91 TOWARDS AN IN VIVO INDEX OF VASOMOTOR TONE INDEPENDENT OF FLOW MEASUREMENT

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When the heart stops, arterial pressure decays asymptotically to a value different from the venous pressure.Our study was performed to test the hypothesis that this decay is influenced by vasomotor tone and contains information that could lead to the development of an index of vasomotor tone.Twelve anesthetized dogs were subjected to brief periods of cardiac arrest by stimulating the distal ends of the cut vagi.This was done approximately 9 times in each dog at varying levels of vasomotor tone produced by IV nitroprusside and methoxamine and decay of central aortic pressure was recorded.A methematical model of the lumped arterial bed was evolved in which each term had recognizable physiologic meaning.Using multiple nonlinear regression analysis, the pressure-time data were fitted to this model and, in most cases, fitted and observed decays were hardly distinguishable to the eye (r^2 values were greater than .9).It was predicted that asymptotic pressure, calculated using the model, would rise or fall as vasomotor tone rose or fell.The average asymptotic pressures under the influence of methoxamine were significantly greater ($P<.05$) than control values and average asymptotic pressures under the influence of nitroprusside were significantly less ($P<.05$) than average control values.It is suggested that these asymptotes may form the basis for developing an index of vasomotor tone independent of flow measurement.

92 HEMODYNAMIC EFFECTS OF E. COLI ENDOTOXIN IN PUPPIES.

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Serial hemodynamic changes following intravenous E. coli 0111B4 endotoxin were evaluated in 28 puppies, 1-18 days and 270-1800 grams. Serial cardiac outputs were measured by a modified reinfusion dye dilution technique. Heart rate and systemic arterial pressures also were measured. Changes were recorded at 5 min, 10 min and at 10 min intervals thereafter for 1 hour following endotoxin. Heart rate dropped from 210/min to 141/min at 5 min and stabilized at a slightly higher level thereafter ($p < 0.01$). Mean systemic pressure decreased gradually from 70 mm Hg to 42 mm Hg at 1 hour ($p < 0.01$). Cardiac index decreased from 266 ml/min/kg to 131 ml/min/kg at 5 min and continued to decline to 115 ml/min/kg at 1 hour ($p < 0.01$). Systemic vascular resistance rose from 0.467 unit to 0.846 unit at 5 min and remained elevated at slightly lower level at 1 hour ($p < 0.01$). Minute work decreased at 5 min from 0.301 Kg-m/kg to 0.134 Kg-m/kg and to 0.066 Kg-m/kg at 1 hour ($p < 0.01$). Mean ejection rate decreased from a control of 10.89 ml/kg/sec to 6.69 ml/kg/sec at 1 hour ($p < 0.01$). Puppies were divided into 3 groups by weight: 270-400 gm (group A), 401-800 gm (group B) and 801-1800 gm (group C). Cardiac function was comparatively more depressed in group C than in groups A and B. We conclude that 1) E. coli endotoxin has negative inotropic and chronotropic effects on the myocardium of newborn puppies, 2) depression of cardiac function is greater in the older, larger puppies.

93 EPIDEMIOLOGY OF THE HYPOPLASTIC LEFT HEART SYNDROME IN ONTARIO, 1971-73.

David H. Cook, Vera Rose, Judy Beamish (Spon. by Richard D. Rowe). The Hospital for Sick Children, Department of Paediatrics, Toronto. This study had as its purposes the description of hypoplastic left heart syndrome (HLH) in Ontario and the testing of a specific hypothesis about geographic clustering of the defect in the vicinity of Toronto. A series of 52 cases of HLH born in Ontario during 1971 to 1973 was studied, and its characteristics contrasted to those of all other infants born during those years. Cases were ascertained from death certificate information made available by the Registrar-General of Ontario. There were no false-negatives among infants known to us to have died from any congenital heart defect. False-positives among the death certificates were distributed as was the pattern of infant deaths. Autopsy rates for infant deaths in different regions of the Province are comparable. The cases clustered beyond chance expectation in the populous southern part of Ontario ($p < .02$), but also seem to be associated with some characteristic(s) of the more 'rural' parts of the Province. Birthweight in full-term infants who had HLH tended to be depressed from that of normal full-term infants ($p < .02$) when the overall distribution is considered. This series contradicted previous suggestions in that we could not demonstrate either elevated parental age or any significant seasonal pattern in dates of conception or of birth.

