CARDIOLOGY

THE FORCE FREQUENCY RELATIONSHIP: A BASIS FOR CATALOGING CHANGES IN CONTRACTILITY. Page A.W. Anderson; W. Robin Howe; Andres Manring; Edward A. Johnson. Duke Medical School, Duke Medical Center, Department of Pediatrics & Physiology, Durham, N.C. (Intr. by M.S. Spach) Supported by HL 11307, HL 5372, & N.C. Heart Association.

The force frequency relationship (FFR) describes the way cardiac contractility depends on the rate and pattern of stimulation. The FFR fulfills one of the requirements for an index of contractility: When the maximum rate of tension developed in a contraction ($\dot{F}_{max}$) is used to measure contractility, the FFR is independent of muscle length. (Circ. Res. 33:665, 1973) We show here that the FFR also fulfills another requirement for an index of contractility, namely that changes in the inotropic state of the muscle are distinguishable from changes in muscle length. Inotropic changes in the FFR fell into three classes: 1. Both ouabain ($5x10^{-7}$M) and increase in Ca concentration (up to 10mM) increased contractility following the regular contraction much more than following an extrasystolic contraction. 2. Phenylephrine $5x10^{-5}$M increased contractility following the extrasystolic contraction much more than following the regular one. 3. The actions of isoproteranol and norepinephrine in small doses $< 10^{-7}$M were similar to those in (1) but with increasing doses, $\geq 10^{-6}$M, $\dot{F}_{max}$ in the steady state contraction exceded that of the post extrasystolic contraction. We conclude that the FFR provides a valuable basis for distinguishing length changes from changes in inotropy and cataloging the actions of positive inotropic agents.

HYPOXIC DRIVE IN NEWBORNS WITH CYANOTIC CONGENITAL HEART DISEASE. Eduardo Bancalari, Lee Mockrin and Mary Jane Jesse, (Intr. by W. W. Cleveland) Department of Pediatrics, University of Miami School of Medicine, Miami, Florida

Children with cyanotic congenital heart disease have been reported to have a decreased ventilatory response to further decrease in PaO_2 (Sorensen, Edelman). When this lack of response begins has not been determined. In order to answer this question, we studied the ventilatory response to an acute decrease in FiO_2 in a warm environment in 6 newborns with severe cyanotic congenital heart disease. The following are the results: Mean basal minute ventilation ($\dot{V}E$) was 1743ml. After one minute of 15% O_2, $\dot{V}E$ dropped to 1551ml and after 3 minutes there was a further decrease to 1519ml. When 4% CO_2 was inspired for 3 minutes, $\dot{V}E$ increased to 3949ml. While breathing room air mean $PaCO_2$ was 37.2mmHg, PaO_2 19.2mmHg and pH 7.35. An important observation is that unlike the normal newborn the decrease in $\dot{V}E$ in all these infants occurred within one minute of breathing low FiO_2. The lack of hyperventilation on low FiO_2 is not explained by mechanical inefficiency because all infants increased their ventilation when breathing CO_2. The lack of hyperventilation with decreased FiO_2 and the normal basal $PaCO_2$, suggest that in these infants the abnormal ventilatory response to hypoxia is present shortly after birth, and is not related to the duration of hypoxia.

ECHOCARDIOGRAPHIC ASSESSMENT OF PATENT DUCTUS ARTERIOSUS IN PREMATURES WITH RESPIRATORY DISTRESS Barry Baylen, Richard A. Meyer, and Samuel Kaplan, Col. of Med., Univ. of Cincinnati, Children's Hosp., Dept. of Ped., Cincinnati, Ohio

Non-invasive evaluation of significant left to right shunting through a patent ductus arteriosus (PDA) is difficult in premature infants with respiratory distress. Sixteen infants (gestation 26-36 weeks; weight 780-1800 grams) were evaluated by serial echocardiography. Group I- Respiratory distress syndrome (RDS) alone (5). Group II- RDS and PDA (6). Group III- PDA alone (4). One additional baby was asymptomatic. Group I- left ventricular end diastolic (LVED) and left atrial (LAD) dimensions were within normal limits. As the RDS resolved, the dimensions remained unchanged. Group II- LVED and LAD were enlarged in 4. Dimensions in 2 treated by ductal ligation reverted to normal whereas in 2 treated medically they remained enlarged. Group III- 2 had congestive heart failure. The LVED and LAD remained enlarged in one treated medically and returned to normal in one treated surgically. Thus, volume overload of the left heart due to a patent ductus arteriosus can be detected echocardiographically in premature infants with respiratory distress. Furthermore, the early return of the LVED and LAD to normal after ligation of the ductus contrasts dramatically with persistent overload of the left heart in patients treated medically. In all groups anemia further increased LVED and LAD.

