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HYPHOSPHATEMIC RICKETS IN BREAST MILK FED PRE-MATURE. Jonelle C. Rowe, Daniel H. Wood, David W. Rowe, Anthony F. Philipps, and John R. Raye. (Spon. by Milton Markowitz) University of Connecticut Health Center, Departments of Pediatrics and Medicine, Farmington, Connecticut.

Breast milk has been the universal standard of infant nutrition; the appropriateness of this in the very small infant has been recently questioned. A case of hypophosphatemic rickets in a 595 gm premature will be presented. The infant was begun on breast milk at 12 d. of age and nourished solely on breast milk until diagnosed at 5 mos. at which time x-rays showed marked rachitic changes and fractures in the long bones. Hyperparathyroidism, Vit D deficiency, dependency and resistance, copper deficiency and inadequate Ca intake were excluded as etiologies. Inorganic phosphate content of maternal milk was normal. At time of diagnosis the pt. was hypercalcemic, hypercalcuric (3.2 gm Ca/gm Cr.), hypophosphatemia (1.6 mg/dl) and had normal alk phos. The tubular reabsorption of phosphate was > 99%.

The hypercalcemia, hypercalcuria and hypophosphatemia connected and the radiologic changes resolved with phosphate supplementation.

This is the first reported case of hypophosphatemic rickets in a premature fed breast milk. We propose it has not been seen in infants fed proprietary formula because these have from 3 to 7 times as much inorganic phosphate as breast milk. With increased emphasis placed on feeding breast milk to small pre-matures this condition may become more common unless anticipated.

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TREATMENT OF CHRONIC DIARRHEA RELATED TO FOOD SENSITIVITY WITH FOOD EXTRACT INJECTIONS. Douglas H. Sandberg. Department of Pediatrics, University of Miami School of Medicine, Miami, Florida.

Thirty children, ages 6 weeks to 7 years with persistent chronic diarrhea unresponsive to a variety of other therapeutic modalities were treated with food extract injections. Diagnosis was made by history, specific food challenges using *in vivo* C3 alteration as an indicator of sensitivity, and intradermal provocative food testing. Other possible causes were eliminated by stool and blood examinations, sweat chloride measurement, gastrointestinal radiography and when indicated, intestinal mucosal biopsy. In 13 patients there was significant failure to thrive. The number of foods tested ranged from 1 to 23. Sensitivity was demonstrated by wheal growth within a 10 minute period and or by induction of symptoms or physical reactions. All patients were found to be sensitive to one or more foods. A good to excellent therapeutic response as measured by cessation of diarrhea and in most improved growth occurred in 23 patients. In 3 patients food extract therapy was considered unsuccessful or marginally helpful; in two it was discontinued. Two patients did not return after a brief period of treatment. No serious reactions to the testing or treatment injections was observed.

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MICROBIAL BILE ACID TRANSFORMATION AND THE RECTAL MICROFLORA IN CONTROL AND CF CHILDREN. C.C. Roy, G. Delage, L. Robitaille, L. Chartrand, A. Fontaine, A. Weber and C.L. Morin, University of Montreal, Hôpital Ste-Justine, Department of Pediatrics, Montreal.

The possibility that the large amounts of bile acids found in CF stools could be related to an abnormal fecal flora giving rise to altered microbial bile acid transformation was examined in 7 CF off antibiotics (CF₁), in 10 on oral cloxacillin (CF₂) and in 6 on I.V. cloxacillin, gentamycin and carbenicillin (CF₃). Their fecal flora and bile acids were also compared to those of 7 control children. The concentration (mg/g wet stool) of bile acids in CF₁ (5.5 ± 1.0) decreased to (2.2 ± .4) in CF₂, and to (1.0 ± .4) in CF₃. A larger % of fecal bile acids in CF₁ were deconjugated and had undergone either dehydroxylation or dehydrogenation as compared to CF₂ and CF₃. Furthermore, the conjugated bile acid fraction in CF₁ showed a lower glycine/taurine ratio (3.6) than in CF₂ (8.4) and CF₃ (5.3) and a higher % of secondary forms. The anaerobic flora (log cts/g wet stool) was strikingly reduced in the CF₃ group (4.3 ± 1.0) as compared to CF₁ (9.1 ± .1) and CF₂ (9.6 ± .2). In this latter group, the total flora was larger and this was mainly accounted for by an increase in aerobic gram negatives. Both the total flora and the anaerobic flora were decreased in CF₁ when compared to controls. This was largely due to a diminution of total anaerobes. The close relationship between the anaerobic flora, bile acid concentration and microbial transformation observed in the CF groups was also present when CF₁ children were compared to controls.

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EVALUATION OF METHODS TO MONITOR INTRALIPID, Richard L. Schreiner, Melvin R. Glick, Edwin L. Gresham, Carleton D. Nordschow (Spon. by Robert L. Baehner), Indiana University School of Medicine, Indiana University Hospitals, Departments of Pediatrics and Clinical Pathology, Indianapolis.

