656 LIVER MALIGNANCY FOLLOWING PROLONGED PARENTERAL NUTRITION (PN). Rita Vileisis, Kelly Sorensen, Frank Crussi and Carl E. Hunt. Northwestern University and Children's Memorial Hospital, Dept. of Ped. and Path., Chicago.

Cholestatic jaundice (ChJ) is a well known and presumably benign complication of prolonged PN. We now report a preterm infant in whom fatal hepatocellular carcinoma (CA) occurred as a consequence of biliary cirrhosis secondary to prolonged PN and PN-associated ChJ. The patient was a 900 gram, 27-week gestation infant. Recurrent episodes of necrotizing enterocolitis led to small bowel resection and resultant short gut syndrome, necessitating PN for a total of 395 days. During this time progressive liver dysfunction developed, with marked conjugated hyperbilirubinemia (maximum 11.2 mg/dl), and increased transaminase levels. Liver biopsy after 112 days of PN revealed periportal fibrosis and intracellular cholestasis. All liver function tests were normal when discharged at age 19 months. He was readmitted six months later with fever, hepatomegaly, icterus, and melanic stools, and expired after the onset of massive GI hemorrhage. At autopsy, two separate hepatic tumor masses were found, one of which had invaded the posterior wall of the duodenum. Microscopically, the histology was that of hepatocellular CA; the tumor was composed of neoplastic hepatocytes arranged in trabeculae or lying in non-cohesive masses, with definite canicular bile stasis and no hematopoietic tissue or mesenchymal components. All non-tumor areas of the liver showed biliary cirrhosis. In summary, this patient demonstrates the oncogenic potential of biliary cirrhosis complicating PN-associated liver dysfunction.

657 ZINC SUPPLEMENTATION OF LOW INCOME PRESCHOOL CHILDREN: EFFECT ON GROWTH. Philip A. Walravens, Nancy F. Krebs, K. Michael Hambidge, Univ. Colo. Health Sciences Center and Dept. of Health and Hospitals, Dept. of Pediatrics, Denver.

Many preschool children from low-income, Mexican-American families have low growth percentiles. Biochemical evidence for inadequate Zn nutrition was found in a group of such children in Denver. The effects of Zn supplementation on growth velocity and indices of Zn nutrition were examined in a double-blind pair-matched controlled study.

Children, 3-6 years of age, were screened, with parental consent if height and weight were at or below the 10th percentile. Inclusion in the study required an additional 2 of the following: hair Zn <100 µg/g, plasma Zn <68 µg/dl, urine Zn <100 µg/24hrs or dietary Zn intake <2/3 of RDA (i.e. <6mg/day). Participating children were assigned to take daily for 1 year either 10 mg of Zn as the sulfate in cherry syrup or a placebo syrup. Growth velocities were calculated from measurements obtained quarterly.

Height increments in cm/year (mean ± S.D.)

	Control	Zn Supplement	p value
Combined Sexes (13)	5.99 ± 0.64	(12) 6.68 ± 0.80	<0.025
Males (9)	5.87 ± 0.33	(7) 6.39 ± 0.60	<0.05
Females (4)	6.24 ± 1.09	(5) 7.09 ± 0.93	N.S.

Mean weight gains of the test and control children were 2.33 ± 1.22 and 1.69 ± 0.65 kg/yr respectively (p<0.10). Dietary intakes increased in the test group. This study shows a beneficial effect of Zn supplementation in a population where marginal Zn deficiency may affect appetite and growth velocity.

658 METABOLIC CONSEQUENCES OF CONTINUOUS AND SEQUENTIAL REGIMENS OF TOTAL PARENTERAL NUTRITION (TPN) IN INFANTS. Michael F. Whitfield (Spon by Paul R. Swyer) University of Sheffield, Sheffield Childrens Hospital

Department of Paediatrics, Sheffield, England.

Sequential blood levels of glucose, alanine, glycerol (gly) lactate, pyruvate, 3-hydroxybutyrate (3HOB) Acetoacetate (AcOAc) and insulin were measured in 16 infants (mean birth weight 2.81 kg, mean gestational age 37.9 weeks) receiving one of 2 regimens of TPN, delivering identical constituents (amino acids (Vamin) 2.8 gm/kg/day, fat (Intralipid) 4.8 gm/kg/day, glucose 12 gm/kg/day). 7 infants who received the nutrients as a continuous infusion of constant composition (continuous regimen, CR) had blood metabolite levels comparable to those of infants fed enterally except for elevation of insulin level (median 20, range 2.5-80 µU/ml). 9 infants receiving the total daily dose of Vamin & intralipid together over 10 hours followed by 10% glucose for 14 hours (sequential regimen, SR) had peak (median, range) blood levels (µM/l) of glycerol (210; 17-259), 3HOB (2084, 129-2692) and AcOAc (195; 9-429) respectively 1.8, 7 and 6 times greater than coincident levels in infants receiving CR. Blood levels of glycerol (p<0.05), 3HOB (p<0.02) and AcOAc (p<0.01) were significantly more unstable with SR. 4 infants in each group had clinically unsuspected hypoglycaemia (<30 mg%). Cholestatic jaundice occurred in 7 of 9 and 3 of 5 infants receiving SR and CR respectively. Wide variations in substrate infusion rate and metabolite levels with SR may have been a factor in the higher incidence of jaundice.

