

ACUTE RENAL FAILURE (ARF) ASSOCIATED WITH CARDIAC SURGERY IN INFANCY. Russell W. Chesney, Bernard S. Kaplan, Keith N. Drummond and Robert M. Freedom, McGill Univ., Montreal Children's Hosp. Research Inst., Montreal and Johns Hopkins Hosp., Dept. of Ped., Baltimore.

ARF is an important complication of cardiac surgery in infants. Nineteen, aged 2 d.-12 mo., of 183 operated on developed ARF (9%). The defects were transposition 5, VSD 5, interrupted aorta 4, PDA 2, A-V canal 1, pulmonary atresia 1 and single ventricle 1. Ten underwent open heart surgery.

Preoperatively 4 were oliguric. Postoperatively 13 were anuric 1-13 days and 5 oliguric 1-5 days; 1 had polyuric renal failure. All had elevated BUN and serum creatinines. Serum K was  $>6.7$  mEq/L in 17, Ca  $<7.5$  mg% in 10, blood glucose  $<30$  mg% in 9 and 9 had metabolic acidosis. Although heart failure was present in all, ARF appeared to be due to intrinsic renal failure in each patient. The causes identified were: hypotension (14), hypertonic angiocath dye shortly before surgery (?), aminoglycosides (19), dysplastic kidneys (1) and tamponade (3). Fluid and electrolyte management problems were difficult but critical. Insulin could not be given to reduce hyperkalemia in hypoglycemic infants. Furosemide up to 10 mg/kg did not induce diuresis. Only 2 of 13 with anuria survived; 13/14 with hypotension died and all normotensive infants survived. Hypotension was identified as the most important factor in the pathogenesis and prognosis of ARF.

This study is the first to examine in detail ARF as an important complication of cardiac surgery in infants.

#### EFFECTS OF PARASYMPATHETIC BLOCKADE ON THE CARDIOVASCULAR RESPONSE TO HYPOXEMIA IN UNANESTHETIZED FETAL LAMBS

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We studied the effects of parasympathetic blockade on the cardiovascular response to hypoxemia in 4 fetal lambs, of 125-130 days gestation. Catheters were placed in fetal and maternal vessels, an electromagnetic flowprobe was placed on the fetal aorta, and the animals recovered over 5 days. Fetal and maternal arterial pressure, O<sub>2</sub> content, and pH were measured repeatedly during the control state and while the ewe breathed 6-10% O<sub>2</sub> and 3% CO<sub>2</sub>. Cardiac output (CO) and its distribution were measured with labelled microspheres during the control state, parasympathetic blockade with atropine (0.2 mg/kg), and during fetal hypoxemia with blockade. During hypoxemia with atropine blockade, fetal heart rate (FHR) increased 43%, arterial pressure rose 11%. Descending aortic blood flow fell by 25%, and CO decreased by 40% despite the elevated FHR (mean 235). Umbilical blood flow was maintained, and the percentage of CO to the placenta increased from 42% to 65%. The fall in CO, the maintenance of umbilical blood flow, and the redistribution of CO found in these studies were comparable to those previously reported for hypoxemia without blockade. Since changes in fetal CO have been shown to vary directly with FHR other factors must be responsible for the fall in CO which we measured. It is possible that the direct effect of oxygen lack on the myocardium may be significant.

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#### REGRESSION OF MYOCARDIAL HYPERTROPHY

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Regression of hypertrophy was studied in 96 adult rats following aortic debanding (dB). Controls were 146 continuously banded (B), and 116 sham-operated (S) rats. Sacrifice was at 3, 7, 14, 21 and 28 days for LV mass, RNA, DNA and hydroxyproline (OHP). Hemodynamics were obtained at 28 days. Aortic gradients were 60-130 mmHg in B, and none in dB and S. Initial  $(dp/dt)^{-1}$ , in  $\text{Sec}^{-1}$ , was lower in dB (212) than in B (235) and S (234) although not significantly. Early debanding (after 10 days of aortic constriction) showed drop in LV mass from 35% to 11% above C ( $P<0.001$ ), and fall in RNA from 37% to 19% above S ( $P<0.05$ ), by 3 days; thereafter dB and S values were comparable. However, DNA in dB was 32% above S ( $P<0.01$ ) at 10 days, and 12% above S at 28 days ( $P=n.s.$ ). Corresponding OHP levels in dB were 90% above S ( $P<0.001$ ), and 80% ( $P<0.025$ ) above S, respectively. Late debanding (after 28 days constriction) showed LV mass in dB still 12% above S ( $P<0.025$ ) at 21 days; RNA was 34% above S ( $P<0.01$ ) at 14 days. DNA and OHP remained elevated throughout the study period in dB-25% ( $P<0.02$ ) and 118% ( $P<0.001$ ) above S at 28 days. LV mass, RNA, DNA and OHP in B remained consistently high. Although mass and RNA regress after relief of pressure overload, the connective tissue, as measured by DNA and OHP, does not do so readily. Its regression is influenced by the duration of cardiac stress.

