

† **582** SEQUESTRATION OF IRON AS FERRITIN IN MACROPHAGES IN SKELETAL MUSCLE OF VITAMIN E-DEFICIENT RABBITS. Jen-Yih Chu, Phitsamai Kanjanangulpan, Albert C. Chou, Daphne deMello and Coy D. Fitch, St. Louis University, Departments of Pediatrics, Pathology and Internal Medicine, St. Louis, MO.

Vitamin E deficiency causes iron-deficient erythropoiesis, severe myopathy and abnormal sequestration of iron in skeletal muscle. In the present study we found that both total nonheme iron (5.0 ± 1.2 vs $8.4 \pm 3.3 \mu\text{g/g}$ wet biceps femoris muscle) and soluble nonheme iron (2.1 ± 0.4 vs 4.3 ± 1.4) were increased in vitamin E-deficient rabbits. By radioimmunoassay using an antibody to rabbit liver ferritin, muscle ferritin increased from 0.47 ± 0.18 for controls to $6.34 \pm 1.70 \mu\text{g/g}$ wet weight in vitamin E-deficient animals. Parenteral iron caused large increases in total and soluble nonheme iron in non-injected muscles of vitamin E-deficient rabbits and exaggerated the difference between the two groups. Uptake of intravenously injected transferrin-bound iron by muscles of vitamin E-deficient rabbits was not increased in a short-term experiment (6 hours), but radioiron accumulated in a long-term experiment (6 days). There was no evidence of erythrocyte sequestration in skeletal muscle. An immunohistological staining method designed to detect ferritin in tissue sections failed to stain muscle from normal rabbits but intensely stained macrophages in the muscle of vitamin E-deficient rabbits. We conclude that macrophages in skeletal muscle of vitamin E-deficient rabbits take up iron from transferrin and incorporate it into ferritin, in which form it is relatively unavailable for erythropoiesis because of slow release.

583 NEPHROLITHIASIS IN CHILDHOOD INFLAMMATORY BOWEL DISEASE. Joseph H. Clark, Joseph F. Fitzgerald and Jerry M. Bergstein. (Spon. by Ira K. Brandt). Indiana University School of Medicine, James Whitcomb Riley Hospital for Children, Department of Pediatrics, Indianapolis, Indiana.

Nephrolithiasis is an infrequent complication of inflammatory bowel disease (IBD) in children. Five patients have been managed at our institution since 1978. Their mean age at stone passage was 12.2 years and mean duration of active IBD was 33 months. The majority had ulcerative colitis, passed calcium phosphate stones, and developed nephrolithiasis during an acute exacerbation of IBD. Only one patient had a previous intestinal resection; no patient had a history of urinary tract infections. We have initiated a prospective evaluation of urinary crystalloid excretion in all newly diagnosed cases of IBD. Twenty-four hour urine determinations at diagnosis in 6 patients were within the normal range for calcium (mean = 151 mg), uric acid (338 mg) and oxalate (24 mg). One month after the initiation of therapy, there was an elevation in mean urinary calcium (222 mg) and uric acid (630 mg) excretion in 4 children receiving daily prednisone and salicylazosulfapyridine (SASP). These differences were not observed in 2 patients receiving SASP alone (calcium, 142 mg; uric acid, 374 mg). The effect of prednisone on calcium excretion is well described but its role in uric acid excretion is poorly understood. Further attention must be paid to the urinary excretion of various crystalline species throughout the course of IBD to prevent the complication of nephrolithiasis.

584 GASTRIC SECRETORY MATURATION OF PRETERM INFANTS. David Clarke and Paul E. Hyman, (Spon. by Rosemary D. Leake), UCLA School of Medicine, Harbor/UCLA Medical Center, Department of Pediatrics, Torrance, California.

To establish normative values for gastric secretory function in preterm infants we studied 34 healthy preterm infants: mean gestational age 30.6 wk (27-35 wk), mean birth wt 1.5 kg (range .82-2.46 kg). Basal acid output (BAO), post-pentagastrin (6 $\mu\text{g/kg}$, sc) maximal acid output (MAO) and fasting serum gastrin (SG) were measured. Infants were studied weekly a mean of 4.6 times (range 1-8 times). Results are expressed as mean \pm SEM. At postnatal age 1 wk BAO was $12 \pm 3 \mu\text{mole/Kg}\cdot\text{hr}$; MAO was $21 \pm 4 \mu\text{mole/Kg}\cdot\text{hr}$ ($p < .01$). Achlorhydria was present in 2 of 21 infants in week 1; both secreted acid by week 2. An increase BAO to $26 \pm 5 \mu\text{mole/Kg}\cdot\text{hr}$ at 8 wk was dependent on postnatal age ($p < .05$). An increase in MAO to $53 \pm 7 \mu\text{mole/Kg}\cdot\text{hr}$ at 8 wk was dependent on postnatal age ($p < .01$) and on postconceptual age ($p < .01$). Although SG increased with postnatal age from $64 \pm 9 \text{ pg/ml}$ at 1 wk to $157 \pm 30 \text{ pg/ml}$ at 8 wk ($p < .01$), there was no correlation between SG and BAO or MAO. BAO, MAO, and SG did not correlate with weight or sex. These results indicate that in healthy preterm infants 1) a response to pentagastrin is present at 1 wk of age 2) BAO equals normal values for term infants and children 2-4 wk after birth regardless of gestational age 3) BAO, MAO and SG increase over the first 8 wk of life and 4) BAO and SG are not influenced by gestational age. In healthy preterm infants 1) postnatal factors appear to be in part responsible for maturation of acid secretory function and 2) SG is not linked to maturation of gastric secretory function.

