GALACTOSE METABOLISM BY ISOLATED PERFUSED LIVERS OF WEANLING RATS. Wallace Berman, Shirley Rogers, Jesusa Bautista and Stanton Segal. Univ. of Penna. Children's Hospital of Phila., Phila., Pa. 19146

The liver is both the principal organ for galactose disposition and also a target of galactose induced toxicity. In the transferase deficient galactosemic individual the neonatal period is the most significant and hazardous. For these reasons we have undertaken studies of galactose metabolism and its consequences in the isolated perfused liver of the weanling rats. In a closed recirculating system using the Mortimore perfusion apparatus 18-21 day old rat puppies were perfused with no substrate, 10 mM glucose and galactose at 1, 2, 4, 10, 20, 40 and 60 mM. Glucose production peaks at 2 mM galactose. Galactose uptake is saturated by the 10 mM level of galactose. ATP decreases with circulating galactose >4 mM. The levels of the Leloir pathway enzymes remain constant with time and substrate level for at least 135 minutes of perfusion. UDPG/UDPgal, G-1-P/G-6-P and lactate/pyruvate ratios remain unchanged at varying levels of galactose. No galactitol or galactonate was produced by these livers at any level of galactose. This system provides an excellent physiological model for the investigation of the regulation and control of galactose metabolism.

EFFECT OF GESTATIONAL AGE ON THE UTILIZATION OF INTRAVENOUS AMINO ACIDS IN THE FIRST WEEK OF LIFE. M.H. Bryan, G.H. Anderson, R.N. Roy, K.N. Jeejeebhoy. The Research Inst. of The Hosp. for Sick Children, Depts. of Pediatrics, Nutrition, Medicine; Univ. of Toronto, Toronto, Canada. (Intr. by P.R. Swyer)

The use of intravenous alimentation in newborn infants has increased where oral nutrition is inadequate. This study reports the response of 28 newborns (gest. 28-40 wks) to a total parenteral infusion of 3.0g L amino acid/kg/day in dextrose during the first 7 days. The infusate contained no tyrosine or cystine. N retention rose with maturity from 28-30 wks and remained constant from 31-40 wks. N losses were as urea and not as α amino N, suggesting that even <31 wk newborns have the capacity to utilize amino acids for gluconeogenesis. Blood amino acid levels were similar on days 1 and 6. Phenylalanine levels were normal and tyrosine levels rose indicating conversion at all gestational ages. Cystine levels were negligible, methionine levels high and taurine/methionine ratios low suggesting that cystine is an essential amino acid in <31 wk infants. These findings reverse with increasing gestation. The evidence suggests that N retention is dependent upon maturation of anabolic protein enzyme systems and that the amino acid contents of available infusates need to be varied according to gestational age.

*Gest. wks.	n	N in mg/k/d	N out mg/k/d	Urea N mg/k/d	α amino N	N retained %
28-30	10	554±52	488±108+	344±61+	26±14	12±19+
31-40	18	581±50 (M±1SD)	346± 77	249±61	23± 9	41±11

+p<0.01

NECROTIZING ENTEROCOLITIS IN INFANTS FED AN ELEMENTAL FORMULA Book, L.S., Herbst, J.J., and Jung, A.L., Department of Pediatrics, Univ.Utah Col.Med., Salt Lake City.

Necrotizing enterocolitis (NEC) is a catastrophic disease that occurs in 5% of admissions to our neonatal ICU. Although a number of associations have been suggested, the pathogenesis of NEC has not been defined. Based on observations in our nursery that: 1) the incidence increases with prematurity (60% of cases occur in <1200 G infants), 2) the incidence increased at about the time increased emphasis was placed on oral alimentation, 3) we have found increased fecal reducing substances in 10-14 infants for 1-5 days prior to developing NEC, 4) fat absorption is decreased in the newborn, we initiated a controlled clinical study to determine the effect of formula on development of NEC. Infants <1200 G were randomized and fed either a standard premature formula (PF) or a 0.67 cal/ml elemental formula (EF) with medium chain triglycerides. The gestational age, birth weight and severity of illness was similar in the two groups. The average rate of increase of formula feeding during the first 8 days was similar (EF=19.4, PF=16.7 ml/kg/day). NEC developed in 7 of 8 infants fed EF and in 2 of 8 fed PF ($p < .005$). The incidence of NEC in the PF group was unchanged from that occurring during the previous 16 months when 20 of 76 infants <1200 G developed NEC. In a retrospective review, 5 infants with 2 or more episodes of NEC had been fed EF on recovery from the first episode. The high osmolarity of EF may be important in the increased incidence of NEC in EF fed infants.

