91 CROSS-SECTIONAL ECHOCARDIOGRAPHY OF ATRIAL SEPTUM IN D-TRANSPOSITION OF GREAT VESSELS

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Seven infants with d-Transposition of Great Vessels(d-TGV) were evaluated by cross-sectional echocardiography(C Echo). The ages ranged 2 days to 7 months. Five patients(pts) were examined pre- and post-atrial septostomy(AS) by balloon or surgical technique and 2 only after AS. All had cardiac catheterization(CC). The atrial septum was examined by a 60° angle mechanical sector scanner. Intact atrial septal image was shown as a thick, solid line from the upper margin of the ventricular septum ending at the posterior wall dividing two atria. The fossa ovalis showed as a concave area from left side. Prior to AS, all pts had a patent foramen ovale, but their C Echo results were that of intact atrial septum. C Echo after the AS showed a large defect. The lower and upper margins of atrial septum were visualized. The diameter of the defect was 7-12 mm. Contrast C Echo from both atria was done during CC using saline. Prior to AS, none or small amount of contrast passed the foramen ovale. After AS, large amount entered from one atrium to another freely. Cross-sectional echocardiography is important for evaluation of the size of atrial septal defect in d-TGV since the atrial level is the critical site of mixing. Its capability of quantitating the size of defect is valuable in those with previous balloon atrial septostomy.

92 DUCTUS ARTERIOSUS: DEVELOPMENTAL RESPONSE TO O_2 AND INDOMETHACIN

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It has been suggested that ineffective constriction in response to an increase in PO_2 is the primary cause for delayed closure of the ductus arteriosus in preterm infants. We studied the isometric contractile effects of increased PO_2 and indomethacin on isolated rings of lamb ductus arteriosus from animals of different gestational ages (87-150 days, term 150 days). Only rings from animals <110 days have a significantly smaller O_2-induced contraction (2.53±.3 g/mm^2, n=16) when compared with rings from animals near term (4.59±.69 g/mm^2, n=9). O_2-contracted rings from all gestational ages contract further upon addition of 1 µg/ml indomethacin. Rings from animals <110 days have a significantly larger indomethacin-induced contraction (1.10±.17 g/mm^2, n=16) than vessels near term (0.52±.12 g/mm^2, n=9). Vessels <110 days have a significantly reduced response to O_2 in comparison with those from older animals; however, inhibition of prostaglandin production in rings <110 days resulted in a total combined O_2 and indomethacin induced tension that is not significantly different from the O_2 or combined O_2 and indomethacin-induced tension developed in rings from animals near term. This is consistent with the hypothesis that, early during gestation, endogenous prostaglandins inhibit the vessel's ability to contract in response to O_2. These observations are also consistent with the ability of indomethacin to constrict the patent ductus arteriosus in preterm infants.

93 MORPHOLOGIC RESPONSES OF THE FETAL MYOCARDIUM TO PULMONARY ARTERY BANDING IN UTERO

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After banding the main pulmonary trunk at ±0.45 gestation, nine fetal lambs were studied subsequently at ±0.85 gestation (133±1.4 d). Eleven fetal lambs acted as controls (130±2.5 d). Right ventricular (RV) systolic hypertension (110±11 mmHg) was produced (systemic arterial systolic pressure 72±7 mmHg). After vital staining with horseradish peroxidase, radioautography with tritiated thymidine and fixation by coronary perfusion, RV and left ventricular (LV) free walls and interventricular septum (S) were weighed and subjected to stereologic point counting analysis with light microscopy. RV mass was greater than in the control group (10.4±.7 vs. 5.1±.4 g, $p < 0.001$). This was due to hyperplasia as evidenced by smaller or unchanged myocyte diameter (6.2±.2 vs. 6.7±.13 µm NS). LV mass was also greater than control (7.6±.7 vs. 5.5±.5 g, $p < 0.05$). This was less than the RV mass increase and was due to hypertrophy (myocyte diameter 6.0±.07 vs. 5.7±.01 µm, $p<0.001$). Increased myocyte mitosis was demonstrated by increased nuclear uptake of tritiated thymidine. S showed similar changes to LV. RV and LV capillaries were larger in diameter than control (RV 5.0±.01 vs. 4.1±.01 µm, $p < 0.001$; LV 5.3±.08 vs. 4.3±.01 µm, $p < .001$) and fewer in number/unit area (RV 44±2.7 vs. 78±4.0; LV 51±3.9 vs. 74±4.6, $p < .001$). It is suggested that the different myocardial response of the ventricles is related to differences in stress of pressure loading on the right and volume loading on the left ventricle.

