HEART RATE PATTERNS AND RISK OF SUDDEN INFANT DEATH SYNDROME (SIDS). Peter G. Katona and Su Ling Hsu (Introduced by Marshall H. Klaus and Jerome Liebman). Depts of Biomedical Engineering and Pediatrics, Case Western Reserve University, Cleveland.

This is a first attempt to test the hypothesis that increased cardiac parasympathetic activity is linked to SIDS. ECG and respiration were recorded from 7 full term infants every 2-4 weeks in their first year while asleep at home. Neither the R-R interval nor its fluctuations during quiet sleep had a maximum when SIDS occurs most frequently (6 to 20 weeks). Average R-R interval was approximately constant at 444± 24 (SD) msec between the 1st and 5th weeks, rising to 546±33 by the 25th. Recordings were also obtained from 7 infants born preterm who were considered "near miss" SIDS cases after hospitalization with apnea of unknown etiology at ages 5 to 16 weeks; 3 were subsequently followed at home. Contrary to hypothesis, the heart rate of hospitalized infants during quiet sleep was higher than that of the controls at the corresponding ages (means 150 vs 131/min, $p<.001$); this difference disappeared within 4 to 16 weeks. During active sleep, however, each of 3 hospitalized infants who had recorded apnea (>15 sec), also had bradycardic episodes (<90 beats/min) 15 sec or longer. Bradycardic episodes observed in the control group had a maximum duration of 10 seconds. The results indicate that while high parasympathetic tone during quiet sleep is not a likely indicator of risk, prolonged bradycardic episodes during active sleep (possibly secondary to hypoxia) are likely correlates of SIDS. (Support by NIH HD 06314.)

THE FUNDAMENTAL DETERMINANTS OF FETAL CARDIAC OUTPUT. Stanley E. Kirkpatrick, MD; Jay Naliboff; James W. Covell, MD; and William F. Friedman, MD, Department of Pediatrics, University of California, School of Medicine, San Diego, California.

It has been suggested recently that the Frank-Starling mechanism may be of no importance in altering fetal cardiac output when compared to changes in heart rate (HR). The relative importance of these output determinants was evaluated in 7 chronically instrumented fetal lambs (128-136 days gestation) in whom continuous determinations were obtained of LV internal dimensions and pressures. Heart rate changes were induced by atrial pacing while resting fibre length was altered by caval occlusion and LA infusion of fetal blood. A highly significant relation was found to exist between stroke volume and LV extent of shortening (ΔD) ($r = +0.99$, $p<0.001$). A progressive decline was noted in LV output (maximum 22%) with increasing frequency of contraction from a HR of 150 ± 15 to 300 ± 5/min. despite a prominent, frequency-induced, positive inotropic effect. In contrast, when LV end diastolic diameter (LVEDD) was altered minimally at any HR, a significant augmentation was noted in ΔD at low LV end diastolic pressure (LVEDP) (3.5 mm Hg), and a 30% increase in ΔD at higher LVEDP (6 mm Hg). It is quite clear that the Frank-Starling mechanism is operative in the intact, undisturbed fetus at normal levels of filling pressure. Moreover, since frequent beat-to-beat variations were observed in ΔD, these data suggest that changing myocardial fibre length is of fundamental importance in fetal cardiovascular homeostasis.

THE VECTORCARDIOGRAM (VCG) IN CYSTIC FIBROSIS (CF). John F. Keane, Amnon Rosenthal, David H. Johnson, Charles R. Tucker, Harry Shwachman, Kon Taik Khaw, Harvard Med. Sch., Children's Hosp. Med. Ctr., Depts. of Ped. and Card., Boston, Mass.

The VCG (Frank system) as an indicator of severity of pulmonary involvement was assessed in 75 patients (pts) with CF. The Shwachman-Kulczycki clinical score (CS), PaO_2, right maximum spatial vector (RMSV) and standard pulmonary function tests were performed. The VCG horizontal loops (HL) were classified on the basis of configuration into four groups: normal, mild (increased anterior forces ± small rightward terminal forces), moderate (increased anterior forces with prominent rightward terminal forces or narrow antero-posterior loop) and severe (diminished anterior forces with marked rightward posterior loop displacement). The CS was similar in the normal and mild groups but progressively decreased in the moderate ($p<.01$) and severe ($p<.05$) groups. Mean PaO_2 for the normal group was 68 mmHg (range 54-76), mild 66 mmHg (57-83), moderate 56 mmHg (48-80) ($p<.05$) and severe 44 mmHg (30-60) ($p<.01$). Vital capacity and forced expiratory volumes were significantly lower ($p<.01$) in the moderate and severe categories when compared to the normal and mild groups. The mean RMSV in the normal group was 0.8 mv (range 0.4-1.1), mild 1.1 mv (0.7-2.2) ($p<.02$), moderate 1.2 mv (0.7-2.9) ($p<.01$) and in the severe 1.8 mv (0.8-2.7) ($p<.01$). During the 8 months the study was in progress, 7 pts died all of whom were in the moderate or severe categories. We conclude that there is a significant relationship between right heart involvement assessed by the HL of the VCG and the severity of CF determined by CS, PaO_2 and some pulmonary tests.

