

**139** THE ROLE OF HEART RATE AND AFTERLOAD ON CARDIAC OUTPUT IN NEWBORN LAMBS. Robert E. Shaddy, Carlos Li, Michael Tyndall, David F. Teitel, Abraham M. Rudolph. Cardiovascular Research Institute and Dept. of Pediatrics, Univ. California, San Francisco.

Previous studies have suggested that increases in heart rate (HR) may represent the primary mechanism available to the newborn for increasing cardiac output (CO). In this study, we examined the independent effects of HR and afterload changes on CO in newborn lambs. Atrioventricular node ablation was performed in eight newborn lambs (3-10 days of age) by formalin injection, and the right ventricle was paced at a HR of 200 (baseline). Nine studies were performed in young lambs (less than 12 days of age), and nine studies in older lambs (greater than 12 days of age). CO/kg increased progressively with increasing HR. The maximum CO/kg attained was similar for both groups, but this was achieved at a higher HR in the younger lambs. From a baseline HR of 200, the maximum increase in CO/kg attained by increasing HR was 6.7% in the younger lambs, and 8.9% in the older lambs. At lower HR's (70/min), oxygen consumption (VO<sub>2</sub>) decreased, and systemic vascular resistance (SVR) increased. With a fixed HR of 200, nitroprusside caused an increase in CO/kg of 10.5% in the younger lambs, and 11.8% in the older lambs. Phenylephrine caused a decrease in CO/kg of 19.3% in the younger lambs, and 10.6% in the older lambs. HR above baseline has limited value in increasing CO in newborn lambs. At lower HR's, CO and VO<sub>2</sub> fall, and SVR increases. Decreasing afterload produces modest increases in CO. Increasing afterload results in decreases in CO, and these changes were most striking in the younger lambs.

**140** MATERNAL DIABETIC CONTROL AND HYPERTROPHIC CARDIOMYOPATHY (HyC) IN INFANTS OF DIABETIC MOTHERS (IDM). Pauline Sheehan, Thomas Rowland, Bhavesh Shah, Vincent McGravey, Edward Reiter. Dept. of Peds, Baystate Med. Ctr., Springfield, MA.

Transient HyC occurs frequently in IDM. This may result from fetal hyperinsulinemia secondary to maternal hyperglycemia in the 3rd trimester. Prospective study was designed to determine the incidence of IDM HyC and to investigate its relationship to 3rd trimester maternal diabetic control, measured by HbA<sub>1c</sub>. 20 insulin dependent diabetic women and their infants and 30 normal babies (controls) were enrolled in the study. Maternal HbA<sub>1c</sub> was obtained at the time of delivery and M-mode echocardiography was performed on all infants at 24-72 hrs. age. Cord blood glucose, follow-up glucose at q 30 min x 4 and chest x-ray were obtained in all IDM. Diagnosis of HyC was based on ratio of absolute ventricular septal thickness related to body weight (S/BW-mm/kg).

	IDM (n=20)	Control (n=30)
Mean GA wks (range)	38.0 (36-40)	40.2 (38-42)
Mean BW kg (range)	3.8 (2.7-4.7)	3.5 (2.6-4.4)
% LGA (>90 percentile)	60	30
Mean Serum HbA <sub>1c</sub> % (range)	7.6 (5.7-9.4)	6.4 (4.8-7.7) P<0.001
Abs. Vent. Sept. Thick/BW	1.19 ± 0.29	0.96 ± 0.17 P<0.002

S/BW ratio 1.3 was seen in 7 IDM (35%) and in 1 control infant (3%), Chi square=8.95, P<.01. None of these subjects had clinical evidence of congestive heart failure. No correlation existed between maternal HbA<sub>1c</sub> at term and incidence of neonatal HyC, macrosomia and hypoglycemia. These data do not support 3rd trimester hyperglycemia with consequent fetal hyperinsulinemia as a trigger mechanism for HyC.

**141** ECHOCARDIOGRAPHIC PREDICTORS OF PULMONARY HYPERTENSION IN CONGENITAL HEART DISEASE. E.B. SIDERIS, M.D., SOULA MESSINIS SIDERIS, R.N.

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Critical review of Echocardiographic (Echo) predictors of pulmonary artery pressure (PA), was attempted in 37 cases who had Echos within 10 days from cardiac catheterization. Preejection period to ejection time (RVPEP/ET), time to peak velocity corrected for RVET (TPV/RVET) and negative presystolic velocity (a wave) were used. All studies were done in infants and children with 4-chamber hearts, normally related great vessels and various defects. All 3 indices correlated poorly with PA in the mixed group. The presence of the "a wave" could separate patients with PA systolic less than 30 mmHg in 30/37 cases. ("a wave" indistinguishable in 4 cases, 3 false estimates) RVTPV/ET less than .35 could separate patients with PA systolic over 30 mmHg if cases with a pulmonary gradient more than 20 mmHg were excluded and RV outflow measurements were used in patients with patent ductus arteriosus (Technical estimation impossible in 5 cases). RVPEP/ET more than .35 could separate cases with PA systolic over 30 mmHg. (Technical estimation impossible in 5 cases, 6 false estimates).

We conclude that there are both technical problems and limitations in the application of all used indices for the non-invasive prediction of pulmonary hypertension in congenital heart disease. Invasive estimation of PA pressure is imperative in cases under question.

