607 NUTRIENT CHANGES IN CRITICALLY ILL CHILDREN ON TOTAL PARENTERAL NUTRITION: Festus O. Adebonojo, Department of Pediatrics, Meharry Medical College, Nashville Tennessee.

In the critical care of children it is increasingly necessary to use total parenteral nutrition. Daily intakes of fluids, calories (amino acids, dextrose, fat), minerals and vitamins were prospectively monitored in 17 sick children aged 6 mos. to 14 years during 24 episodes of total parenteral nutrition (TPN). Essential laboratory observations were also prospectively made. Twelve children had one, 3 had two and 2 had three episodes of TPN administrations. Duration of TPN for children weighing less than 10 kg was 13±3 days (mean ±SD) and for those over 10 kg, it was 10±2 days (mean ± SD). To maintain normal serum electrolytes, mean daily intakes for most minerals remained constant throughout TPN administration, except for sodium and chloride where the mean daily requirements decreased from 4.0 and 4.4 meq/kg respectively to 2.6 and 2.8 meq/kg respectively at 10-12 days. Moderate elevations in values of SGOT/SGPT were observed in most patients between 7 and 10 days and LDH and CPK between 17 to 20 days of treatment. Significant biphasic elevations in blood sugar was noted at days 5 to 7 and days 18 to 21, when the mean blood sugar rose from 100 mg/dl to 147 mg/dl and 132 mg/dl respectively. The mean daily requirements for fluids, calories and vitamins did not change significantly once optimal intakes have been achieved. In 3 children, serum levels of magnesium, zinc and copper were carefully monitored and no changes were found.

● 608 CRURAL DIAPHRAGMATIC CONTRACTION: A COMPONENT OF THE GASTROESOPHAGEAL (GE) ANTI-REFLUX MECHANISM. Steven M. Altschuler, Todd E. Nixon, John T. Boyle. (Sponsored by John B. Watkins). Univ. PA School of Med., Children's Hosp. of Phila., Div. of Gastroenterology, Phila.

Previous studies in our lab have shown that the crural diaphragm contributes to the high pressure zone (HPZ) at the GE junction and relaxes in response to swallowing. The purpose of this study was to determine if crural contraction affects the integrity of the antireflux barrier. Anesthetized cats breathed spontaneously following bilateral cervical phrenicectomy. The effect of increasing electrical stimulation of crural diaphragm using surgically placed bipolar electrodes was measured on a) HPZ pressure measured by a perfused intraluminal manometric assembly and b) gastric volume and pressure required to induce a drop in intra-esophageal pH following rapid gastric perfusion of HCl (pH=4) retrograde through a duodenostomy tube.

RESULTS in 10 cats:

Crural Voltage (v)	0	4	8	16
HPZ (mmHg)	30 ± 2	36 ± 4	47 ± 5**	62 ± 6**
Gastric Vol (ml)	126 ± 14	132 ± 16	164 ± 21*	197 ± 30*
Gastric Press (mmHg)	19 ± 2	19 ± 1	22 ± 2*	29 ± 3*

± SEM (*p <.05, **p <.01 compared to 0 crural voltage)

CONCLUSIONS: (1) Electrical stimulation of the crural diaphragm results in a voltage dependent increase in HPZ pressure. (2) A significant voltage dependent increase in gastric volume and pressure is required to induce gastroesophageal reflux during the electrical stimulation of the crural diaphragm. These results suggest a role for active crural contraction in the anti-reflux barrier at the GE junction.

609 INTERNAL STANDARD (PEG) FOR BALANCE STUDIES IN PREMATURES. Vanessa Z. Ameen, Geraldine K. Powell, Univ. of Texas Med Branch, Dept. of Pediatrics, Galveston.

Studies of absorptive capabilities of prematures have been limited by incomplete stool collections where loss of even small portions of the usual 5-10 gm stool output/day introduces a large percent error. We evaluated the usefulness of a continuous oral internal marker (PEG) to correct for stool losses and variations in fecal flow in 4 prematures(Av. wt 1.7 kg). Total formula and PEG intake were monitored and 24-hour stool collections done using plastic-lined diapers. Formula and stools were analyzed for PEG and carbohydrate(CHO) content utilizing methods that yielded 95% recovery of PEG and CHO.

PEG	Stool wt(gm)/day				PEG recov		CHO(mg)/day			
(mg)	uncorr	V	corr	V	(%/day)	V	uncorr	V	corr	
50	11.9± 4.0	34	17.6±3.8	22	50± 9	18	95±50	53	252±70	28
100	8.5± 5.9	69	26.6±4.8	18	52± 24	46	115±70	61	267±70	26
100	8.3± 7.0	84	10.3±3.0	29	74± 61	82	65±44	68	116±90	78
100	15.1±14.7	97	14.5±2.5	17	108±104	96	30±30	100	24±12	50

Conclusions: 1) PEG output/gm stool became constant(Av. variability 15%) by 3-5 days(vs 2 wks in adults). 2) There was wide variability in daily stool wt (0-32.6 gm) and %PEG recov(0-217%) but these correlated strongly(r=.92,p<.001). %PEG recov did not vary with the dose adm. 3) Therefore stool wt and CHO excretion per day could be corrected by %PEG recov, decreasing their coefficient of variation(V).

Our results confirm previous concerns about the adequacy of stool collections and suggest an internal marker may be feasible for correction in premature balance studies.

610 MATERNAL DIETARY HISTORY AS INDEX OF FETAL VITAMIN D STATUS. D Anderson, B Hollis, B Levine, W Pittard. CWRU, Dept Nutrition & Peds, Cleve, OH.

