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LOWER ESOPHAGEAL SPHINCTER PRESSURE IN ILL PRETERM INFANTS. Dean L. Antonson, Jon A. Vanderhoof, and Charles L. Paxson, Jr., Univ. of Neb. Coll. Med., Omaha, Neb. (Spon. by G.C. Rosenquist).

Nasojejunal feedings are frequently advocated for ill preterm infants on the basis of lower esophageal sphincter (LES) incompetence. We previously evaluated LES pressures in healthy term infants and found them to be indicative of sphincter competence. We have now measured LES pressures in 7 ill preterm infants receiving respiratory assistance. Pressure recordings were obtained using a single lumen side opening perfused catheter, and compared with pressures from healthy term and preterm infants (see table). The groups differed in gestational age (GA), and postnatal age (PA) but LES pressures were unaffected by these two variables. Even the smallest infant studied (720gm, pH 7.20) exhibited normal LES pressure (40 mmHg). Subsequent to studies, all infants were fed by continuous drip gastric lavage without any clinical evidence of regurgitation or aspiration.

Infants	PA (da.)	LES(mmHg)	GA(wk)
Healthy term (n=10)	2	39.4	40
Healthy preterm (n=10)	15	39.4	35
Ill Preterm (n=7)	13	35.0	33

Our data indicates that the LES is competent at an early developmental age. The use of nasojejunal instead of nasogastric feedings can not be justified on the basis of suspected LES incompetence.

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THE EFFECT OF REYE'S SYNDROME SERUM ON MITOCHONDRIAL RESPIRATION IN VITRO. June R. Aprille and Gregory K. Asimakis (Spon by John D. Crawford) Tufts Univ., Dept. Biology, Medford. MA, Harvard Medical School, Dept. Pediat. and Mass. General Hosp., Children's Service, Boston, MA.

Recently we showed that serum from Reye's Syndrome (RS) patients has an effect on the respiratory function and morphology of isolated rat liver mitochondria (mito.) suggesting the existence of a pathogenic serum factor. We now report further investigation into the biochemical mechanism of action of the serum factor. In vitro respiratory rates of isolated rat liver mito. were assessed polarographically as described previously (Science 197:908, 1977). State 4 respiration was markedly increased in the presence of RS serum as compared to control serum. To distinguish among several mechanisms for the increase in state 4 respiration, inhibitors of specific mito. functions were tested as possible antagonists of the RS effect. The inhibitors used were: oligomycin, an inhibitor of mito. ATPase, ruthenium red, which blocks mito. Ca^{++} transport; and three site-specific e^- transport chain inhibitors: rotenone (site I), antimycin A (site II), KCN (site III). In each case RS serum was added to the assay in the presence of inhibitor to see if the usual stimulation of respiratory rate could be blocked. Of the inhibitors thus tested only KCN abolished the effect of RS serum. We concluded that the putative serum factor stimulated respiration by directly or indirectly reducing components of the e^- transport chain at a point beyond phosphorylation site II. (Sup. by Chas. H. Hood Foundation)

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SEROLOGIC MARKERS OF HEPATITIS A (HAV) AND B (HBV) IN BILIARY ATRESIA (BA) AND NEONATAL HEPATITIS (NH). William Balistreri, Edward Tabor, Jacques Drucker, and Robert Gerety (Spon by P.Holtzapple) Univ. of Pa. Sch. of Med., Dept. of Peds., Phila. and Bur. of Biol.,FDA, Bethesda,Md.

Etiological speculation regarding BA and NH has implicated perinatal virus infection, however no consistent agent has been found. We sought serologic evidence of HAV or HBV infection in BA and NH by screening, at 2-6 months, 18 infant-mother pairs and 6 unpaired pts. Specific, sensitive radioimmunoassays (RIA) were used to test for HBV surface antigen (HBsAg) and antibody (anti-HBs); complement fixation for antibody to HBV core antigen (anti-HBc). Antibody to HAV (anti-HAV) was assayed by RIA, as well as the less sensitive immune adherence assay (IAHA).

	# tested	HBsAg(+)	anti-HBs(+)	anti-HBc(+)	anti-HAV(+) RIA	anti-HAV(+) IAHA
BA infants	16	0	0	0	2	0
mothers	14	0	0	0	6*	4
NH infants	8	0	0	0	3	1
mothers	4	0	0	0	2*	2

*= anti-HAV(+) found in 20-45% of USA women of childbearing age

There was no evidence of active or past HBV infection. Both BA infants with detectable anti-HAV were born to anti-HAV(+) mothers; serial testing in one revealed declining titers, suggesting transplacental transfer. Of 3 anti-HAV(+) NH infants, maternal antibody was present in one; serial titers showed disappearance by 7 mos. Maternal serum was not available in the remaining two. Thus,it is unlikely that either HAV or HBV had an etiologic role in BA or NH. A non-viral etiology or other non-A,non-B hepatitis viruses must be considered when assays become available.

