● **620** THE EFFECTS OF HUMAN MILK AND LOW PROTEIN FORMULAE ON THE PROTEIN METABOLISM OF PRETERM INFANTS.

P. Pencharz, L. Farri, A. Papageorgiou. (Spon: T. Heim) The Hospital for Sick Children, Toronto; and The Jewish General Hospital, Montreal, Quebec, Canada.

The effects of pooled mature human milk and two low protein formulae (whey or casein predominant) on the rates of whole body amino nitrogen flux (Q), protein synthesis (S) and breakdown (B) were studied in 30 preterm appropriate for gestational age infants, birth weights 1500-2000 g. The infants were studied once they were receiving approximately 120 kcal/kg/d. Intakes of total nitrogen and energy were similar in all 3 groups as were nitrogen balances and length and weight growth.

Group	n	Intake	Protein	Energy	Q	S	C
			gN/kg/d	gN/kg/d	gN/kg/d	gN/kg/d	gN/kg/d
Casein	10	2.3±0.1	123±2	2.15±0.49	13.1±3.1	10.9±3.0	
Whey	10	2.4±0.1	125±5	2.04±0.52	12.4±3.2	10.3±3.2	
Human	10	1.8±0.2	123±3	2.99±0.76	18.4±4.8	16.3±4.8	

Rates of Q, S and C are all significantly increased ($p < 0.01$) in the human milk group. The only apparent explanation for this difference is the lower true protein intake ($p < 0.01$). These results suggest that preterm infants adapt metabolically to a lower protein intake by a more intense recycling of endogenous proteins and amino acids. Furthermore, the level of protein intake received by the breast fed group may approximate the lowest limit of protein requirement, as any further reduction would probably exceed the limits of metabolic adaptation.

● **621** TROPIC EFFECTS OF BOMBESIN AND SECRETIN ON NEONATAL RAT PANCREAS. Paul F. Pollack and Travis E. Solomon.

UCLA Center for the Health Sciences, Dept. of Pediatrics, and CURE-Wadsworth VA Hospital, Los Angeles. (Spon. by Cynthia T. Barrett.)

Secretin (SEC) and Bombesin (BNP) produce pancreatic hyperplasia and hypertrophy in adult rats. Their effects in newborn animals have not been studied. We investigated the effects of SEC and BNP on pancreatic growth at 2 postnatal ages. Neonatal rats were injected subcutaneously with BNP, 5 µg/kg, or SEC, 100 µg/kg, or saline, every 12 hours for 7 days beginning at 6 or 13 days of age. Pups remained with their mothers until 12 hours after their last injection, when they were weighed and sacrificed. Weight gain did not differ among groups. Pancreatic weight, total DNA and protein (PRO) were measured and are shown below as ratios of treatment group values to control group. * $p < 0.05$, ** $p < 0.01$; E=number in treatment group; +content per 100 gms. body weight; t=4.

6-13 Days:	Wt.	DNA	PRO	PRO/DNA	Wt.+	DNA+	PRO+
BNP(E=17)	1.05	1.06	1.06	0.99	1.04	1.08	1.10*
SEC(E=8)	1.19*	1.23*	1.38*	1.13	1.18*	1.29†	1.39**
13-20 Days:							
BNP(E=5)	1.29*	1.04	1.45*	1.37*	1.32**	1.06	1.49**
SEC(E=11)	1.37**	1.14	1.52**	1.34**	1.29**	1.12	1.51**

1) SEC and BNP caused significant pancreatic hypertrophy at 13-20 days. Only SEC caused hypertrophy, and possibly hyperplasia at 6-13 days. (2) Tropic effects of GI peptides may vary as a function of postnatal age. (3) BNP and SEC may play an important role in early pancreatic growth and development.

622 GIARDIASIS, MUCUS AND MALABSORPTION. A SCANNING (SEM) AND TRANSMISSION (TEM) ELECTRON MICROSCOPIC STUDY OF SMALL BOWEL MUCOSA. J. Rainer Poley, Sheila Rosenfield

and Albert W. Klein. Eastern Virginia Medical School, Departments of Pediatrics and Anatomy, Norfolk, Virginia, 23507.

Infestation with Giardia lamblia (GL) may be associated with malabsorption, the pathogenesis of which continues to be under investigation. Recent observations in 2 children with giardiasis, including careful examination of biopsied small intestinal mucosa by SEM and TEM, has provided additional information, which may help to explain presence or absence of malabsorption. Two children, aged 2 and 2½ years with diarrhea of 3 to 4 months' duration underwent diagnostic workup, including small bowel biopsy. Stool examinations were negative, and serum immunoglobulins, and mucosal morphology by light microscopy normal in both. Biochemical indices indicated malabsorption in one of the children, but not in the other. SEM of the mucosa of the child with malabsorption showed that wide areas of villous surfaces were covered with sheets of mucus of variable thickness (1.5-4.0 µm). The microvilli were shortened. By contrast, the villous surfaces of the child without malabsorption were free of mucus, and most GL were trapped in mucus at the base of the villi. Mucosal invasion was not a major finding. These observations suggest that increased secretion (crypts) and deposition of mucus produces an effective diffusion barrier to nutrients, explaining malabsorption phenomena and, possibly, subsequent adverse trophic effects on the mucosa. The proclivity to secrete mucus in response to the infestation with GL in humans may be determined genetically and/or environmentally.