Nephelometric measurement of light scattering index (LSI) and visual estimates of turbidity have been advocated to monitor serum Intralipid (IL) levels. Ten percent IL was diluted with either saline or serum to various concentrations (0-250 mg/dl). The LSI showed an excellent correlation with known IL standard solutions in saline or serum (R=.99) and triglyceride (TG) concentration (R=.98). One hundred fifteen blood samples were obtained from 35 patients (28 neonates) receiving continuous IL. The LSI was plotted against free fatty acids (FFA) (R=.60), cholesterol (Chol) (R=.22) and TG (R=.18). The ability of clinical personnel to grade visually the degree of turbidity was evaluated by having them assign a turbidity score of 0-4+ to 39 hematocrit tubes which contained clear, hemolyzed, or icteric serum, each of which had IL concentrations varying from 0-291 mg/dl. The 15 tubes of identical IL concentration of 99 mg/dl were graded between 0-4+ by 2 of the observers, between 1-4+ by 3 of the observers and between 0-3+, 1-3+ and 2-3+ by 1 each of the observers. This study shows that: 1) *in vitro* nephelometric LSI correlates well with IL concentrations. However, 2) correlations of nephelometric LSI with FFA, Chol and TG are poor; and 3) personnel are unable to grade turbidity by visual examination of hematocrit tubes. Therefore, infants on IL should be monitored by TG and FFA levels.

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HEMATOLOGICAL AND BIOCHEMICAL CHANGES IN PREMATURE INFANTS ON 3 COMMERCIAL INFANT FORMULAS. Nathan Rudolph, Oded Preis, Eleftherios Bitzos, and Mario R. Reale (Spon. by Charles D. Cook) Downstate Med. Ctr., S.U.N.Y. Dept. of Ped., Brooklyn, N.Y.

Thirty healthy premature infants with birth weights between 1000g and 1600g were randomly assigned at 1 wk of age to 3 groups to determine the effect of 3 commercial infant formulas (Similac 20, Similac 20 with Iron, Isonil - each containing 15 I.U. vit. E/liter) on various hematological, biochemical and growth parameters. During their hospital stay weekly determinations were made of hemoglobin (Hb); hematocrit (Hct); reticulocyte count; hydrogen peroxide hemolysis; red cell glutathione peroxidase (GSH-Px) and selenium (Se); plasma GSH-Px and Se; and serum Ca, P, alkaline phosphatase, protein, cholesterol and vit. E levels.

Mean weekly rates of declines in Hb and in Hct, respectively, did not differ significantly in the 3 groups of infants. Among the infants with the highest rates of declines in Hct and Hb were some with persistently low serum vit. E levels; these were noted in all groups. Red cell and plasma GSH-Px and Se levels did not correlate with the rate of decline in Hb or Hct levels, nor was there a direct and constant correlation between hydrogen peroxide hemolysis and the rate of Hb decline. A decline in serum phosphorus levels was noted in all 3 groups, but was significantly greater (p<.01) in the Isonil group. Under the conditions of our study, iron at the concentration used in Similac with Iron (12 mg/L) did not appear to accelerate hemolysis. Individual variability in the hematological responses was noted irrespective of the formula used.

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SUGAR HYDROLASES OF THE INFANT RAT INTESTINE AND THEIR ARRANGEMENT ON THE BRUSH BORDER MEMBRANE. Steven M. Schwarz, Peter Burrill, Philip Sunshine and Kenneth K. Tsuboi, Stanford Univ. School of Medicine, Dept. of Pediatrics, Stanford, Ca.

The brush border membrane of the immature rat intestine contains lactase and maltase as the predominant sugar hydrolases, through the suckling period of the animal. These enzymes were isolated in high purity (lactase S.A. 23, maltase S.A. 58) from infant rat intestine and monospecific antisera (rabbit) were prepared to each. Brush border membranes from 15-day old rats were isolated in excess of 25-fold purification (using lactase as a membrane marker enzyme) by procedures yielding membranes at 2 to 3-fold greater purity from infant rat intestine than reported previously (Galand and Forstner, Biochem.J.(1974)144:293).

Incubation of brush border membranes with anti-maltase and anti-lactase resulted in binding of each antibody in stoichiometric equivalence to that obtained with the respective pure enzyme. Thus, it could be concluded that each of the sugar hydrolases exist on the membrane fully exposed and equally available to antibody as is the free enzyme in solution. Furthermore, the binding of antibody to one did not affect the immuno-titration curve of the other nor its extractability by papain or Triton X-100. Each of the sugar hydrolases, therefore, is a brush border membrane surface component, which is independently placed in relation to the other and arranged so that its unique antigenic determinants are fully available for immunoreactivity. (Supported by N.I.H. grants HD-49, HD-02147, HD-00391, CA-14917.)