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SERUM SULFATED (S) AND NONSULFATED (NS) BILE ACID (BA) CONCENTRATION VIA DUAL-BEAM SPECTROPHOTOFLUORIMETRY (DBSF). William F. Balistreri, Marcelle J. Shapiro and Roger D. Soloway (Spon. by P.G. Holtzapple) Univ. of Pennsylvania, School of Med., Depts. of Peds. and Med., Phila.,Pa

The concentration and fluctuation of BA in serum may be the most sensitive index of hepatic dysfunction. Of existing methods, gas-liquid chromatography (GC) is complex and radioimmunoassay (RIA) is limited by availability of specific antibodies. We have modified the DBSF method (Siskos, et.al.,J Lipid Res 18:666,'77) to measure NS+S BA by differences in fluorescence (f) between sample and reference cuvettes. Paired extraction with 1) isopropanol-reduces f due to protein, and with 2) ethanol/acetone-causes solvolysis, allowing enzymatic oxidation at 3α-position. Total solvolysis of standards was confirmed by TLC. Recovery of ^{14}C-taurocholate was >92%. BA concentration by DBSF correlated with GC (r=0.97) and with RIA for cholylglycine (r=0.91). Normal fasting total BA (8.6±2.5 SD μmol/l) was followed by a postprandial (90') two-fold increase due to influx via the enterohepatic circulation. No overlap with normals was found in acute (27+10.5) or chronic (79+43) hepatitis. There were no significant differences in values obtained at comparable ages in 9 patients with neonatal hepatitis (105+51) and 16 patients with biliary atresia (132+61). S comprised a varying percentage of total BA, being virtually absent (2-5%) in normals, increasing to 15-25% with severe cholestasis. CONCLUSION: A valid modified DBSF assay for S+NS, which is reproducible, rapid and easily performed on 0.1ml of serum may be a sensitive screen for liver disease.

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RECURRENT ABDOMINAL PAIN (RAP) OF CHILDHOOD DUE TO LACTOSE INTOLERANCE: A PROSPECTIVE STUDY. R.G. Barr, J.B. Watkins, and M.D. Levine. Harvard Med. School, Children's Hosp. Med. Ctr., Boston, Mass.

The role of lactose intolerance was assessed prospectively in 47 consecutive children (4.2-15 yrs: mean 9.5)presenting as outpatients with RAP. Milk ingestion and pain frequency (documented by diary)and symptom production following lactose ingestion (2gm/kg; max. 50 gm) were correlated with lactose malabsorption determined by breath hydrogen excretion (> 10 parts per million above baseline)- an accurate technique for demonstrating disaccharide malabsorption in children(Perman,JA et al, Ped. Res. 11:488, 1977). Lactose malabsorbers underwent a 3-stage elimination diet including a regular diet control period. There were no differences between lactose malabsorbers and absorbers with regard to amount of milk ingested (1.6 vs 1.7 glasses/day; p>0.5)or pain frequency (11.7 vs 8.4 episodes/week; p>0.5). Lactose malabsorption occurred in 20 children(43%), 9/34 were Caucasian, 8/11 Black and 2/2 Hispanic. Cramps or diarrhea were reported in 82% of malabsorbers and 41% of absorbers. 11 of 20 malabsorbers have completed the diet trial; in 10 of 11 pts., pain frequency was reduced (paired t test; n=8; p<0.05): 3 patients refused to continue the control diet due to symptoms. Conclusion: In RAP, lactose malabsorption is present and significantly contributes to symptoms in at least 1 in 4 pts, regardless of ethnic background. Milk ingestion, pain frequency and symptom response to lactose are unreliable indicators of lactose malabsorption. Thus, documentation of lactose malabsorption is indicated in children with recurrent abdominal pain.

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TOTAL PARENTERAL NUTRITION (TPN) CHOLESTASIS IN PREMATURE INFANTS. Ernest F. Beale, Robert M. Nelson, Richard L. Bucciarelli, William H. Donnelly, Donald V. Eitzman, University of Florida College of Medicine, Shands Teaching Hospital, Dept. of Ped. and Path., Gainesville.

Of the 221 infants admitted in 1976 weighing ≤2000 gm, 33% received TPN for periods ranging from 1 to 111 days. Of these infants receiving TPN 25% (16) developed direct hyperbilirubinemia (≥1.5 mg%) secondary to TPN cholestasis. The onset of direct hyperbilirubinemia occurred at a mean of 40 days but varied from the end of the 1st week to the 13th week of TPN, without any apparent time of peak incidence. The incidence of TPN cholestasis was 8.8% in infants receiving TPN for 10 days and increased progressively to 47% at 40 days. The highest incidence of direct hyperbilirubinemia was found in the very premature infant. The incidence in the <1000 gm group was 53%. The incidences in the 1000-1499 gm and 1500-2000 gm groups were 18% and 12% respectively. Comparing the means for birth weights, length of TPN, and protein intakes in the infants with and without elevated direct bilirubins revealed the following data:

	INCIDENCE	BW	DAYS	TOTAL gm/kg	AVERAGE gm/kg/day
Direct Bilirubin <1.5	75.4%	1332	14	31	1.9
Direct Bilirubin ≥1.5	24.6%	1098	53	115	2.2

It appears that very low birth weight infants are particularly susceptible to TPN cholestasis but this is probably due to the fact that they required TPN for longer periods of time. The length of TPN and the total amount of protein administered were the greatest risk factors in the production of TPN cholestasis.