720 EARLY FEEDING AND LIVER DISEASE IN α1-ANTITRYPSIN DEFICIENCY. John N. Udall, Marvin L. Dixon, Anna P. Newman, James A. Wright, James C. Brent, Harvard Medical School, MGH/CHMC Dept. of Pediatrics and Harvard School of Public Health, Boston, MA.

Enhanced uptake of intestinal proteases may contribute to neonatal liver disease in patients with α1-antitrypsin (α1-AT) deficiency. Breast milk, because of its antiprotease activity, may influence the expression of liver disease in these patients. To test this hypothesis, we identified 36 patients with α1-AT deficiency (33 PiZZ, 3 PiSZ). Data was obtained on the severity of liver disease and early feeding practices for 29 (21 males, 8 females) using medical records and parental questionnaires. Severe liver disease (esophageal varices or ascites) was assessed in infants breast fed (Brf) exclusively for at least the first month of life and in infants who were bottle fed (Bof) or Bof with supplemental breast feedings. Six of 11 patients with severe liver disease died.

	Total	Severe Liver Disease	Deceased
Bottle fed	19	9	5
Breast fed	10	2	1

The calculated odds ratio for severe liver disease and dying in Bof infants are 3.6 and 3.2 respectively. Even though $p > 0.1$ it appears that Bof infants with α1-AT deficiency are at an increased risk of developing severe liver disease or dying as compared to Brf children. Our findings suggest that early feeding practices are a factor in the variability of liver disease in children with α1-AT deficiency.

721 FEEDING PATTERN OF VERY LOW BIRTH WEIGHT NEONATES ≤1500 GMS. (VLBW). J. Urrutia, T. Mathew, E. Poole. (Spon. by M. G. Robinson), Medical College of Ohio, The Toledo Hospital, Dept. of Ped., Toledo, Ohio.

Previous studies of parenterally alimented neonates had established a minimum of 60 calories/kg./day and 80 calories/kg./day to achieve positive nitrogen balance and growth respectively. In order to determine how long it takes for a population of VLBW neonates to reach those parameters, 71 VLBW consecutively admitted from September 1981 to May 1982 were prospectively studied. Excluded were congenital gastrointestinal anomalies and infants reverse transported prior to completion of study. Their mean gestational age and birthweight were 30.2±2.3 wks. (range 24-37 wks.) and 1133±230 gms. (range 600-1500 gms.) respectively. 61 VLBW began enteral feeding at 11±11 days from birth (D); 60 VLBW regained BW at 18±5 days; 51 VLBW reached 1800 gm. at 48±18 days, and 43 VLBW reached 2000 gm. at 57±19 days. Days to reach different calories, protein, fat intake levels from birth were:

TOTAL CALORIES (C)			PROTEIN			FAT (INTRALIPID)		
C/Kg/D	No.	D M±SD	Gm/Kg/D	No.	D M±SD	Gm/Kg/D	No.	D M±SD
60	61	10±6	1	61	7±4	1	35	15±9
80	61	18±10	2	61	11±6	2	23	24±11
100	61	30±17	3	61	17±8	3	9	28±11
120	52	50±22						

We believe that protein intake can be supplied at a faster pace than results shown above and speculate that quicker achievement of positive nitrogen balance is possible.

722 CORRELATION OF BIRTH WEIGHT TO ANTHROPOMETRIC VARIABLES IN THE NEONATE. M. Vadapalli, A. Zubrow, C. Robinson, G. Bhatia, D. Bateman and I. Fennoy. Department of Pediatrics, Harlem Hospital Center, College of Physicians & Surgeons of Columbia University, New York, N.Y.

The weight at birth indicates the nutritional status of the neonate. Skinfold thickness has been shown to reflect fat content of the body. The aim of the present study is to assess the correlation of birth weight with limb circumference (calf upper arm) and skinfold thickness (triceps, ileac crest and chest). One hundred newborns were studied from normal nursery. The analysis of results are as follows:

STATISTICAL ANALYSIS

Variables	Coefficient	P.Value
Intercept	-1.01146	
Calf circumference	.24820	.0001*
Upper arm circumference	.13592	.0237+
Triceps skinfold	.41317	.9766
Ileac creast skinfold	.01145	.2568

*+ ($P < .05$)

The results of this data show a linear correlation between the circumference of the calf and the upper arm to the birth weight. Similar results are obtained when analyzing the data for sex and race. These circumferences can be used as another valuable parameter in assessing growth and nutrition of the baby at birth. Skinfold thickness measurements do not correlate with birth weight.

