145 LONG-TERM FOLLOW-UP OF PATIENTS WITH REPAIR OF AORTIC COARCTATION IN INFANCY. Jill H. Morriss, Dan G. McNamara, and Denton A. Cooley, Baylor College of Medicine, Texas Children's Hospital, Dept of Pediatrics (Cardiology) and Surgical Section Texas Heart Institute, Houston.

Twenty nine patients who survived surgical repair of thoracic coarctation of the aorta in infancy have been followed 10 - 22 (mean 14.8) years. Preoperatively, all patients had congestive heart failure refractory to medical management; 27/29 were under 6 months of age. Of the 29, 8 had associated left-to-right shunts at ventricular (7) or atrial (1) level. Initial procedure was resection and end-to-end anastomosis with continuous suture. Subacute bacterial endocarditis occurred in 1 patient in this series who had associated aortic valve stenosis. Symptoms of subclavian steal syndrome occurred 9 years postoperatively in 1 patient. No residual hypertension was present in any patient unless associated with recurrence of coarctation. There were no deaths. Follow-up catheterization on 25 patients has been performed with specific attention to recoarctation. Reoperation was required in 10/29 at a mean age of 13 years with reoperation anticipated in at least 4 more patients. At the time of repeat surgery, patch angioplasty was performed in 5/10, resection and end-to-end anastomosis in 2/10, and Dacron tube grafts were used in 3/10.

In this series, significant restenosis after coarctation repair in infancy has occurred in 48% of patients (14/29), an additional 5/29 have gradients across the coarctation site of greater than 25 mm Hg. Only 3/29 have neither residual gradient nor recoarctation angiographically proven by postoperative catheterization.

146 THE EFFECT OF HYPOXIA ON MYOCARDIAL HIGH ENERGY PHOSPHATE CONTENT IN THE NEWBORN AND ADULT RABBIT Takafumi Nagatomo, Makoto Nakazawa, Jay M. Jarmakani, UCLA Medical Center, Department of Pediatrics, Los Angeles, California.

The effect of hypoxia on high energy phosphate content in the myocardium of the newborn and adult rabbit was determined and compared with mechanical function. All studies were done using the arterially perfused septa, and hypoxia was induced using perfusate equilibrated with 95% N_2 and 5% CO_2. Adenosine triphosphate (ATP) and Creatine Phosphate (CP) were enzymatically determined by a fluorometric method. In the newborn, myocardial ATP content decreased after 2 minutes (min) of hypoxia but returned to control level between 10 and 30 min of hypoxia, and then decreased to 50% of control at 60 min. In contrast ATP content in the adult rabbit decreased continuously during hypoxia reaching 53% of control at 30 min and 29% at 60 min. The change in CP in both groups was similar to ATP. In the adult, reoxygenation after 10 min of hypoxia resulted in complete recovery, but in partial recovery after 30 or 60 min of hypoxia. The changes in myocardial ATP content was similar to changes in mechanical function in both groups. The addition of Iodoacetic acid to the hypoxic perfusate in the newborn rabbit resulted in a significant depression in mechanical function and ATP content. The data suggest that the myocardium in the newborn as compared to the adult might be dependent on glycolytic rather than aerobic metabolism.

147 EFFECTS OF DOPAMINE ON RENAL AND CARDIAC HEMODYNAMICS DURING DEEP HYPOTHERMIA. Douglas B. Nielsen and Herbert D. Ruttenberg (Spon. by John J. Herbst). University of Utah College of Medicine, Department of Pediatrics, Salt Lake City, Utah.

The technique of deep hypothermia (DH) for cardiac surgery results in altered renal and cardiac function: low flows and high resistances. We studied the effects of dopamine infusion (6.1 to 7.5 ug/kg/min) on renal and cardiac hemodynamics in 6 dogs during surface cooling to 20°C rectal (DH) under ether anesthesia; 6 control dogs received no dopamine. Open chest dogs were studied with electromagnetic flow probes on the aortic root and left renal artery. Data were recorded every 2° from normothermia (38°) to 20°C. Dopamine had no significant effects on the hemodynamic parameters. From normothermia to 20°C (DH) there were net decreases in heart rate (81%), mean aortic pressure (44%), cardiac output (66%), renal flow (75%) and pre-ejection period (PEP)/left ventricular ejection time (LVET) (55%). Net increases occurred in renal resistance (256%) and systemic resistance (210%), PEP (180%), and LVET (370%). Stroke volume and LV ejection work increased significantly ($p < 0.005$) to 28°C and then decreased to normothermic values at 20°C. The data indicate no effect of dopamine in the usual clinical doses. Hypothermia resulted in increased inotropic state to 28°C and marked decreases in cardiac and renal hemodynamics at 20°C. We hypothesize that below 28°C, decreased myocardial compliance is an important factor in the decreased inotropism.