PORTACAVAL SHUNT AND LIPID SYNTHESIS. H. Peter Chase and Thomas E. Starzl. Univ. of Colo. Med. Ctr., Depts. of Pediat. and Surgery, Denver, Colorado.

A 12 y.o. girl with homozygous type II hyperlipoproteinemia has had marked improvement following portacaval (PC) shunt (Lancet, ii:944, 1973). In an attempt to explain the reduction in serum cholesterol (chol) levels, 7 dogs were given end-to-side PC anastamoses and 3 had sham operations. Fasting presurgery mean serum chol levels ± 1 SD were 193 ± 29 mg% in control and 196 ± 14 mg% in test dogs. Chol levels were statistically lower ($p < .01$) 1 month following surgery (controls = 205 ± 27 mg%; tests = 127 ± 21 mg%) and thereafter. The rate of chol synthesis from acetate-2-^{14}C was measured in vitro on liver biopsies from the same dogs. There were no differences in the fasting rate of chol synthesis in the pre-shunt dogs or at 1 or 4 weeks after surgery. However, fed dogs at 6 weeks had a higher rate of chol synthesis for the controls (1060 ± 325 cpm/μg chol) compared to the tests (599 ± 236 cpm/μg chol). In 7 other dogs, all blood from the portal vein was supplied to the right lobes and from the IVC to the left lobes of the liver. The right lobes had a higher rate of lipid synthesis. In 8 other dogs, the pancreatic blood was supplied to the right lobes and the intestinal blood to the left lobes. The left side now showed a higher rate of lipid synthesis. These studies indicate that, at least in the dog, food or other factors absorbed from the intestine, more than pancreatic hormones, regulate the rate of hepatic chol synthesis.

STUDIES ON THE ETIOLOGY OF ACUTE INFANTILE DIARRHEA. Robert Bortolussi, Maria Szymanski, Richard Hamilton and Peter Middleton. Res. Inst., Hosp. for Sick Children, Dept. of Ped., Univ. of Toronto, Toronto, Canada.

Over a 14-month period we examined gastric and duodenal juice and stools from 17 affected infants in the acute phase (within 3 days of onset) and convalescent phase (5 and 20 days later) of their illness. All were hospitalized with severe dehydration, none had bacterial pathogens cultured from stools. In the acute phase, duodenal juice contained virus particles (seen by electron microscopy) in 12 of 17 cases, a specific diplorna virus (orbi) in 10, adenovirus in 2. The diplorna virus was seen in stools of 6 of the 10 infants. Virus was not seen in gastric juice. In the convalescent phase virus was not seen in duodenal juice or in stool. In acute phase duodenal juice fasting total bile salt concentrations were above the critical micellar concentration; there was negligible free bile acid, IgA concentrations, 8.4 ± 1.1 mg/100 ml (m ± sd), were normal as they were in convalescent juice, 7.4 ± 2.6. Candida albicans was cultured from 3 acute phase duodenal juices, one of which contained diplorna virus. Bacteriological studies on the same juices found no known pathogens. Our findings support the view that non-bacterial acute diarrhea in infants is viral. We have identified a specific agent in the majority of our cases which does not appear to disturb bacterial flora or to alter secretory IgA concentration in the upper gut.

QUANTITATIVE ESTIMATION OF RIBOFLAVIN DEFICIENCY IN A LOW SOCIOECONOMIC PEDIATRIC POPULATION BY A NEW METHOD Harold S. Cole, Rafael Lopez, Jack M. Cooperman, Dept. of Pediatrics, New York Medical College, New York, New York

Riboflavin deficiency may be more prevalent than previously suspected in children in whom the main staples of their dietary regimen are cereal grains and beans. The latter are poor sources of this vitamin. However, laboratory methods that could specifically detect the deficiency were not available. Recently, it has been shown that the determination of the degree of saturation of the enzyme erythrocyte glutathione reductase with FAD, a riboflavin coenzyme, was a sensitive and specific indicator of riboflavin deficiency in adults. We have shown that the method is applicable to infants and children. We then studied 100 infants and children ranging from 1 week to 16 years of age. None had diarrhea, vomiting or malabsorption. 11 were found to be deficient, and with one exception, their ages ranged from 4 to 16 years. One infant, 8 months old who was deficient, had neuroblastoma. In general, their diets were characterized by low milk, meat and egg consumption. None had cheilosis or corneal vascularization. Most were below the 50th percentile for height and weight. Because of riboflavin's important role in biological oxidation and reductions, a deficiency of this vitamin may affect growth and development of children. Since many children consume large amounts of starches and sugar, riboflavin deficiency may be more widespread.