CATHETERIZATION AND PULMONARY FUNCTION STUDIES IN 10 INFANTS WITH RESPIRATORY DISTRESS SYNDROME (RDS) AND PATENT DUCTUS ARTERIOSUS (PDA). F. Blanton Bessinger, Jr., William A. Neal, Carl E. Hunt, and Russell V. Lucas, Jr.. Univ. of Minn., Dept. of Ped., Mpls., Minn.

In an attempt to define parameters of prognostic value, catheterization studies were performed in 10 infants with severe RDS & PDA who were considered candidates for closure of the PDA because of congestive heart failure, a continuous murmur, & left ventricular hypertrophy on EKG. Birth wts. were 800-1860 gms. PDA murmurs were noted at 3-21 days, & catheterizations were performed at 33-120 days of age. Angiography confirmed the PDA & ruled out other shunts. Qs, Qp, & L→R shunts were measured by cardiogreen dye curves, FA & PA mean pressures were 56 & 34 mmHg. Mean values for Qs & Qp were 1.8 & 4.4 L/min/M^2. $AaDO_2$ levels were variable. $aADCO_2$ levels >10 mmHg were associated with severe lung disease and/or pulmonary edema. 9/10 infants had ductal shunts ≥55%. One pt. had negligible L→R shunt and later died of bronchopulmonary dysplasia (BPD). Six infants underwent surgery successfully; 3 improved and 3 were unchanged (2 later died of BPD). In the 3 remaining nonoperated patients, 2 improved spontaneously & 1 died suddenly before operation. Postoperative improvement in $aADCO_2$ levels provided retrospective confirmation of benefits of ligation of the PDA in the 3 improved pts. No hemodynamic parameters correlated with results in these infants.

LEFT ANTERIOR HEMIBLOCK (LAH) AND COMPLETE REPAIR OF TETRALOGY OF FALLOT (TF). R. R. Bocala, B. Guller, G. K. Danielson and R. H. Feldt (Intro. by J. W. DuShane), Mayo Med. Sch., Rochester, Minn.

The pre- and postop ECG's of 272 consecutive survivors of repair of TF operated 1960 to 1965 were analyzed for incidence of LAH and it's possible association with significant dysrhythmia or sudden death. Mean length of follow-up was 10.2 years.

22 of 272 (8%) developed LAH after TF repair; 6 of these developed 1 to 12 years after repair. 2 of 22 reverted to normal in late follow-up. There were no significant dysrhythmias or sudden deaths in this group. 10 of 22 (45%) had transient atrio-ventricular (AV) conduction disturbances during or immediately after surgery. Anatomy, and type of repair did not differ from group as a whole. 4 additional patients developed "atypical" LAH after repair and all remained well at late follow-up.

6 patients had LAH prior to repair and 5 remained in LAH postop. All remain well.

240 patients had typical ECG postop. One died suddenly in late follow-up and another 3 cases have persistent significant AV conduction disturbance. 39 of these 240 (16%) had transient AV conduction disturbance during or shortly after surgery.

Our data show that: LAH is uncommon after TF repair, is not associated with significant dysrhythmia or sudden death and is related more frequently to transient AV conduction disturbance during or shortly after repair.

AN ALTERNATIVE EXPLANATION FOR SUPERIOR AXIS IN OSTIUM PRIMUM ASD OTHER THAN LEFT ANTERIOR HEMIBLOCK Borkon,A.M.,Varghese,P.J.,Pieroni,D.R.,Ho,C.S. and Rowe, R.D. Department of Pediatrics, Johns Hopkins Hospital, Baltimore, Maryland

Although a superior frontal plane QRS axis (SA) is considered pathognomonic of a left anterior hemiblock (LAH), the latter has not been proven to be the basis for the SA in ostium primum defects (OP). To elucidate the mechanism of SA in OP, the influence of abnormal hemodynamics (AH), ventricular hypertrophy (VH) and right bundle branch block (RBBB) on the SA was studied pre and postoperatively in 29 patients with OP. A mean SA of -75 was found with right ventricular pressure ≥40mmHg or left to right shunts >3:1 and -50 with lower values. Preoperatively mean SA was -70 in presence of VH and -30 in its absence. Postoperative resolution of VH resulted in a shift of mean SA from -70 to -30 with 3 returning to normal. With persistence of VH there was no change in the SA. Surgically induced RBBB shifted the SA more superiorly and rightward. The dependence of SA on AH, VH and RBBB suggest that LAH is not the basis for SA. In OP early activation of the posterobasal region of the left ventricle through an abnormal posterior fascicle results in minimal SA and is then exaggerated with AH, VH and RBBB. Thus SA in OP with RBBB does not represent a true bifascicular block and has a different natural history and clinical significance.