We have reported that the serum concentration of vit D_2 contributes significantly to total vit D status of pregnant women at term and that fetomaternal vit D status at term is intimately related. Therefore, to determine the accuracy of predicting fetal vit D status from a history of maternal vit D intake, we elected to determine the correlation between maternal dietary vit D intake and fetomaternal 25OH vit D_2/D_3 status at term. Pregnancy diet histories were obtained within 48 hrs following uncomplicated full term deliveries from 17 women(16 black,1 white). Daily vit D intake was determined with a computer data base using the recalled consumption of vit D containing foods and drugs. Plasma from mother-infant diads collected at delivery had 25OH vit D_2/D_3 extracted, chromatographed and quantitated using competitive protein binding assays. These values were then correlated with maternal vit D intake. All mother-infant diads were vit D sufficient. The correlation between the mother's recalled total D intake and either maternal or fetal $25OHD_2/D_3$ and total 25 OHD at term was not significant with r values <.3,p=NS. This striking absence of correlation demonstrates that accepted dietary history gathering techniques are unacceptable methods of assessing maternal vit D status at term and cannot be used to anticipate fetal vit D status at delivery.

Dietary[a]	Maternal[a]		Cord Blood[a]	
Vit D(IU)	25-OH-D_2[b]	25-OH-D_3[b]	25-OH-D_2[b]	25-OH-D_3[b]
833±349	6±5	8±3	4±3	4±2

a=Mean±SD b=ng/ml

● 611 DISTINCTIVE HEPATOPATHY IN LOW BIRTHWEIGHT INFANTS IN ASSOCIATION WITH E-FEROL® INFUSION. W.F. Balistreri, K.E. Bove, Children's Hosp. Research Foundation, Cincinnati; N. Kosmetatos, K.E. Wedig, D.J. Frank, Good Sam. Hosp., Cinti.; C. Bodenstein, Sacred Heart Hosp., Spokane; J. Haas, Child. Ortho. Hosp., Seattle; V. Saldivar, Santa Rosa Hosp., San Antonio.

We obtained autopsy-derived tissue from 16 of 38 fatalities reported in infants who had received I.V. vitamin E. All infants were preterm (B. Wt = 580 to 1500 gms), born between 10/83 and 3/84, who died at 1-12 wks of age. Each had received E-FEROL®, which contains 25 U/mL of dl-α-tocopheryl acetate solubilized with Polysorbate 80 (9%) and 20 (1%), in a reported dose of 25-100 U/Kg/d for 6 to 44 days (total dose = 576-3100 U). The clinical course was one of progressive deterioration, characterized sequentially by unexplained thrombocytopenia, uremia/oliguria and cholestasis. The histologic features reflected progressive injury: Kupffer cell exfoliation, hepatocytolysis, and sinusoidal dilatation with accumulation of cellular debris and free-floating cells (< 1 wk of infusion); attenuation of liver cell plates with extreme panlobular congestion (1-2 wks); cholestasis, early intralobular fibrosis (2-3 wks); and ultimately marked fibrosis with sinusoidal obliteration. There was no regression apparent after discontinuation. This distinctive hepatopathy is not consistent with shock; other forms of venous occlusion were not found. For one nursery, it was possible to track all 36 infants who had received the preparation:

(All values = group mean)		B. Wt	Total Dose	Dose/Kg/d	Duration
Definite cases	(n=3)	1042g	1238 U	37.1 U	32.3 d
Possible "	(n=6)	1008g	460 U	34.3 U	15.3 d
Not affected	(n=27)	2136g	318 U	16.9 U	9.9 d

Conclusion: We believe that this unique hepatopathy represents the cumulative toxic effect of the constituents of E-FEROL®.

● 612 DIFFERENCES IN PROCESSING OF ORALLY ADMINISTERED PROSTAGLANDIN $F_{2\alpha}$ IN THE GASTROINTESTINAL TRACT AND LIVER OF SUCKLING AND WEANLING RATS. Alan D. Bedrick, and Otakar Koldovský. University of Arizona College of Medicine, Departments of Pediatrics and Physiology, Tucson, Arizona 85724.

The presence of prostaglandin $F_{2\alpha}$ (PGF) in milk led us to explore developmental differences in its metabolism between suckling and weanling rats. Disposition of orally administered ^{3}H-PGF (20 μCi/kg/b.w.) to 12-day-old suckling (Su) and 30-day-old weanling (We) rats was evaluated quantitatively by determination of total radioactivity (TRA) recovery; and qualitatively by column and thin layer chromatography of extracts of liver (L), gastric wall and lumen (G), and small intestinal wall and lumen (I) two hours after administration. Recovery of TRA in these organs was 32.6% ± 2.1,8 (mean ± SE, N) of counts administered in Su, and 22.7% ± 4.8,5 in We ($p < 0.03$). In Su, G values tended to be higher than in We (11.1% ± 1.3,8 vs 8.5% ± 3.2,5); I values were 15.9% ± 0.7 vs 10.3% ± 1.8 ($p < 0.01$). Despite the presence of higher nonabsorbed TRA in G and I of Su, increased TRA was present in L [9.1% ± 1.7 vs 3.4% ± 0.6 ($p < 0.05$)]. Qualitatively, there was no substantial difference in distribution of TRA (unmetabolized parent PGF vs metabolites) in G and I. However, L of Su when compared to L of We, exhibited more unmetabolized PGF (12% ± 0.9 vs 7% ± 1.1 of TRA in L ($p < 0.01$) and less of the polar metabolites (21% ± 3.7 vs 35% ± 3.1 ($p < 0.02$).

Conclusions. Qualitative and quantitative differences in metabolism of orally administered PGF were found between suckling and weanling rats. Despite slower absorption of PGF from the gastrointestinal tract of suckling rats, a larger proportion of prostaglandin was found in the liver in an unmetabolized form.