EFFECT OF INCREASED OXYGEN-HEMOGLOBIN AFFINITY ON CARDIAC TISSUE OXYGEN UNLOADING OF LAMBS FOLLOWING INHALATION OF CARBON MONOXIDE (CO): M. Delivoria-Papadopoulos, C.D. Park, J.H. Chen, and R.E. Forster II. Univ. of Pennsylvania, Sch. of Med., Philadelphia, Pa.

Previous studies in newborn lambs have shown that a decrease in whole blood oxygen affinity results in an increased coronary sinus P<sub>O<sub>2</sub></sub> and in a decreased cardiac output for the same oxygen consumption ( $\dot{V}_{O_2}$ ). The present studies were designed to investigate the response of the heart to an acute shift of the oxyhemoglobin equilibrium curve to the left. Polyvinyl catheters were placed chronically in the right ventricle, carotid artery and coronary sinus of 4-8 wks. old lambs by direct visualization during thoracotomy. Measurements of blood [HbO<sub>2</sub>] and [HbCO] % saturation, P<sub>O<sub>2</sub></sub>, P<sub>CO<sub>2</sub></sub>, pH and P<sub>50</sub> in these vessels and total  $\dot{V}_{O_2}$  were obtained before and after blood [HbCO] was raised 12-15% by CO inhalation. Arteriovenous oxygen content difference (AVD) and cardiac output were calculated. In the 11 animals studied mean coronary sinus P<sub>O<sub>2</sub></sub> decreased from 24 to 17 mmHg, AVD across the heart decreased from 7.7 to 6.1 ml O<sub>2</sub>/100 ml blood and the total cardiac output increased from 165 to 250 ml/m min. x kg. These data suggest that by increasing blood O<sub>2</sub> affinity there may have been an increase in coronary blood flow presumably as a secondary increase to myocardial hypoxia.

#### PERSISTENT PULMONARY HYPERTENSION IN CYANOTIC TERM NEONATES WITHOUT ANATOMIC HEART DISEASE

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A group of 19 cyanotic term neonates, whose minimal evidence of pulmonary pathology led to an initial diagnosis of cyanotic congenital heart disease, was studied to delineate the nature of the right to left shunt. The mothers' pregnancies were complicated in 90%. Initial Apgar scores were often high (80%  $>7$ ). Electrocardiograms were normal or showed right ventricular hypertrophy.

Five infants (10-39 hours) had cardiac catheterization; none had anatomic heart disease. All had high pulmonary vascular resistance, equalling or exceeding systemic resistance. Pulmonary venous oxygen saturations ranged from 92-100%; simultaneous aortic saturations were 57-83%. Angiography showed massive right to left shunting at the ductus arteriosus in all cases.

The mechanism of the postnatal persistence of fetal right to left flow through the ductus arteriosus is unclear. The associated maternal and placental problems may contribute to chronic fetal hypoxia. Goldberg (Pediat. 48:528, 1971) described an increase in the muscularity of the fetal pulmonary vascular bed after intrauterine hypoxia. This may contribute to these infants' abnormal pulmonary vasoconstriction.

BLOOD PRESSURE STUDIES IN BLACK CHILDREN S.K. Dube, S. Kapoor H. Ratner, F. Tunick, (Intr. by L. Glass) Jewish Hosp. and Med. Center of Brooklyn, Dept. of Pediatrics, Brooklyn, N.Y.

Because of the paucity of data on normal B.P. values in black children, we have studied 1668 asymptomatic black children (827 boys and 841 girls) ranging in age from 4-17 years. These were determined by auscultation using appropriate sized cuffs, mercury manometers and pediatric size Littman stethoscopes. All B.P.s. were obtained under office conditions in the supine position. Apprehensive, uncooperative, crying or obviously excited children were excluded.

The mean systolic B.P. ranged from 99 mm Hg (S.D. 12 mm Hg) in 4 year old boys to 115 mm Hg (S.D. 11 mm Hg) in 16 year olds while mean diastolic pressure ranged from 59 mm Hg (S.D. 12 mm Hg) to 71 mm Hg (S.D. 10 mm Hg). In girls, the mean systolic B.P. ranged from 100 mm Hg (S.D. 13 mm Hg) in 4 year olds to 114 mm Hg (S.D. 11 mm Hg) in 14 year olds; and the mean diastolic pressure ranged from 62 mm Hg (S.D. 12 mm Hg) in 4 year olds to 70 mm Hg (S.D. 8 mm Hg) in 15 year olds.

A significant positive correlation was noted between systolic and diastolic pressures and weight (but not height) in both sexes. Maximum systolic and diastolic pressures were higher in our group of patients than have been previously reported in white children of similar ages. Longitudinal follow up studies of these children based on these data will relate these findings to the development of cardiovascular, renal and other diseases among the black population.