† **585** SERUM LIPASE AS A MEASUREMENT OF PANCREATIC FUNCTION IN CYSTIC FIBROSIS. G.J. Clegghorn, P.D. Durie, G.G. Forstner, L. Benjamin & F. Dati. (Spon. by J.R. Hamilton). The Hospital for Sick Children, Toronto, Ontario, Canada & Behringwerke AG, Marburg, West Germany.

A highly sensitive and specific ELISA immunoassay capable of measuring serum pancreatic lipase (S.Lip) has been developed. We assessed the usefulness of S.Lip for monitoring pancreatic function in CF. S.Lip was measured in 133 CF pts, 86 with steatorrhea (CFPI), 47 without steatorrhea (CFPS) and 83 controls. Ages are shown in the table. * S.Lip: $\mu\text{g/L}$. (mean \pm SEM).

| Age (yrs) | n | CFPS | n | CFPI | n | CONTROL |
|-----------|----|-------------------|----|-----------------|----|----------------|
| 0-1 | 3 | 67.1 ± 19.1 | 18 | 56.4 ± 10.8 | 10 | 6.3 ± 1.1 |
| 1-3 | 3 | 109.4 ± 95.43 | 15 | 19.9 ± 3.9 | 21 | 10.7 ± 1.6 |
| 3-5 | 1 | 39.4 | 10 | 20.2 ± 6.92 | 14 | 19.1 ± 4.6 |
| 5-10 | 6 | 24.9 ± 2.8 | 14 | 4.4 ± 1.0 | 18 | 12.3 ± 1.3 |
| 10-15 | 12 | 69.9 ± 17.4 | 10 | 4.5 ± 3.0 | 14 | 17.2 ± 2.4 |
| > 15 | 22 | 62.8 ± 14.3 | 19 | 2.2 ± 1.1 | 6 | 22.1 ± 8.6 |

Our control data shows S.Lip to rise with age. In CFPI and CFPS (0-lyrs), S.Lip was significantly elevated ($P < 0.001$). S.Lip decreased with advancing age in CFPI, to values below controls and CFPS after 5 yrs ($P < 0.001$). In contrast, in CFPS there was no age related decrease, in fact, there was a significant increase compared to controls after 10 yrs ($P < 0.005$). Elevated S.Lip in older CFPS pts and CF infants may reflect ongoing pancreatic damage. Low S.Lip levels were only seen in older CF pts and accurately distinguished between CFPS and CFPI. Serial measurement of S.Lip may be useful for monitoring pancreatic function in C.F.

586 EFFECT OF METOCLOPRAMIDE ON GASTROESOPHAGEAL REFLUX (GER) IN INFANTS. Richard B. Colletti, Department of Pediatrics, University of Vermont, Burlington (Sponsored by J. D. Dickerman).

In adults metoclopramide increases lower esophageal sphincter pressure, shortens gastric emptying time, and is effective in the treatment of reflux esophagitis. To evaluate the efficacy of metoclopramide in infants we recorded esophageal pH continuously in 5 infants, ages 5-8 months, with GER. For each infant data were collected for 120 minutes after each of three sequential apple juice feedings. The first two feedings provided baseline control data. Immediately prior to the third feeding each infant received metoclopramide 0.2 mg/kg IM. After metoclopramide, the frequency of reflux episodes decreased from 10.2 to 9.8 (Not Significant, NS); the mean duration of reflux episodes increased from 4.5 to 6.9 minutes (NS); and the percent of time pH was < 4 increased from 33% to 42% (NS). None of the 5 infants had a 25% or greater reduction in any of the 3 measures. The radionuclide gastric emptying time was prolonged in 2 and normal in 1 of the 3 infants tested. Failure to demonstrate the effectiveness of metoclopramide may be due to a lack of efficacy of metoclopramide in infants, or to small sample size, insensitivity of the test method, inadequate dosage or route of drug administration.

● **587** SERUM BILE ACIDS IN THE HUMAN FETUS. C. Colombo, A. Roda and F. Sereni, Dept. of Pediatrics Univ. of Milan and Inst. of Chemistry Univ. of Bologna, Italy.

We determined the serum levels of different bile acids (BA) by RIA in 56 human fetuses and mothers. Serum was obtained immediately after legal abortion, performed between the 14th and 21st week of gestation. Conjugated cholic (CCA) and chenodeoxycholic acid (CCCA) were determined in 33 cases, conjugated lithocholic acid (CLCA) in 20 and sulfolithocholyglycine (SLCG) in 15. In fetal blood, mean concentrations of CCA ($0.8 \pm 0.4 \mu\text{Mol/L}$), CCDA ($4.5 \pm 2.7 \mu\text{Mol/L}$) and CLCA ($1.7 \pm 1.04 \mu\text{Mol/L}$) were significantly higher than those in the mother (CCA: $0.34 \pm 0.17 \mu\text{Mol/L}$, $p < 0.001$; CCDA: $0.79 \pm 0.34 \mu\text{Mol/L}$, $p < 0.001$; CLCA: $0.7 \pm 0.3 \mu\text{Mol/L}$, $p < 0.001$). SLCG serum levels were lower in fetuses ($0.15 \pm 0.09 \mu\text{Mol/L}$) than in the mothers ($0.40 \pm 0.3 \mu\text{Mol/L}$, $p < 0.01$). No correlation between levels of BA and gestational age was found.

Conclusions: 1) this study supports the concept of a state of physiologic cholestasis during development 2) placental BA transfer occurs mostly from fetus to the mother 3) there is evidence of lithocholic acid synthesis in the fetal liver via the alternate pathway for BA synthesis, with predominant formation of chenodeoxycholic acid 4) Sulfation of BA is poorly developed at this age of gestation.