623 FLUCTUATIONS IN THE RATE OF IV FLUID ADMINISTRATION TO VERY LOW BIRTH WEIGHT (VLBW) INFANTS. Tonse N.K. Raju,

Elizabeth Chow-Tung, Dharmapuri Vidyasagar. University of Illinois, Department of Pediatrics, Chicago, Illinois.

Rapid blood volume expansion in VLBW infants has been suggested as a cause of ICH. We calculated, prospectively, hourly fluctuation in the rate (ml/kg/day) of fluid actually received in 10 acutely ill, VLBW (B.Wt. & Gest. Age: 1.07±0.07 kg and 30±0.7 wks), during the first 72 hours. Volume actually received were noted hourly, without nurses' knowledge. Fluctuations from ordered rate were calculated for 614 hours. Mean IV fluid ordered and actually received did not differ, however, wide hourly fluctuations in the rate occurred in all infants. Table gives % of time fluctuation in rate of fluid actually received (expressed as ml/kg/day) occurring in the study infants. During 614 hours only 4% of time infants got

Fluct. rate	±1-10 ml	±11-30 ml	±31-60 ml	±61-90 ml	±90 ml
ml/kg/day					
% time:	23.5%	31.2%	25.5%	10.3%	6%

ordered amount at ordered rate. 25% of time hourly fluctuations were >50 ml/kg/day. Excess and deficit rates were equal, thus total volume received was equal to ordered amount. Rapid rates >80 ml/kg occurred more often in ICH infants (Stat. N.S.). Fluctuations were due to voltage variation (10%), transfusion (10%), infiltrations of IV and the 'adjustments' nurses make to make up ordered volume at the end of 8 hour shift. We conclude: a) Both fluid volume and rate actually received by the infant should be checked hourly in VLBW infants; b) These fluctuations may be responsible for hypo or hyperglycemia, appearance and disappearance of PDA murmurs and possibly ICH in VLBW infants.

624 SERUM GASTRIN RESPONSE TO INTRAGASTRIC PROTEIN AND CYSTEINE IN NEONATAL SWINE. Bradley M. Rodgers,

Kenneth D. Blake and Farhat Moazam. University of Florida College of Medicine, Shands Teaching Hospital, Departments of Surgery and Pediatrics, Gainesville, Florida.

Previous work has shown a decreased gastrin release in neonatal humans and swine in response to intragastric protein. To further study gastrin release in neonatal swine, serum gastrin determinations were made following intragastric instillation of protein and the amino acid cysteine, known to release antral gastrin in adult animals. Six animals, less than 48 hours of age, had catheters placed in the femoral and portal veins. In three animals, intragastric Sustagen (1.7 gm protein/kg) was followed by intragastric cysteine (100mM, pH 7.4 at 10 cc/kg), while three received cysteine. Simultaneous femoral and portal venous samples were analyzed for gastrin at 1, 5, 15, 30, 45 and 60 minutes. The simultaneous femoral and portal vein gastrins did not differ statistically at any time. Basal gastrin was elevated in all animals when compared to adult values ($p < 0.1$). Following intragastric cysteine infusion, there was no significant elevation in gastrin in the portal (317 vs 264 pg/ml) or femoral (253 vs 254 pg/ml) veins. After protein challenge with Sustagen, there was a delayed, but significant, elevation in gastrin in portal and femoral samples at 30, 45 and 60 minutes (386 vs 573 pg/ml P.V. and 411 vs 569 pg/ml F.V.) ($p < 0.5$). The results of these studies indicate a significant alteration in the release of gastrin in response to intragastric amino acids in the neonatal swine when compared to adult animals.

625 RICKETS IN CHILDREN WITH CHOLESTATIC LIVER DISEASE: EVALUATION AND TREATMENT. C.C. Roberts, L.S. Book,

G.M. Chan, and M.E. Matlak (Spon. by J.J. Herbst). Univ. of Utah, Dept. of Peds. and Surg., Salt Lake City, UT

Rickets, often leading to fractures and bone deformities, occurs in children with cholestatic liver disease (CLD) because of decreased intestinal absorption of minerals and impaired hepatic hydroxylation of vitamin D. The purpose of our investigation was to determine the frequency of rickets and its response to 1,25 (OH)₂ vitamin D therapy in 18 children with CLD, ages 2 months to 5 years, over a two-year period. Serial measurements of bone mineral content were obtained using photon absorptiometry of the wrist and serum values of 25-OH vitamin D and bilirubin measured on CLD patients receiving 400-1200 IU vitamin D by mouth. In 3 of 18 children bone mineral content (BMC) was normal for age. All had direct serum bilirubin <2.0 mg/dl and normal serum 25-OH D (N=10-40 ng/ml). In 15 of 18 children BMC was >2 SD below the mean BMC of normal age-matched controls; serum 25-OH vitamin D was also low (<10 ng/ml). Once rickets was diagnosed, 12 patients received .05 to .1 µg/kg/day 1,25 (OH)₂ vitamin D. Eight of 12 children treated with 1,25 (OH)₂ vitamin D had improvement of bone disease indicated by a doubling of the BMC and an increase in the BMC/body-weight ratio to the normal range. Conclusion: Metabolic bone disease is common in children with CLD. Photon absorptiometry is a simple and accurate technique for identifying and monitoring children with hepatic rickets. In children with CLD oral 1,25 (OH)₂ vitamin D may be effective for the treatment of hepatic rickets.