAE were selected for study. There were 27 pts with ventricular septal defect(Group 1); 15 pts with mitral insufficiency(Group 2); 25 pts with patent ductus arteriosus(Group 3); and 28 pts with cardiomyopathy(Group 4). Our results show that 1)in pts with no ECG criteria for LAE 67% and 96% had increased LAS and LA/AO ratio on echo respectively. 2)in pts with LAE on ECG 67% and 73% had increased LAS and LA/AO ratio on echo. 3)66% and 57% of pts with increased LAS and LA/AO ratio by echo showed LAE by ECG. 4)with increasing magnitude of LAS by echo, LAE by ECG became more predictive. Overall sensitivity, and predictive value of LAE by ECG criteria were 66% and 67% and for the groups, Gr 1: 100%, 33%, Gr 2: 75%, 90%, Gr 3: 21%, 100%, Gr 4: 81%, and 81% respectively. We conclude that current ECG criteria for LAE in children are not as sensitive or predictive as previously suggested.

171 HEMODYNAMIC BASIS FOR RENAL FAILURE IN REYE SYNDROME. John J. Mickell, Wallace F. Berman, John D. Ward. (Spon by H. Maurer) Medical College of Virginia, Department of Pediatrics, Richmond, Virginia.

The relationship of intravascular volume depletion (IVVD) to hemodynamics and renal failure in Reye syndrome (RS) was studied in three comatose children managed with mannitol osmotherapy (dosage total 7 ± 0.7 gm/kg) without barbiturate loading. Common hemodynamic parameters proved insensitive indicators of IVVD, remaining elevated (CVP 8 ±2 torr, PWP 10 ± 2 torr, MAP 109 ± 30 torr, CI 3.2 ± 0.9 l/min/m^2) despite microcardia, reduced stroke index (25 ± 8 ml/beat/m2), and intense peripheral vasoconstriction (SVR 33 ± 7 units). Nonoliguric renal failure (hourly output 2.7 ± 1.2 ml/kg) developed in 2 of 3 cases (peak BUN 50 ± 2 mg/dl and peak creatinine 3.3 ± 0.5 mg/dl). Peak serum osmolality was 337 ± 6 mOsm/l. Systemic vasoconstriction responded to volume expansion on 6 occasions (5% albumin 11 ± 6 ml/kg) with a 38% decrease (P=0.005) in SVR (20.5 ± 2 units) and a 41% increase (P=0.047) in stroke index (35 ± 12 ml/beat/m^2). Filling pressures rose slightly (CVP 10 ± 2 torr, PWP 11 ± 3 torr), MAP fell slightly (92 ± 11 torr), and CI rose (P=0.010) by 28% (4.1 ± 0.7 l/min/m^2). Fluid challenge effects were transient but diagnostic. Renal failure resolved with liberalized fluid intake. The data suggest that a vasoconstrictive response to IVVD may cause a redistribution of renal blood flow and play a role in the development of renal failure in RS.

● **174** EFFECTS OF HYPOXIA AND REOXYGENATION (re-O_2) ON CREATINE KINASE (CK) RELEASE AND TISSUE Ca^{2+} UPTAKE IN THE NEWBORN MYOCARDIUM. Toshio Nakanishi, Helen H. Young, Tatsuo Shimizu, Kenya Nishioka, Jay M. Jarmakani, UCLA Medical Center, Dept. Pediatrics, Los Angeles, California

Sarcolemmal damage and tissue Ca^{2+} uptake were determined during hypoxia and re-O_2 in isolated, arterially perfused newborn (NB) and adult (A) rabbit hearts. Tissue $^{47}Ca^{2+}$ uptake was measured by γ-probe. CK release from the tissue was determined in the venous effluent and was used as an indicator of the sarcolemmal damage. During 40 min of hypoxia, tissue Ca^{2+} was unchanged. CK release (IU/g dry wt) was observed only in the A. During re-O_2, tissue Ca^{2+} increased [ΔCa^{2+} (mmol/kg dry wt)] and CK release was observed, and these two values in the A were significantly greater than in the NB. Recovery of mechanical function [+dT/dt (max), % of control] during re-O_2 in the NB was significantly greater than in the A.

	40 min Hypoxia			re-O_2		
	perfusate	n	CK release	+dT/dt (max)	ΔCa	CK release
NB	Glucose (+)	6	0	90 ± 12	0.5 ±0.3	1+1
	Glucose (-)	6	0	66 ± 8	1.7 ±1.1	5+3
A	Glucose (+)	6	1+1	33 ± 9*	8.1 ±1.5*	54±13*
	Glucose (-)	6	25±7*	2 ± 1*	14.2 ±1.5*	147 ±24*

(*=P< 0.05; NB vs A)

These data suggest that tissue Ca^{2+} gain and/or sarcolemmal damage may be responsible for the impaired recovery of myocardial mechanical function after hypoxia.