94 LEFT VENTRICULAR WALL STIFFNESS IN YOUNG SPONTANEOUSLY HYPERTENSIVE RATS.

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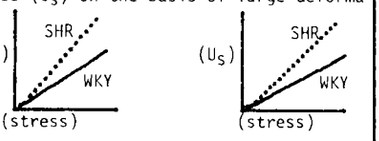
Eight week-old spontaneously hypertensive rats (SHR) and normotensive Kyoto-Wistar controls (WKY) were studied in order to determine the effect of hypertrophy on passive elastic left ventricular wall stiffness. After perfusion cooling and arrest, excised hearts were mounted on mitral and aortic cannulae and maintained in calcium-free Krebs solution (4°C). From multiple static pressure-volume cycles over a 0-10 mmHg pressure range, LV chamber distensibility ($\Delta V/\Delta P$) data were fitted to the equation $P=A+Bexp[C(\Delta V)]$. Unstressed chamber volume (V_o) and LV mass (LVM) were then determined. Assuming a spherical geometry for the LV, elastic wall stiffness moduli, (E_S) from linear elasticity theory and uniaxial stiffness (U_S) on the basis of large deformation theory, were derived from the pressure volume data. At any given stress level, LV wall stiffness is greater in the young SHR than in WKY early in the development of myocardial hypertrophy and hypertension.

	BW (g)	LVM (g)	V_o (ml)	LVM/BW
SHR (N=6)	180±9.4	0.602±.04	0.314±.02	3.34±.10
WKY (N=6)	125±7.1	0.327±.02	0.153±.02	2.60±.10
p	<0.001	<0.001	<0.001	<0.005



95 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 overload (LVPO). In this study we measured in situ LV performance in the rat 2, 4 and 8 wks after removal of an aortic band (B) producing LVPO of 4 wks duration. Parameters included LV and aortic pressure, heart rate (HR), cardiac index (CI), stroke index (SI), peak flow velocity (pFlV), and contractility indices (Vpm, Vmax). CI and SI remained significantly depressed in debanded (dB) compared with sham operated rats 4 wks after complete relief of LVPO (174±14 vs 216±8 ml/min/kg and 0.46±0.04 vs 0.61±0.02 ml/kg). By 8 wks CI and SI in dB returned to normal (200±8 ml/min/kg and 0.63±0.03 ml/kg). In continually B rats CI and SI remained normal at 4 wks but declined by 8 wks. pFlV though decreased in both B and dB at 2 wks (13.4±0.6 and 12.3±0.7 vs 16.5±0.3 ml/sec/kg), rose in dB by 4 wks (16.6±0.9) but further declined in B (12.1±1.2). Vpm and Vmax were not significantly different among the three groups. These data suggest the existence of an abnormality in myocardial function after LVPO which is apparently compensated for by the presence of myocardial hypertrophy. However, during the early regression of hypertrophy or with persistent LVPO the abnormality becomes evident. Functional recovery eventually does occur after relief of LVPO. These data further suggest the usefulness of ejectile indices of contractility in the identification of such abnormalities.

96 EFFECT OF EXERCISE TRAINING ON MYOCARDIAL FUNCTION AND HYPERTROPHY.

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Cardiac function and the development of left ventricular hypertrophy (LVH) were studied in rats preconditioned by exercise training (ET) consisting of eight weeks of running on a treadmill. At the end of the ET a group of exercised (E) and sedentary (S) rats were subjected to an in situ hemodynamic evaluation under conditions of altered afterload (AL), preload (PL) and hypoxia (HY). A group of E and S rats also underwent aortic banding or a sham operation. The only significant hemodynamic difference between E and S at rest was a slight elevation in heart rate (HR) in E. With either ↑ or ↓ preload no significant hemodynamic differences were noted between E and S. After relief of ↑AL cardiac index (CI) remained depressed in S (165±10 ml/min/kg) but returned to normal in E (214±12ml/min/kg). During recovery from HY, CI was significantly higher in E than in S (216±12 vs 170±14ml/min/kg). These differences were related to changes in stroke index (SI) rather than HR. At the end of ET there were no significant differences in body wt (237±2 vs 234±2g) or LV wt (LVW) (507±4 vs 514±9 mg) between E and S. S rats developed significant LVH at 3 days (570±18 mg) but had no hypertrophy at 1 day (542±9 mg) after aortic constriction. Exercised rats, however, developed significant LVH by 1 day (611±11mg) after pressure overload. These data suggest that the heart from an exercised animal more ably tolerates an increase in afterload and hypoxia, and can respond with compensatory LVH more rapidly than that from a sedentary animal.