**142** ELECTROPHYSIOLOGIC EVALUATION OF TETRALOGY OF FALLOT (TOF) REPAIR: EARLY POSTOPERATIVE STUDIES. Sharanjeet Singh, M. Andre Vasu, Vincent Parnell, Robert Boxer, Michael LaCorte, Division of Pediatric Cardiology, North Shore University Hospital, Manhasset, NY 11030

Cardiac arrhythmias are common after repair of TOF and are associated with sudden death. We performed 9 electrophysiologic studies in 5 patients (pt.), ages 6-16 yrs. with TOF 1-26 days after repair to identify pt. at risk for arrhythmias. Rapid atrial pacing and programmed atrial and ventricular stimulation were carried out using intracardiac catheters (2 pt.-2 studies) or epicardial pacing wires placed intraoperatively on the right atrium, right ventricular apex (RVA) and outflow tract (RVOT, 5 pt.-7 studies). Epicardial pacing studies were performed at the bedside or in the intensive care unit (ICU). Junctional rhythm was present at the first study in 2 pt. Wenckenbach periodicity occurred at an average cycle length of 236 + 88 msec. Dual pathway atrio-ventricular conduction, anterograde and retrograde, was observed in 1 pt. Atrial flutter was induced in 1 pt. Ventricular effective (VERP) and functional (VFRP) refractory periods (RP) determined at the RVA (VERP 193.8 + 19.3, VFRP 212.5 + 19.8 msec) and RVOT (VERP 197.5 + 19.2, VFRP 215.3 + 19.3 msec) were similar. The ventricular RP determined by intracardiac or epicardial stimulation were identical. Repetitive ventricular responses were observed in 1 pt. Single (S<sub>1</sub>, S<sub>2</sub>) or double (S<sub>1</sub>, S<sub>2</sub>, S<sub>3</sub>) extrastimuli failed to induce ventricular arrhythmia. These observations suggest that electrophysiologic studies can be safely carried out in the immediate post operative period at the bedside or in the ICU and may be helpful in identifying pt. at risk of developing cardiac arrhythmia.

**143** MYOCARDIAL METABOLIC AND HEMODYNAMIC CHANGES DURING ACUTELY INDUCED ASPHYXIA IN FETAL LAMBS. R. Kendrick Slate, Anthony M. Mills, and Abraham M. Rudolph. Cardiovascular Research Institute and Dept. of Pediatrics, Univ. California, San Francisco.

Two days after instrumentation, we measured heart rate (HR), arterial blood pressure (BP), left ventricular myocardial blood flow (MBF), and myocardial consumption of oxygen, glucose and lactate in 6 fetal sheep (118-128 days gestation) *in utero* during acute progressive asphyxia induced by gradual constriction of a common uterine artery snare. Ascending aortic blood oxygen content decreased by 70% from 7.5 to 2.2 ml/dl. Ascending aortic blood oxygen saturation fell from 60% to 15%. pH fell from 7.40 to 7.04, while PCO<sub>2</sub> increased from 46 to 72 torr. Coronary sinus PO<sub>2</sub> fell from 11.6 to 6.6 torr. MBF increased 317% above the control of 239 ml/100g/min. Hemoglobin concentration was maintained from 9.3 to 10.8 g/dl during the study. HR, BP, and rate-pressure product remained unchanged. Myocardial oxygen consumption and extraction were maintained until an arterial oxygen saturation of 20-25%, after which they decreased. During hypoxemia, myocardial glucose consumption markedly increased and net lactate uptake by the myocardium occurred until an arterial PO<sub>2</sub> of 12 torr (O<sub>2</sub> saturation of 21%) was reached. Below this level lactate production occurred in 3 of 6 fetuses. Thus the fetal lamb has a marked reserve to increase myocardial blood flow and maintain myocardial oxygenation and substrate delivery during acute severe asphyxia.

**144** SERUM ARGININE VASOPRESSIN RESPONSES IN CONGENITAL HEART DISEASE. Julian M. Stewart, Guillermo Zeballos, Paul K. Woolf, Harry S. Dweck and Michael H. Gewitz, New York Medical College, Departments of Pediatrics and Physiology, Valhalla, New York.

In addition to its antidiuretic properties, arginine vasopressin (AVP) is a potent vasoactive hormone. If unopposed by baroreceptor mediated reflexes, even low serum levels of AVP may exert pressor effects. Since early life is associated with an enhanced ability to release AVP and with an increase in its cardiovascular effects, we evaluated whether congestive heart failure (CHF) is a stimulus for AVP release. AVP levels were measured by RIA using the modified Bentonite technique in 40 infants and children divided into 5 groups: Group I (n = 7) with CHF; Group II (n = 6) with cyanotic heart disease; Group III (n = 3) with persistent neonatal pulmonary hypertension; Group IV (n = 12) stressed infants with respiratory disease but no heart disease; and Group V (n = 12) healthy unstressed controls. Group IV had statistically higher AVP levels (13 pg/ml ± 6) when compared to Group V normals (6 pg/ml ± 2) (p < 0.05). Groups II & III were not significantly different from Group V (7 pg/ml ± 5 and 6 pg/ml ± 2 respectively). CHF patients (Group I) had variable AVP levels with peak levels markedly elevated above other groups (102 pg/ml ± 135), (p < 0.05) and further increased with worsening clinical status.

Additional data suggest that hyperosmotic angiographic contrast media increase AVP levels by 2 to 10 times pre-dye levels in most instances (n = 12 of 15 patients, p < 0.02).

We conclude that AVP levels are increased in children with CHF and that diagnostic interventions such as angiography may further elevate AVP. This may result in increased cardiac afterload further impairing already diminished cardiac function.