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EXERCISE BLOOD PRESSURE RESPONSE-A PREDICTOR OF SEVERITY IN AORTIC STENOSIS: Bruce S. Alpert, Teruo Izukawa, William B. Strong (Spon by P.S. Rao) Section of Pediatric Cardiology, Medical College of Georgia, Augusta

To assess whether the increase(I) in systolic blood pressure (SBP) with exercise may predict the severity of aortic stenosis (AS), we performed treadmill exercise tests on 116 healthy children and 40 with AS. The Ellestad or Bruce protocol was used in all subjects. A maximal test was determined by heart rates 180 or extreme fatigue. SBP was obtained at rest and within 15 seconds of termination of exercise. There was no relation between SBPI and either sex or age in either group. The AS patients had a lower SBPI, 30.6 ± 14.9 mmHg, than the control group, 43.1 ± 18.7 mmHg ($p < 0.001$). SBPI values of < 35 or ≥ 35 mmHg separated patients with left ventricle to aorta gradients above and below 50:

	SBP Increase (mmHg)	
	< 35	≥ 35
Gradient (mmHg)	≤ 50	18
	> 50	13

If a SBPI value of ≥ 35 is obtained, there is a low probability (7%) of a gradient > 50 . Of 9 AS patients with gradients > 50 mmHg, 8 had SBPI < 35 . Exercise SBPI appears to be a valuable part of the total non-invasive assessment of children with AS. While a low value is not always predictive of a high gradient, a high value (≥ 35) suggests AS not of sufficient severity to routinely warrant catheterization.

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DEVELOPMENTAL CHANGES IN FETAL AND NEONATAL CARDIAC CONTRACTILITY. Page A.W. Anderson, Andres Manring and Carlyle Crenshaw, Jr. Duke University School of Medicine, Department of Pediatrics, Durham, North Carolina.

In order to better understand how cardiac contractility changes with development in the infant, six fetal lambs (119-132 days gestation) were chronically instrumented to monitor left ventricular pressure, peak first derivative of pressure ($\dot{P}_{max}$), and end-diastolic (ED) and end-systolic (ES) dimensions. The lambs were studied up to and following birth (gestational age 137-142 days). Descriptors of contractility were $\dot{P}_{max}$, percent fractional shortening (ED-ES/ED;FS) and post-extrasystolic potentiation ($\dot{P}_{max}$ of potentiated systole/ $\dot{P}_{max}$ of previous regular systole; FIR). ED increased with gestational age (e.g. 15.8 ± 0.4 mm, $\pm$ SD, 122 days gestation; 22.9 ± 0.3 mm, 140 days), but mean $\dot{P}_{max}$ for the lambs ($1462-2452$ mmHg/s) did not increase significantly until 24 hours prior to delivery, rose by 50-120% at 3 hours of age, and then fell back to fetal levels by 7-10 days of age. Fetal FS (21-31%) followed a time course similar to that of $\dot{P}_{max}$. In contrast, fetal FIR increased significantly until 24 hours prior to delivery, fell significantly with delivery and then rose back to fetal values and higher by 30 days of life. All the changes around birth are similar to those induced by isoproterenol infusions and may reflect changes in sympathetic tone. In contrast, the long term increase in FIR is similar to that induced by mild hypertrophy suggesting that developmental changes in contractility share a common origin with those of mild hypertrophy.

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ECHOCARDIOGRAPHIC CORRELATES OF LEFT VENTRICULAR HYPERTROPHY DIAGNOSED BY ELECTROCARDIOGRAPHY IN CHILDREN Peter Baron, (Spon. by Paul C. Gillette),

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To determine the anatomic correlates of electrocardiographic left ventricular hypertrophy (LVH) we performed an M-mode echocardiogram (echo) in 92 children (29 normals, 27 with aortic stenosis and 36 with aortic insufficiency). The ECG was normal in 53 and met criteria for LVH in 39 (including 1 normal). From the echo we measured LV end-diastolic diameter (EDD), septal and posterior wall thickness (PWT) and calculated LV mass. All normal subjects had a normal echo.

Of the 39 children with LVH by ECG, 19 (50%) had increased EDD and PWT, 8(20%) had only increased EDD, 8(20%) had only increased PWT and 4(10%) had a normal echo. In 20(51%) LV mass was increased. In all patients (pts) with a product of EDD X PWT $> 5.2 \text{ cm}^2$ the ECG showed LVH.

Of the 53 children with a normal ECG, 44(83%) had a normal echo, 6(11%) had only increased PWT, 3(6%) had only an increased EDD and none had increased EDD and PWT. LV mass was increased in 7(13%); 11(24%) of pts with an abnormal echo had a normal ECG.

We conclude: (1) LVH on ECG may be due to an enlarged LV cavity, increased PWT or both, (2) children with EDD X PWT $> 5.2 \text{ cm}^2$ consistently have LVH on ECG and (3) Most children with aortic valve disease and LVH by ECG have abnormal echo measurements.

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CONSTRICTION AND RELAXATION OF THE DUCTUS ARTERIOSUS IN SMALL PRETERM INFANTS. Barry G. Baylen, Miriam Heiferman, Mary Krouse, and George C. Emmanouilides.

