187 Q-K_d IN NEONATES: RELATIONSHIP TO PDA. Sergio O. Saia, Stanley Reisman, Frank Mannino (Spon. by Louis Gluck) University of California at San Diego, Dept. of Pediatrics, La Jolla, California.

The time interval in milliseconds (ms) from the onset of the QRS complex of the EKG to the onset of Korotkoff sounds in the arm at diastolic cuff pressure (Q-K_d) was determined with a microprocessor based instrument (NB-200 Blood Pressure Interval Analyzer; Nicolet Biomedical). The Q-K_d is dependent primarily upon the pre-ejection period & pulse transmission time. A previous study in older patients suggested a shortening of the time with PDA (Am. J. Cardiol. 19: 147, 1967). Forty-one neonates ((wt. 2148 (±835), 700-4000 g; G.A. 34.7 (±3.8), 27-42 wk)) were studied & over 300 Q-K_d determinations obtained. The standard deviation of repeat Q-K_d's in a neonate was within 7% or less of the mean of five repeat determinations. Q-K_d correlated significantly with weight (80 ms <1000 gm to 109 ms >3000 gm) corr. coef. .492, $p < .01$; $y=.0078X + 8.14$. The Q-K_d correlated significantly but less well with gestational age. Ten infants treated (Indomethacin or surgery) for a PDA were evaluated serially. There was no correlation of Q-K_d with the PDA open or closed. This negative correlation was probably due to other cardiovascular parameters such as heart failure which also effects Q-K_d. An unexpected finding was that the instrument was extremely accurate in determining systolic & diastolic blood pressure indirectly. In summary, the Q-K_d was not helpful in diagnosing patency or closure of the ductus arteriosus in neonates.

188 THE USE OF GRADED EXERCISE AND AMBULATORY MONITORING IN THE EVALUATION OF VENTRICULAR DYSRHYTHMIAS IN CHILDREN. Guillermo R. Sanchez, Anna C. O'Riordan, Richard M. Donner, Iain F.S. Black, Spon. by Angelo M. Di George Temple University Medical School, St. Christopher's Hospital for Children, Department of Pediatrics, Philadelphia, Pa., 19133

Sixteen patients 7-21 years old with ventricular extrasystoles (PVC) on previous EKG underwent graded exercise (GMX) and 24 hr. ambulatory monitoring (AM). GMX and AM were performed within 4 weeks in 12 and 3 months in 2 pts. Three pts had had surgery for tetralogy of Fallot (TOF) and 3 pts for ventricular septal defect (VSD), one pt had QT prolongation, one hypertrophic cardiomyopathy, and one had recovered from acute myocarditis. Seven pts had idiopathic PVC (IPVC). Just prior to GMX, 8 pts (I) had single unifocal PVC (UPVC), one (II) paired PVC (PPVC), one (III) a brief run of ventricular tachycardia (VT), one (IV) multifocal PVC (MPVC), and 5 (V) no PVC (including one TOF and one VSD). One pt in group I (IPVC) showed no change in PVC with GMX, and one pt in group V (VSD) developed UPVC post GMX. The pt with VSD (III) developed sustained VT post GMX. GMX suppressed PVC in the remainder. In all cases, AM confirmed the ventricular arrhythmia present during graded exercise. In addition, in the pt with PPVC, AM also documented an episode of ventricular tachycardia.

The results indicate that ambulatory monitoring is as effective as, and may be superior to, graded exercise testing in the evaluation of ventricular dysrhythmias in children.

189 NON-INVASIVE EVALUATION OF THE EFFECT OF TOLAZOLINE IN PERSISTENT FOETAL CIRCULATION. G.G.S. Sandor, A.J. McNab, V.J. Ebelt, E. Ling, M.R. Pendray, University of British Columbia, Vancouver, B.C. (Spon. by J. P. Skala)

The cardiac response to Tolazoline was carefully monitored by echocardiography (ECHO) in 6 patients who had persistent foetal circulation (PFC) and were treated with Tolazoline (T). Echo was performed just prior to T and 10 mins., 1, 4 and 24 hours after initial therapy. The mean weight was 2163 g.(range 1080-3870 g), gestational ages 28-41 wks. Two full term neonates had severe meconium aspiration and 4 premature neonates had hyaline membrane disease and asphyxia. All had labile PO_2's and were extremely difficult to manage with maximal ventilation. T was given for hypoxia or to attempt to reduce ventilation requirements. Five patients responded immediately with an increase in PO_2 of >20 torr. The other had incompletely resolved pneumothoraces.

Pre and post therapy ECHO results for ejection fraction (EF), fractional shortening (FS), velocity of circumferential fibre shortening (VCF) left ventricular (LV) and right ventricular (RV) pre-ejection period (PEP) to ejection time (ET) ratios (PEP/ET):

Mean	EF%	FS%	VCF	LV PEP	RV PEP	LV PEP/ET	RV PEP/ET
Pre	48	25	1.7	77.5	91	.53	.72
10 min.	53	27.7	1.9	65	74	.45	.55
1 hour	55	29	1.9	68	72	.47	.45
4 hours	57	31.5	2.1	58	58	.38	.38
24 hours	58	31	1.9	53	51	.32	.28

These results show marked abnormalities in left and right sided cardiac function prior to T and a very rapid improvement in right sided indices followed by the left sided indices with T.

