457

THE EFFECT OF POSTNATAL AGE ON THE METABOLISM OF INTRAVENOUS FAT EMULSION. Gilberto R. Pereira, Charles A. Stanley, William W. Fox, Lester Baker. (Spon. by Jean A. Cortner), Univ. of Pa. Sch. of Med., Dept. of Peds., and The Children's Hospital of Phila., Philadelphia, PA.

To determine the effect of postnatal age on serum clearing of intravenous fat, 17 preterm, AGA infants with gestation age of 29 ± 0.4 SEM wks. and birthweight of 1.2 ± 0.07 SEM kg. and less than 3 wks. of age received 1 gm/kg of Intralipid over a 4 hour infusion. Plasma levels of triglyceride (TG), free fatty acids (FFA) and beta-hydroxibutyrate (BOB) were determined at 0, 4, and 8 hours after the infusion was started. Basal levels of TG, FFA, and BOB were similar in the 2 groups. Infants of less than 1 wk. old had higher peak TG levels at the end of the infusion (4 hr.), than infants 2-3 wks. old ($p<0.01$). 8 hours after the beginning of the infusion TG levels remained significantly higher in the younger group ($p<0.05$).

		Δ TG (mg/dl)		Δ FFA (mM/l)		Δ BOB (mM/l)	
Age	No.	0-4 h	0-8 h	0-4 h	0-8 h	0-4 h	0-8 h
< 1 wk.	(8)	229±46	23±9	1.2±.2	.4±.2	.06±.03	.01±.02
2-3 wks.	(9)	98±25	7±3	.8±.2	.2±.1	.04±.2	.01±.09

The rise in TG levels seen in our older group was lower than that reported in infants of less than 33 wks. of gestation and comparable with levels obtained from infants of more than 33 wks. of gestation studied within the first 48 hours from birth. This suggests an acceleration in the capacity to tolerate intravenous fats after the first week of extra-uterine life.

458

CHRONIC ACTIVE HEPATITIS IN CHILDREN. Jean Perrault, Allen L. Goldbloom, Ernest Cutz, and James Weber (Spon. by Gunnar B. Stickler). The University of Toronto and the Hospital for Sick Children, Department of Paediatrics and of Pathology, Toronto, Ontario.

In a 20-yr period we saw 33 children (7 boys, 26 girls), 1 to 18 yr old, with chronic active hepatitis. Presenting signs and symptoms were hepatomegaly (91%), jaundice (82%), splenomegaly (76%), bleeding diathesis (50%), anorexia (48%), and lethargy (47%). Laboratory abnormalities (mean values) included increased levels of SGOT (595 IU), bilirubin (7.1 mg/dl), and γ-globulin (3.7 g/dl), prolonged prothrombin time (16 sec), and positive anti-smooth muscle and anti-glomerular basement membrane antibodies. Pi type (protease inhibition) MZ was found in 3 patients, MS in 1, and MM in the other 25 tested. All patients were HB_SAg-negative. Average follow-up (FU) has been 49 mo; 4 are not taking medications (FU 95 mo), 12 are well and taking steroids or azathioprine (or both) (72 mo), whereas 10 still have clinical features (34 mo). Only 4 patients died, 3 of whom had cirrhosis at onset. The remaining 3 patients were seen only in the past few months. Therapy was similar in all groups. Medication was titrated, based on SGOT level; attempts to discontinue treatment failed in 41%. In 80% of patients, SGOT, bilirubin, and albumin levels were normal within 1 yr and remained so, but γ-globulin levels were increased (>1.4 g/dl) in 39% at 1 yr and 63% at 4 yr. Histologic pattern at onset (except for cirrhosis, seen in 7) or abnormal Pi type did not influence the course. γ-Globulin rather than SGOT level might be the most useful parameter.

459

EFFECTS OF CROMOLYN ON INFLAMMATORY BOWEL DISEASE. Yvette Piovanetti, Samuel A. Kocoshis, Daniel G. Sheahan and Joyce D. Gryboski, Yale U. Sch. of Med., Yale-New Haven Hosp., Depts. Ped. & Path., New Haven, Ct.

Clinical and histological effects of Cromolyn (DSCG) were studied in 12 patients with inflammatory bowel disease (IFBD) (5 Crohn's and 7 UC). Patients had refractory disease for at least 1 year. Treatment varied from 6-18 months and was not associated with adverse effects. Granulated mast cells were estimated in the lamina propria of normal control specimens (22) and others with IFBD. Biopsies were classified as normal, healed or chronic active. Index of disease activity was recorded for each patient. Results were in mast cells/mm^2: Normal-343; UC-323 and Crohn's-304. During DSCG treatment: in Crohn's disease 3 of 5 (60%) improved clinically and 2 had progressive disease. Those who improved showed a mean increase of 105 mast cells/mm^2 (from 267 to 372). In UC, 6 of 7 improved clinically (85%). Four of these showed a mean increase of 148 mast cells/mm^2 (from 239 to 387/mm^2). Two who improved clinically but had no increase in mast cells had active histologic disease pre and post-treatment. Overall, those who responded best to drug both by clinical and histologic criteria showed an increase in mast cells. DSCG is of value in the therapy of IFBD, and its effects may be directly related to its action on mast cells.