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Small prematures with respiratory distress (RD) are at high risk for patent ductus arteriosus (PDA) and left-right shunt. Nearly all infants < 1000 gm ($n=32$) admitted to NICU over a 12 mos period underwent early evaluation for PDA. Mean birth weight was 827 ± 32 gm ($\pm$ SE), gestational age 27 ± 3 wk and age at study 12 ± 2 hr. Severity of RD was expressed as an RD score (< 3 mild, > 5 severe). PDA was evaluated by echo (LA/Ao, left ventricular dimensions=LVED) and aortography. Based on the caliber of PDA (Ductus/aorta diameter=PDA/Ao) infants were placed in 3 groups.

	$< 1/3$ ($n=17$)	$1/3-2/3$ ($n=6$)	$> 2/3$ ($n=9$)
PDA/Ao (n)			
RD score	$4.3 \pm .84$	6.3 ± 1.9	7.1 ± 1.2
LA/Ao ($m \pm$ SE)	1.31 ± 0.7	$1.25 \pm .03$	$1.57 \pm .11$
Abnormal LVED (n)	4	1	6

The PDA was constricted in 17 grp I infants who also had less RD ($P < .01$). Those infants with larger PDA had larger LA/Ao ($P < .05$). However, 10/17 infants with constricted ductus and mild RD had abnormal LA/Ao (4 with abnormal LV). Only 15/32 ultimately received indomethacin (4 grp I, 4 grp II).

Conclusions: (1) Early constriction of PDA is a common occurrence in very small infants with mild-moderate RD. (2) Abnormal echo dimensions in small asphyxiated infants may be related to factors such as left-right ductal shunt, LV dysfunction or artifacts. (3) Ductal relaxation occurs and contributes to the later increase of left-right shunting.

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TWO-DIMENSIONAL ECHOCARDIOGRAPHY (2D ECHO) IN THE CLOSED CHEST NEWBORN LAMB: METHODOLOGY AND ANATOMICAL OBSERVATIONS. Barry G. Baylen, Yoshiro Yoshida,

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Echocardiography has not been applied to investigation of cardiac anatomy or function in newborn experimental animals. Consequently, we have investigated the feasibility of 2D Echo for cardiac imaging in the conscious closed chest lamb.

M-mode and 2D Echo studies were obtained from 16 closed-chest recumbent lambs: (age=120 days gestation-term-3 weeks; wt=1.8-8 kg). All cardiac chambers and valves were consistently and successfully imaged by transducer angulation from a left and a right precordial position. Cardiac structures were verified by contrast echo using selective injections of green dye and echocineangiographic studies. In addition, postmortem examinations demonstrated anatomical relationships and the orientation of cardiac chambers to the echo transducer. The ventricles are in a "side by side" position and optimal imaging of the left ventricular "long axis" was obtained from a right thoracic position. Complete study was readily accomplished in 10-20 minutes.

We have examined the relevant anatomical relationships and developed a method for chronic M-mode and 2D echo study of the conscious closed chest lamb. It is believed that echo can be used for the investigation of cardiac function in animal models simulating human neonatal cardiopulmonary distress. Such application could potentially expand the role of echocardiography in the human neonate.

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THE EFFECTS OF OUABAIN IN LAMBS WITH DEPRESSED MYOCARDIAL FUNCTION William Berman Jr., Steven M. Yabek and Ramona Shortencarrier. The University of New

Mexico, School of Medicine, Bernalillo County Medical Center, Department of Pediatrics, Albuquerque, New Mexico.

The inotropic effects of digoxin have been difficult to demonstrate in healthy fetal and newborn lambs. We examined the physiologic effects of digoxin in six newborn lambs in whom myocardial function had been depressed by halothane anesthesia (0.5-1.0%). Resting measurements of arterial and venous pressures, cardiac output, left ventricular dp/dt, and the left ventricular pre-ejection period (PEP) to left ventricular ejection time (LVET) ratio were made prior to ouabain administration ($50 \mu\text{g} \cdot \text{kg}^{-1}$ bolus followed by $0.05 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ for 45 minutes). At rest, mean cardiac output ($369 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$), dp/dt ($3833 \text{ mm Hg} \cdot \text{sec}^{-1}$), PEP/LVET (0.306) and stroke work ($1320 \text{ mm Hg} \cdot \text{ml}$) were not affected by ouabain. Forty-eight hours later, halothane anesthesia via controlled ventilation reduced cardiac output to $262 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, increased PEP/LVET to 0.411, reduced stroke work to $766 \text{ mm Hg} \cdot \text{ml}$ and reduced left ventricular dp/dt to $2758 \text{ mm Hg} \cdot \text{sec}^{-1}$; heart rate and systemic vascular resistance did not change. Ouabain administration during the halothane anesthesia improved myocardial function indices to levels not different from control values. These studies suggest the apparent resistance of healthy, immature sheep to the inotropic effects of cardiac glycosides may reflect their already high level of resting function rather than end organ resistance to drug action.