187 SINUS NODE DYSFUNCTION IN CHILDREN. Steven M. Yabek, (Spon. by Robert E. Greenberg), Dept. of Pediatrics, UNM School of Medicine, Albuquerque, New Mexico.

Over a three year period, 30 pediatric patients (pts) with electrocardiographic evidence of sinus node dysfunction (SND) were identified. Their ages ranged from 3 days to 21 years. Twenty-two pts had significant heart disease. In 13, the onset of SND followed corrective cardiac surgery, and of these, 8 were operations for atrial septal defect and transposition repair. In two pts, SND followed acute myocarditis. Eight pts with SND had no obvious heart disease. Three groups of patients were identified. Ten pts had persistent bradycardia (<50 beats/min) which failed to respond appropriately to exercise or atropine administration (Group I). Sixteen pts had periods of sinus arrest with no spontaneous atrial activity for periods greater than the equivalent of two P-P intervals (Group II). Nine pts had repeated episodes of Mobitz type I or II 2° sino-atrial exit block (Group III). Four pts satisfied criteria for more than one group. Only one child had tachy-bradyarrhythmias. Seven pts with SND were symptomatic, having either syncopal (3) or near syncopal (4) episodes. Of these, three had prolonged sinus node recovery times following overdrive atrial stimulation and two had prolonged sino-atrial conduction utilizing the atrial extrastimulus technique. The three children with syncope have received permanent demand pacemakers. These data show that: a) symptomatic and asymptomatic SND is not uncommon in children; b) SND in children may result either from a failure of impulse formation or from impaired sino-atrial conduction.

DEVELOPMENTAL BIOLOGY

188 LYSOSOMAL HYDROLASES AND NEUTRAL LIPASE OF CULTURED FIBROBLASTS AND ADIPOSE TISSUES OF MONKEYS AND CHILDREN. Festus O. Adebajo, Paul M. Coates, Charles L. Hamilton and Jean A. Cortner. University of Pennsylvania School of Medicine and Children's Hospital of Phila., Pa.

Monolayer cultures of normal skin fibroblasts (NSF) from 4 children, lipid storage fibroblasts (LSF) from 2 children with lipid storage diseases - a Wolman's disease and a cholesterol ester storage disease -, a subline of "lipid-storing" Swiss mouse fibroblasts (3T3-L1), human adipose tissues (HAT) obtained at surgery from 5 normal children and monkey adipose tissues (MAT) from 13 adult monkeys (*Macaca mulatta*), were incubated at 37°C for 2 hr. in 5% CO₂ atmosphere with and without 5x10⁻⁴M isoproterenol (Iso) and 5x10⁻³M theophylline (Theo.). Neutral lipase (NL) at pH 7.0, lysosomal acid lipase (LAL), hexosaminidase (Hex) and β -galactosidase (Gal) each at pH 4.0, were assayed on the aqueous fractions of cell and tissue homogenates using 4-methylumbelliferyl compounds as substrates. Basal specific activity (n mole/min/mg prot.) of NL in HAT (3.14±0.67) was highest, being 3 to 5 times higher than the values for LSF, 3T3-L1 or MAT. Basal activity of AL was 28.41±7.06 in NSF and 17.33±7.02 in 3T3-L1 but less than 10% of these values in LSF, HAT and MAT. Basal activities of Hex and Gal. were higher in NSF, LSF and 3T3-L1 than in HAT and MAT. Stimulation with Iso. plus Theo. produced no effects on the activity of any of the enzymes studies with the exception of NL activity in HAT which increased by 50%.

189 HORMONE STIMULATION OF LIPOLYSIS IN ADIPOSE TISSUES OF NORMAL CHILDREN AND OF NORMAL AND OBSE MONKEYS. Festus O. Adebajo, Jean A. Cortner, Paul M. Coates and Charles L. Hamilton. University of Pennsylvania School of Medicine and the Children's Hospital of Phila., Pa.

Human adipose tissue (HAT) obtained at surgery from 5 normal children and monkey adipose tissue (MAT) from 9 adult monkeys (*Macaca mulatta*) - 4 obese nondiabetic and 5 lean normal - were incubated with and without 5x10⁻⁴M isoproterenol (Iso) plus 5x10⁻³M theophylline (Theo) at 37°C for 2 hr. in 5% CO₂ atmosphere. Aliquots of incubating medium, collected at 0, 1 and 2 hr., were assayed for glycerol. Basal glycerol release (n mole/min/mg prot.) was lower in lean normal - MAT (1.01±0.43) than in obese nondiabetic - MAT (3.26±1.78 p < .05). HAT released the same basal level of glycerol (2.91±0.42) as obese nondiabetic MAT. With Iso. plus Theo. the increase in glycerol production was 3 fold in obese nondiabetic MAT, 6 fold in HAT and 20 fold in the lean normal-MAT. Thus, basal lipolysis is lower in normal monkey adipose tissue than in obese monkey adipose tissue or the normal human adipose tissue, but lipolysis is more markedly stimulated by isoproterenol in the adipose tissue of the normal monkey than in the normal human adipose tissue or the adipose tissue of obese nondiabetic monkey.