148 THE REDISTRIBUTION OF CARDIAC OUTPUT DURING CONGESTIVE HEART FAILURE IN NEWBORN ANIMALS. Jim B. Norton, Jr., Stephen P. Barrier. (Intr. by M. J. Elders) Dept. of Ped., Univ. of Arkansas for Medical Sciences, Little Rock, Arkansas, National Center for Toxicological Research, Jefferson, Arkansas.

The two purposes of this study were to assess the efficacy of a new technique to produce a controlled state of congestive heart failure (CHF) in newborn animals, and to assess alterations in organ flow during CHF. One to 10 day old Spanish goats were studied during general endotracheal anesthesia (Ethrane and N_2O). Flows were measured using the radioactive microsphere technique. CHF was produced with a continuous infusion of sodium citrate (C) into the left atrium. Phenylephrine (P) was used to maintain arterial pressure as cardiac output (CO) fell after addition of C. Hemodynamic changes in this experimental preparation simulate a cardiomyopathy. Flow measurements during C + P infusions compared to control or P periods reflected a sparing of brain flow until total CO fell to extremely low levels. Control flow = 0.61 ml/min · gm^{-1} ± 0.14 SE. Brain flow during C + P = 0.48 ml/min · gm^{-1} ± 0.16 (NS) with normal pressure and mild drop in CO. Myocardial flow increased signficantly ($p < 0.01$) during P infusion and with P + C flow was maintained until CO fell; then subendocardial perfusion was most reduced. Renal flow was the first to fall as CO dropped, followed by skin and skeletal muscle flows. Small bowel perfusion increased with P but fell as CO was reduced by C. During shock systemic arterio-venous shunting occurred, suggested by a rise in the percentage of recirculating microspheres.

149 RECIPROCAL INHIBITION OF CARDIAC RESPONSES TO NOREPINEPHRINE AND INSULIN. Dov B. Nudel, John C. Lee and S. Evans Downing, Departments of Pathology and Pediatrics, Yale Univ. School of Medicine, New Haven, CT.

The effects of insulin (IN) and norepinephrine (NE) on left ventricular contractility (LVc) were studied in 20 piglets. Aortic pressure, cardiac output, heart rate and temperature were controlled, and LV dP/dt max was taken to reflect LVc. Hypoglycemia was prevented by giving glucose as necessary. LVc increased 28% ($p < .02$) 45 min. after IN (20U). Following β-blockade (practolol, 1 mg/kg), IN caused a 55% increase in LVc. NE dose-response (DR) curves were obtained before and after IN. Maximal changes in LVc with each dose of NE (0.5-2.0 ug) were significantly less in hearts exposed to IN. Duplicate DR curves were identical, indicating that prior exposure to NE did not inhibit subsequent responses. However, in those piglets initially subjected to a NE dose-response curve, IN elicited only an 11% increase of LVc, and this was not significant. Thus, the inotropic response was attenuated ($p < .01$) in animals previously exposed to NE. Conversely, in animals first subjected to β-blockade, the response to IN was greater ($p < .005$). These data indicate a reciprocal blocking action by these hormones on piglet myocardium. The cellular mechanisms responsible for these findings remain to be elucidated, but likely involve modulation of Ca++ fluxes.

150 CONTINUOUS DISTENDING AIRWAY PRESSURE IN THE MANAGEMENT OF CARDIORESPIRATORY FAILURE DUE TO PDA. Apostolos N. Papageorgiou and Marie-Françoise Desgranges (Spon. by Harvey J. Guyda) McGill University, Jewish General Hospital, Dept. of Neonatology, Montreal, Canada.

Patent ductus arteriosus (PDA) is a frequent complication in the recovery phase of respiratory distress syndrome. Ligation is recommended when heart failure cannot be controlled medically. Criteria and timing for surgery are controversial. Continuous negative or positive distending airway pressure (C.D.A.P.) was used in five infants < 1500 grams birthweight who had P.D.A. and had failed to respond promptly to routine medical treatment i.e. fluid restriction, digitalization and diuretics. Significant improvement was recorded in the following: 1) Number of apneic spells; 2) heart rate; 3) respiratory rate; 4) chest retractions; 5) pCO_2; 6) radiologic picture of pulmonary edema; 7) tolerance to oral and IV alimentation; 8) daily fluctuation of weight; 9) electrolyte imbalance due to occasional use of diuretics. A negative pressure respirator was used in most cases for obvious technical advantages. Mean pressure was 4cm H_2O. We conclude that C.D.A.P. is helpful in overcoming cardiorespiratory failure due to P.D.A.