723 GASTRIC EMPTYING VS. ESOPHAGEAL FUNCTION IN GASTRO-ESOPHAGEAL REFLUX (GER). Jorge Vargas, Marvin Ament, William Berquist. UCLA School of Medicine, Dept. of Peds., LA, CA

Delayed gastric emptying is known to be a mechanism responsible for gastroesophageal reflux. Using an isotope labeled meal to measure gastric emptying, we studied 116 infants and children to determine the frequency of abnormality in gastric emptying and correlate it with the clinical presentation and other esophageal function tests.

The 5 main clinical presentations were: Recurrent Respiratory Symptoms (I), 40%; 1 Failure to Thrive (II), 31% Recurrent Vomiting (III), 75%; Apnea and Cyanosis (IV) 8%; and Severe Mental and Motor Retardation (V) 14%. The Lower Esophageal Sphincter Pressure (LESP) was significantly lower in Groups I and V ($x=19\pm10.6$ and $x=21.8\pm6$ respectively - $N=35\pm9.5$ mm Hg). The pH monitoring parameters used were the postprandial and total % of time of $pH < 4$ ($N=6.3 \pm 2.2\%$ and $6.6 \pm 1.4\%$ respectively). Groups I, III and IV showed the most significantly abnormal values.

48% of 116 showed delayed gastric emptying, exceeding the limits of isotope retention at 90 minutes (N=50% retention at 90' with solid meal and $25.2 \pm 6.07\%$ with infant formula. No significant correlation was found between esophageal motor abnormalities and delayed gastric emptying, including LESP and patterns of dysmotility of the esohageal body. Delayed gastric emptying correlated significantly with abnormal values of postprandial reflux.

The gastric emptying study should be included as a routine test in the evaluation of patients with GER.

724 FAILURE TO THRIVE AND ZINC DEPLETION IN BREAST-FED INFANTS: COMPLICATIONS OF UNPLANNED PREGNANCY IN NURSING MOTHERS. Philip A. Walravens, Clare E. Casey, K. Michael Hambidge, University of Colorado Health Sciences Center, Department of Pediatrics, Denver.

In a study of failure to thrive, 3 infants with a similar pattern of growth deviation were encountered. Weight gain ceased at 6 months in two and at 8 months in the 3rd. Breast milk was the main source of calories for all infants and refusal of milk supplements and lack of interest in solids was reported. Dietary analysis for a 6 month girl showed an average intake of 580 ml breast milk and 100 kcal daily from solids. The zinc content of breast milk was in the normal range (0.49 - 0.64 mg/l) for the stage of lactation. Total daily zinc intake approximated only 0.9 mg however, or 18% of the RDA. In a 9 month male, dietary intake consisted of frequent short bouts of nursing and 280 kcal from solids. In this infant, anorexia and poor weight gain persisted for 6 additional months until zinc supplements were provided and catch-up growth occurred. Low plasma zinc levels (57 & 67 μg/dl) were found when assayed in two of the infants. Retrospectively it was realized that the mothers became pregnant at the time the growth failure began in the infants. Unexpected pregnancy should be considered if breast-fed infants show an unexplained failure to gain weight. Zinc may furthermore cause anorexia and delay catch-up growth until supplements are provided to correct the depletion.

725 ULTRASTRUCTURAL HALLMARKS OF SOME CHOLESTATIC PEDIATRIC DISEASES. A.M. Weber, B. Tuchweber, J. Beauparlant, I Yousef, C.L. Morin and C.C. Roy. Hôpital Ste-Justine, Depts of Pediatrics, Nutrition and Pathology, University of Montreal.

In adult cholestatic liver diseases, hepatocytic microfilament accumulation has been considered as a non specific change. By contrast in experimental models (e.g. phalloidin), cholestasis has been ascribed to microfilament accumulation and dysfunction. These changes have received little attention in familial cholestatic diseases affecting children. Therefore, canalicular regions were blindly studied by two examiners (AW, PB) in 3 Byler's Like Syndrome (B) 5 North American Indian Cirrhosis (NAC) 5 Ductular Hypoplasia (DH) 3 uncorrected biliary atresia (BA) and 4 non cholestatic cryptogenic cirrhosis (CC). A rating of (0) to (+++) was applied to canalicular changes; microfilament accumulation; increase in lysosomes. Results were expressed as percent of moderate to severe anomalies (++) and (+++).

In the familial entities (B, DH, NAC) microfilament accumulation (21, 52, 42% respectively) was striking and in B paralleled bile canalicular changes (31%). Lysosomal bodies accumulation was marked in DH (60%) and NAC (43%). In BA and CC microfilaments accumulated (33, 42%) despite little changes in canaliculi (18, 14%). In conclusion, microfilament accumulation appears to precede canalicular changes in early obstruction suggesting an adaptive mechanism. In familial pediatric cholestasis both canalicular changes and microfilament accumulation are seen but ultrastructural studies do not permit to conclude about microfilament contractility.