190 POSTNATAL DECLINE OF SERUM ALPHA FETOPROTEIN (AFP) CONCENTRATION. Eugene Ainbender, Edwin G. Brown, Catherine M.P. Kierney and Avron Y. Sweet. Department of Pediatrics, Mount Sinai School of Medicine, New York.

The concentration of serum AFP of 35 newborn infants was measured serially during the first 3 to 12 weeks of life. In general, the concentration at birth was found to be inversely related to gestational age. However, the wide range of values makes the use of the serum AFP concentration at birth of little value in establishing the duration of gestation.

The individual curves of decline of serum AFP concentration are classified into 3 groups. Group I has a rapid regular fall with a half-life of 4.5 ± 0.8 days. The 14 infants in this group are AGA term and AGA preterm with uncomplicated postnatal courses. Group II has a slow regular fall with a half-life of 8 to 14 days. This group is made up of 2 very small AGA preterm infants with marked respiratory difficulties and 1 SGA preterm infant. Group III is characterized by serial values which decline irregularly with spikes and plateaus so that the half-life cannot be determined. Of the 18 members of this group, 1 AGA term infant had asphyxia and seizures, and 17 preterm infants had a variety of problems including SGA, sepsis, severe RDS and shock.

The curves of Groups II and III may be the result of continued postnatal AFP synthesis by the liver in response to chemical, circulatory or toxic hepatic injury. Several infants in this group had prolonged hyperbilirubinemia and 1 infant had marked cholestasis with hepatic failure.

191 EXPERTISE OF THE EXPERT MEDICAL WITNESS IN CONGENITAL MALFORMATION LAWSUITS. Robert L. Brent, Jefferson Medical College, Department of Pediatrics, Phila., Pa.

During the past 15 years I have had the opportunity to review the data from over 100 potential congenital malformation lawsuits. Many came to trial and a review of the testimony and depositions of expert witnesses has been most revealing. Distortion of the facts occurs very commonly. While over 50% of the plaintiff's expert medical witnesses (19/31) distorted the truth, this occurred in less than 10% of the defendant's expert witnesses. Just as important as the distortion of the truth is the gross evidence of lack of scholarship and objectivity. Many expert witnesses become partisans rather than objective experts. Several case histories will be presented to demonstrate the type of distortion that appears and the severity of the lack of objectivity. It is apparent that medical experts are confused about what is the proper role of a qualified medical expert. Medical schools and clinical training programs do not prepare a physician for the role of the expert witness. Consequently, notoriety and financial compensation may be more important to expert witnesses than the need to be an expert. It is suggested that improving the quality of expert medical testimony might ameliorate somewhat the malpractice crisis. This can be accomplished by providing formal education to medical students and residents about the proper role of an expert witness and by exposing to public scrutiny the testimony of medical expert witnesses.

(Supported by HD 630; EY-76-S-02-3268.A001)

192 CEREBRAL OXIDATIVE METABOLISM IN INTRA-UTERINE GROWTH RETARDATION. JD Brown, SJ Vannucci, JW Wolf & RC Vannucci, (Spon by NM Nelson). Penn State Univ Coll Med, M S Hershey Med Ctr, Dept Ped, Hershey, PA.

Ligation of the uterine arteries during late gestation leads to retarded somatic growth and compromised cerebral development in experimental animals (Wigglesworth, 1964). The pathogenesis of the altered brain growth may stem from chronic tissue hypoxia and/or substrate deficiency. Pregnant rats of 17 days gestation were subjected to laparotomy, following which the uterine artery supplying one uterine horn was ligated. The opposite horn and its blood supply were left undisturbed, its fetuses serving as controls. Dams were sacrificed at term by decapitation & fetuses delivered by C-section. Fetal brains were removed & analyzed. Animals from the ligated horn exhibited decreased body (-25%) & brain (-10%) weights compared to controls. Brain dry weight was also lower (-7.4%), but lipid content was not different. Oxidative metabolites & high-energy reserves (mM/kg w.w.) were:

	Glycogen	Glucose	Lactate	P-creatine	ATP
Control	7.76	1.77	2.72	1.56	2.30
Experimental	6.62	1.90	2.64	1.93	2.26
P	N.S.	N.S.	N.S.	N.S.	N.S.

The absence of significant differences in brain metabolites between control & experimental groups reflects adequate oxygenation of, & glucose delivery to, the brains of the growth retarded fetuses. Thus, a deficiency of substrates other than glucose appears to be the critical factor in the cerebral under development of experimental intra-uterine growth retardation.