460

SUBSTRATE UTILISATION DURING ISOCALORIC, ISOVOLAEMIC PARENTERAL NUTRITION(TPN) WITH GLUCOSE/AMINO-ACID(GL/AA) AND FAT EMULSION(IL). G. Putet, T. Heim, J.Smith, P. Swyer. Depts. Paed. and Med. Eng., Univ. of Toronto; the Research Inst. of The Hosp. for Sick Children, Toronto, Canada.

Indirect calorimetry and urinary nitrogen excretion has been used to define the proportions of AA, GL and fat oxidised during TPN over >3 hr. periods under thermoneutral conditions in 14 appropriately grown infants between 1Kg.-3.6Kg. in weight, 27-41 wks. gestational age, from 7-48 days postnatal age. Selected results:

	AA-GL			IL		
	Intake	Oxidised	Stored	Intake	Oxidised	Stored
Kcal/Kg.day	61±10	48±3	27±7.0	65±10	52±5	12.0±10.8
AA(g/Kg.d)	2.7±0.3	0.9±0.4	1.7±0.4	0	0.8*±0.2	0
CH_2O "	13.2±0.2	8.2±0.2	5.0±1.3	1.4±0.2	2.3*±1.7	0
Fat "	0	1.5*±0.7	0	5.8±0.8	4.2±0.9	1.6±1.1

*partly or completely from body stores

Means±s.d.

During AA/GL infusion 64% of Kilocalories(Kcal.) were derived from carbohydrate(CH_2O). Following isocaloric lipid infusion 82% of Kcal. were derived from lipid oxidation demonstrating metabolic flexibility according to substrate available. This change was accompanied by a 15% increase in metabolic rate and a heat storage of 4.1 Kcal/Kg.d. Further studies showed that, below 60Kcal/Kg.d there was a proportionately increased endogenous fat and AA oxidation. At levels > 70 Kcal/Kg.d endogenous fat oxidation became negligible and AA oxidation was minimised.

461

ROLE OF DIETARY PROTEIN AND AMINO ACIDS IN THE MODULATION OF PLASMA SOMATOMEDIN ACTIVITY IN RATS. Robert Reeves, J. Voss, L. Dickinson, J.A. Lee, F.J. Ma, M.J. Elders. Dept of Ped. UAMS, Little Rock, AR.

The mechanism of growth retardation in many nutritional deficiency states has not been delineated. Somatomedin (SM) is depressed after prolonged fasting or in severe malnutrition despite elevated growth hormone (GH) concentrations, suggesting both nutrition and GH may act as modulators of SM. We have studied the effects of varying concentrations of dietary protein (2.5, 5, 10, 20%) and specific dietary amino acid deficiencies on body and organ weights, GH, SM, cartilage glycosaminoglycans (GAGS) and biochemical composition of brain and liver. Changes in body weights and organ weights were similar for 2.5% protein, a valine deficient or a tryptophane deficient diet (-10%). An increase of 177% was observed for animals on a complete amino acid diet or a 20% protein diet. Arginine deficiency had an intermediate effect. Biochemical alterations were more striking in liver than in brain. Serum SM ranged from 0.20 units for rats on the valine and tryptophane deficient diet to 1.44 units for the rats on the complete diet. SM was positively correlated with the protein content of the diet, body and organ weight changes, food efficiency and cartilage GAGS content and negatively correlated with GH concentrations. These data suggest that dietary protein and amino acids play a direct role in the modulation of SM activity and the growth retardation associated with some nutritional deficiency states may be related to its additional effect on SM activity.

462

TREATMENT OF ACUTE DIARRHEA WITH ORAL ELECTROLYTE SOLUTIONS. Jorge T. Rodriguez, Ricardo Blanco, Irene M. Gray, (spon. by Buford L. Nichols), Dept. of Pediatrics, Roosevelt Hosp., Guatemala and Columbus, Ohio.

Fifty-two children 6 to 29 months with good nutritional status, and a mild to moderate dehydration secondary to acute diarrhea received ad libitum one of four oral electrolyte solutions for 48 hours. The solutions differed only in type and level of carbohydrate: 5% glucose, 5%, 10%, and 12.5% corn syrup solids (CS). Electrolyte levels were similar in all solutions (Na^+ 30, K^+ 20, Cl^-30 mEq/l). At 48 hours 47/52 (90%) were normally hydrated by clinical observations with no difference among treatments. Mean intakes (all subjects) were 138 ml/kg (day 1) and 153 ml/kg (day 2) with no difference among groups. In 48 hours urine specific gravity (1.012 to 1.008) and serum urea (15.0 to 9.6 mg/dl) decreased and serum sodium (134 to 137 mEq/l) increased with no difference among treatments. Stool weights were 36.1 g/kg (day 1) and 45.1 g/kg (day 2) with no difference among treatments. Greater numbers of glucose positive stools were observed with 10% and 12.5% CS solutions than with either 5% solution. Stool electrolytes were Na^+20 and K^+ 23 mEq/kg stool/day and did not differ as to treatment. Stools of 7/52 (14%) patients contained bacterial pathogens. Diarrhea worsened in patients in each group (12.5%CS, 7/13; 10%CS, 5/13, 5%CS 1/13; 5% glucose, 2/13). Oral electrolyte solutions with 30 mEq Na/liter were effective in rehydration and electrolyte maintenance. Solutions with 5% glucose or CS resulted in less carbohydrate malabsorption.