CHANGES IN PRESSURE WAVE TRANSMISSION ALONG HUMAN AORTA WITH AGE AND ARTERIAL DISEASE.

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Previous studies have shown that in children the amplitude of pressure waves increases progressively along the aorta and a diastolic wave appears in peripheral arteries. These alterations become less with age or arterial disease so that the wave is transmitted virtually unchanged in older patients (pts.) with arterial degenerative disease. Pressures were measured in aortic root, aortic arch, and either iliac or brachial arteries during diagnostic catheterization in 198 pts., aged 1 day to 70 yrs. There was no significant difference in amplification for pts. with normal arteries and normal aortic valves (81), aortic stenosis (48), or aortic insufficiency (9). Amplification averaged 1.56±.26 for normal pts. aged 0-19 yrs., 1.23±.19 between 20-39, and 1.17±.13 over 40. For each age range, pts. with arterial disease had a lower mean amplification, but with considerable overlap after 40 yrs. Slightly improved separation of normal and abnormal was obtained using catheter-tip manometers, Fourier analysis and amplification of individual harmonic terms. Pulsewave velocity, using foot-to-foot transmission time and measured catheter lengths from aortic arch, averaged 455±78 cm/sec in 50 normals, 464±82 in 39 with aortic stenosis, and 622±245 in 18 with arterial disease.

(Supported by NIH-HL14207-03).

CHOLESTEROL PERICARDITIS WITH EFFUSION

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In this communication we describe cholesterol pericarditis with massive effusion in a 13 year old girl who presented initially with hypertension, peripheral edema, albuminuria and hematuria. Family history was consistent with Type II hyperlipoproteinemia. On cardiac examination the heart sounds were muffled and distant. No murmur was heard. The liver was enlarged and firm. The electrocardiogram showed generalized low voltage. Chest rontgenogram revealed a large cardiac shadow. The echocardiogram suggested the presence of massive pericardial effusion. Cardiac catheterization with angiography confirmed such a diagnosis. Serum cholesterol varied from 600 to 900 mgm.% Pericardiocentesis yielded one litre of bloody fluid which did not clot on standing. The cholesterol content of the fluid was 315 mm.%. Repeated tappings were followed with rapid accumulation of the fluid. Subtotal pericardiectomy was done. The pericardium was markedly thickened and the heart was covered with a golden flaky exudate which was easily peeled off. Smear and culture of the fluid and flakes were negative for bacteria and fungi. Her blood pressure returned to normal after the pericardiectomy. The patient was placed on low cholesterol diet and cholysteramine. Cholesterol pericarditis is a rare entity and to our knowledge it has not previously been described in the pediatric age group.

INHIBITION OF CLOSURE OF THE DUCTUS ARTERIOSUS. J.B. Lakier, M.A. Heymann, A.M. Rudolph. Univ. of Calif., San Francisco, Dept. of Peds., Cardiovascular Res. Inst. San Francisco.

In many congenital cardiac lesions postnatal survival is dependent on ductus arteriosus (DA) patency. We studied a method for maintaining DA patency in lambs by formalin injection into its wall. In 10 late gestation fetal lambs, left thoracotomy was performed and 0.5-1.0 ml colored 10% formalin injected subadventitially along the ductal length; a pulmonary artery (PA) catheter was placed. 8 fetuses survived operation and delivered alive. Postnatally, catheters were placed in left ventricle, carotid and femoral arteries. Cardiac output and flow distribution were measured with radionuclide microspheres in 6 lambs. The lambs were killed 6 hrs-21 days postnatally. In normal lambs the DA is constricted within 12 hrs. 6 lambs with formalin-injected DA had mean PA pressures >60% aortic pressure and angiographic evidence of DA patency. All developed severe cardiac failure, and at autopsy had widely patent DA's. In 2 early studies the formalin injection was incomplete, and the DA was only probe patent. Histologically DA muscle showed various stages of fibrous replacement; intimal integrity was maintained. The DA was injected with formalin 6 hrs. after birth in 1 lamb which followed the same course as the 6 with large DA's. With this background we injected formalin into the DA in a newborn infant with tricuspid atresia in whom 3 surgical attempts to increase pulmonary flow were unsuccessful. Although the DA dilated and pulmonary flow increased, the infant died from pulmonary complications.

NIH Grant HL06285