94 PREDICTION OF SYMPTOMATIC PATENT DUCTUS ARTERIOSUS (sPDA) FROM PERINATAL RISK FACTORS.

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In order to quantify the relationship between perinatal risk factors and the incidence of symptomatic PDA (requiring digoxin) in preterm infants, linear discriminant analysis was used to devise a predictive scoring system. Using a training set of 100 consecutively admitted infants (44 with sPDA) with a birthweight of 1500 g or less and who lived at least 72 hours, 5 factors were found that correlated significantly ($P<.01$) with subsequent incidence of sPDA: 1. any continuous distending airway pressure (CDAP) within the first 24 hours of life ($r=.71$); 2. small for gestational age (SGA) ($r=-.62$); 3. hyaline membrane disease (HMD) ($r=.62$); 4. acute intrapartum stress (AIS) ($r=.54$); 5. birthweight in grams ($r=-.52$). Based on the discriminant coefficients, a predictive score for sPDA could be derived for an infant by adding to the birthweight 466 if the infant was SGA, and subtracting 220, 456, or 104 for the occurrence of CDAP, HMD or AIS, respectively. When training set infants with a score <852 were classified as "sPDA" and >852 as "non-sPDA", 80% were classified accurately. Sequential discriminant analyses adding one factor at a time yielded improved classification with each step, substantiating the independent discriminating power of each factor. This model may help identify population differences that are thought to contribute to the variable incidence of sPDA between neonatal intensive care centers, and may prove useful in clinical management and teaching.

95 NONINVASIVE ASSESSMENT OF PATENT DUCTUS ARTERIOSUS (PDA) SHUNT MAGNITUDE WITH IMPEDANCE CARDIOGRAPHY (IC).

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When thoracic impedance (TI) is monitored continuously, as with an apnea monitor, a small negative deflection in the signal occurs with each heart beat. If this cardiac deflection is enhanced by computer averaging of TI segments synchronized with the ECG R wave, a characteristic waveform called the impedance cardiogram is produced. Since this deflection (ΔZ) may be theoretically explained by the redistribution of blood into and out of the lungs during the cardiac cycle, the technique was evaluated as a possible way to assess the magnitude of ductus flow in preterm infants with symptomatic PDA. 69% of 147 studies of 21 infants with PDA showed a ΔZ greater than 2 standard deviations above the mean of infants without PDA. Serial studies of infants who developed PDA revealed an increasing ΔZ associated with the onset of clinical shunting. During the course of ductus shunting ΔZ correlated significantly with a shunt score based on physical findings of PDA. ΔZ increased 22-31% following blood transfusion, an event likely to produce a momentary increase in ductus shunt. These data demonstrate empirically that IC may be a sensitive indicator of changes in ductus shunt magnitude as well as having objective diagnostic value. As no manipulation of the infant is required except placement of chest electrodes, the technique has the additional potential for continuous monitoring of shunt magnitude in critically ill infants.

96 MYOCARDIAL PERFORMANCE DURING REGRESSION OF LEFT VENTRICULAR HYPERTROPHY.

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We have previously shown that increased myocardial mass but not increased connective tissue returns toward normal after relief of left ventricular pressure (LVp) overload. In the present study we measured in situ LV performance in the rat 2, 4 and 8 wks after relief of supravalvar aortic constriction (SAC) of 4 wks duration. Parameters included LV and aortic pressure, cardiac index (CI), stroke power and work (SP, SW), peak flow velocity (pF1V), flow acceleration (dF/dt) and contractility indices (Vpm, Vmax). In animals relieved of SAC (dB), LVp was similar to those of control animals (C). CI in dB was significantly lower than in C at 2 and 4 wks, 97.2 ± 10.3 vs 197.9 ± 16.6 ml/min/kg, $P<0.001$, (2 wks); and 173.8 ± 14.5 vs 226.7 ± 8.2, $P<0.01$, (4 wks). By 8 wks CI in dB and C did not significantly differ, 229.0 ± 16.4 vs 212.0 ± 9.2, P=NS. Similar changes were found in SP and SW. Parameters of myocardial contractility, dP/dtmax, dF/dt, pF1V, Vpm and Vmax were not significantly different between C and dB. Animals with continued aortic constriction (B) had lower values of pF1V and dF/dt than C. LV-body wt ratios (LV/BW) in dB were less than B at 4 wks but still significantly greater than C. By 8 wks LV/BW in dB and C were not significantly different. These data suggest that alterations in ventricular performance persist for a time following complete relief of pressure overload but eventually do return toward normal. The changes which did occur were those of pump function rather than muscle contractility.