190 PREVALENCE, SPECTRUM AND PATHOGENESIS OF EKG EVIDENCE OF CONDUCTION SYSTEM INVOLVEMENT IN DUCHENNE'S MUSCULAR DYSTROPHY (DMD). Shyamal K. Sanyal, Warren W. Johnson. St. Jude Children's Research Hospital. Memphis, TN

Twelve-lead electrocardiograms were analyzed in 50 boys, 5 to 18 years of age, with clinical, biochemical and muscle biopsy evidence of DMD. Cardiac conduction system abnormalities were noted in 27 patients (54%). Intra-atrial conduction abnormalities were most common and included: abnormal PV^1 index in 14 patients, a short P-R interval in 11 and coronary sinus rhythm in 2. Intraventricular conduction abnormalities were noted in 5 patients: right bundle branch block in 2, left anterior hemiblock in 2 and bifascicular block in 1. First-degree atrioventricular block was seen in 1 patient. Seven patients had more than one type of condution defect. Serial follow-up observations disclosed that the conduction abnormalities were progressive.

Morphology of the cardiac conduction system was studied systematically in 4 patients. In each case, within 1-2 hours of death, hearts were perfused at 4°C for four hours either with 2.5 glutaraldehyde or with 10% formalin. By contrast to age and sex-matched normal controls, DMD hearts showed multifocal areas of degenerative changes characterized by vacuolozation, fatty infiltration, and nuclear pyknosis. Other changes included variation in size and staining of myofibers, splitting and loss of myofibers and moderate-to-severe fibrosis. These dystrophic changes at the cellular level were similar in each patient and involved, with varying degrees of severity, the sino-atrial node, atrial preferential pathways, the atrioventricular node, bundle of His and subendocardial as well as intraventricular right and left bundle branches.

Our observations indicate a high prevalence of cardiac conduction abnormalities in patients with DMD and establish that the EKG abnormalities result from dystrophic involvement of the cardiac conduction system.

191 CAN WE PREDICT CORONARY ARTERY ANEURYSMS IN KAWASAKI DISEASE? Richard A. Schieber, Masato Takahashi, Alan B. Lewis, Wilbert Mason. Univ. of Southern California School of Medicine, Childrens Hospital of Los Angeles, Dept. of Pediatrics, Los Angeles, California

Kawasaki disease (KD) may cause coronary artery aneurysms (CAA) in 20% of affected children. To evaluate the relative usefulness of two-dimensional echocardiography (2DE) and an established clinical scoring system in predicting the presence of CAA, 50 consecutive patients (pts.) with KD were studied prospectively. 2DE's were analyzed for CA abnormalities. Selective coronary arteriography (SCA) was then performed in a) 4 pts. with suspicious or definitely abnormal 2DE, b) 3 pts. with high clinical score only, and c) 1 pt. each with heart failure and myocardial infarction.

The left main, and proximal segments of the anterior descending, circumflex, and right CAs were visualized in 79%, 37%, 5%, and 77% of 137 studies, respectively. Suspicious or definite lesions were identified in 38% of visualized left CA studies and 11% of right CA studies. CAA were demonstrated by SCA in 4/4 pts. with abnormal 2DE and in one pt. with myocardial infarction. However, none of the 4 pts. without definite 2DE lesions but with high clinical score or heart failure had proven CAA. The mean clinical score of 5 pts. with abnormal SCA was not significantly different from 4 pts. with normal SCA ($p > .1$).

In conclusion, the proximal CAs, excluding the circumflex artery, often can be seen by 2DE in children. 2DE appears more reliable than clinical scoring in identifying pts. with proximal CAA. SCA should be performed in all pts. with persistently abnormal 2DE or evidence of myocardial infarction.

192 PURULENT PERICARDITIS (PP)--CHANGING ETIOLOGY AND TREATMENT Eugene D. Shapiro and F.Jay Fricker (Spon. by R.H. Michaels) Univ.of Pittsburgh Sch. of Med., Children's Hospital of Pittsburgh,Dept. of Pediatrics,Pittsburgh

All cases of bacterial pericarditis admitted to Children's Hospital from 1961-1980 were reviewed. To exclude patients with sympathetic pericardial effusion, only cases with pericardial fluid that was culture or CIE positive were analyzed. The organisms that caused PP with median age for each were:

	S.aureus (SA)	H.influenzae B (HIB)	S.pneumoniae
1961-70	4	1	1
1971-80	1 (6 yrs)	6 (18 mos)	0 (4 mos)

The most common bacterial cause of PP may be shifting from SA to HIB. All children presented with fever and ST-T wave EKG abnormalities. Associated findings for children with SA and HIB were:

	Friction Rub	Tamponade	Osteomyelitis	Empyema	Pneumonia
SA	5/5	1/5	4/5	1/5	2/5
HIB	0/7	6/7	0/7	5/7	2/7

All children received antimicrobials. Four children recovered without surgical drainage and 2 recovered after pericardiectomy. Eight children were treated with pericardiotomy with tube drainage. Two of these 8, both of whom had thick pericardial pus, developed constrictive pericarditis requiring total pericardiectomy. One of the two died. The other children have not had evidence of cardiac abnormalities on follow-up evaluations. Patients with thick pericardial pus should undergo pericardiectomy not pericardiotomy.