659 POOR WEIGHT GAIN IN JEJUNALLY FED LOW BIRTHWEIGHT (LBW) INFANTS. Michael F. Whitfield (Spon. by Paul R. Swyer) University of Sheffield, Jessop Hospital for Women. Department of Paediatrics, Sheffield, England.

Several studies have suggested that LBW infants fed by the nasogastric (NJ) route gain weight more rapidly than those fed nasogastrically (NG), despite greater fat malabsorption during NJ feeding.* 40 appropriately grown LBW infants (mean birth weight 1.29 kg, range 1.04-1.49 kg; mean gestational age 29.6 weeks) were allocated to continuous NJ or intermittent NG feeding at birth and their growth followed till 6 months after expected date of delivery (EDD+6 months). Infants in the NJ group were transferred to NG feeding when they reached 1.5 kg body-weight. Although mean (±SD) enteral caloric intake in the two groups was comparable (NJ 121.3±11.0; NG 121.7±9.6 cal/kg/day) mean (±SD) weight velocity during NJ feeding (11.4±3.6) was less than during NG feeding (15.8±3.5 gm/kg/day, p=0.02), and bodyweight at EDD was lower in the NJ group (mean weight index ±SD; NJ group 0.89±0.04; NG group 0.92±0.04; p<0.05). After transfer to NG feeding, infants in the NJ group grew more rapidly from EDD to EDD+3 months (mean weight velocity ±SD; NJ group 7.69±1.57, NG group 6.49±1.59 gm/kg/day; p<0.05) so that at EDD+3 months and EDD+6 months there was no significant difference in bodyweight between the two groups. Because of poorer weight gain during NJ feeding, LBW infants fed by this route from birth should be transferred from NJ feeding as soon as an adequate enteral caloric intake can be tolerated by the NG route. *Roy et al (1977) J Peds 90:431.

660 MILD LEAD POISONING IS STRONGLY ASSOCIATED WITH IRON DEFICIENCY. Ray Yip, Thomas N. Norris, Arnold S. Anderson. (Spon. by Peter R. Dallman). Children's Health Center of Minneapolis, Minneapolis.

Anemia is often found in children with severe or symptomatic lead poisoning and is attributed to the inhibition of heme synthesis by lead. During a one year period of using Erythrocyte Protoporphyrin (EP) as a screening test in an urban pediatric clinic, 191 children had elevated EP (> 35 µg/dl whole blood) and had further evaluation of lead and iron status. 43 children had a confirmed elevation of blood lead (Pb > 30 µg/dl), 33 (77%) with EP > 50 fulfilling the diagnosis of undue lead absorption, a relative mild form of lead poisoning. Of the 43 children with evidence of mild lead poisoning, 38 (86%) also had evidence of iron deficiency based on ferritin < 15 ng/ml and/or transferrin saturation < 16%; 13 (30%) were anemic. This is significantly higher than the rate of iron deficiency of 9% and rate of anemia of 4% in the same general population. This strong association of mild lead poisoning with iron deficiency is in accord with the observation of increased lead absorption in iron deficiency. It is likely that iron deficiency strongly predisposes to lead poisoning and explains, at least in part, the high prevalence of anemia associated with lead poisoning.

661 IS TOTAL PARENTERAL NUTRITION (TPN) CHOLESTATIC? I. Zahavi, E.A. Shaffer and D.G. Gall. GI Res. Unit, Faculty of Medicine, Univ. of Calgary, Calgary, Alta.

TPN therapy has been associated with cholestasis though the cause is unknown. The effect of TPN on bile formation was evaluated in 10 studies performed in three adult dogs. The bile duct was directly cannulated via Thomas cannulas, while bile salt secretion was maintained by IV taurocholic acid infusion. After a 2 h basal period, either an amino acid solution (2.5% Travasol) with 10% glucose or a lipid emulsion (Intralipid) was added for 2 h followed by a second control period. Amino acid-glucose (AA) resulted in a significant increase in bile flow (BF) which rose 33% while bile salt (BS) secretion increased transiently (11%) then returned to basal levels (Table). Cholesterol, phospholipid and bilirubin output did not change significantly.

	Basal	1	AA (h)	Post AA
			2	
BF (ml/min)	0.24±0.02	0.28±0.02*	0.32±0.03*	0.29±0.03
BS (µM/min)	48.4±3.0	53.8±2.6*	50.3±1.5	45.5±2.6

*p < 0.05

Intralipid had no effect on bile flow or solute secretion. Thus, TPN therapy is not cholestatic but rather produces a cholerisis which is largely independent of bile salts. The temporary increase in bile salt secretion without any change in lipid composition suggests a washout effect from the biliary tree.