EXPERIMENTAL PRODUCTION OF NECROTIZING ENTEROCOLITIS IN NEWBORN GOATS. Intr. by M. J. Sweeny.
Robert A. deLemos*, James H. Rogers, Jr.*, and Gary W. McLaughlin*. Department of Pediatrics, Wilford Hall USAF Medical Center, Lackland AFB, Texas, and Department of Pediatrics University of Texas Medical School at San Antonio, Texas.

To determine if there might be a relationship between oral feedings and necrotizing enterocolitis, 30 newborn goats were divided into 3 groups and fed 3 separate formulae. Ten received mother's milk, ten received mother's milk dialyzed to raise its osmolarity to 1.5 times normal and 10 received a standard 20 calorie/oz. commercial formula. Half of the goats in each formula group were artificially colonized with E. Coli.

No animal fed unmodified mother's milk developed any significant abnormality. All of the goats fed hypertonic mother's milk developed diarrhea; 6 had milena; 8 had emesis. In this study group mucosal necrosis was seen in all animals at post mortem examination and 4 of the 5 animals colonized with E. Coli developed pneumatosis intestinalis. All of the newborn goats deprived of mother's milk died of sepsis.

These studies suggest that both mucosal damage and colonization with gas-forming bacteria are necessary for the development of necrotizing enterocolitis. One pathway leading to mucosal damage may be the ingestion of hypertonic formula.

EXPERIMENTAL FANCONI SYNDROME: INTESTINAL TRANSPORT OF SODIUM, AMINO ACIDS AND GLUCOSE. Ramon A. Exeni, Raul A. Wapnir, Francisco de Rosas, Melinda McVicar and Fima Lifshitz, North Shore Univ. Hosp., Dept. of Ped., Manhasset, NY and Cornell Univ. Med. Col., Dept. of Ped., New York, NY.

The effects of Na maleate-induced Fanconi Syndrome on intestinal transport were assessed in rats by an in vivo perfusion technique. Na maleate was given i.p. (1.5 or 9.0 mmoles/kg) to 150 g male Wistar rats. Glycosuria and proteinuria ensued in all animals within 30-60 min. After 90-180 min, 20 cm jejunal segments were perfused for 2 hr under urethane anesthesia. The solutions perfused were Krebs-Hanseleit bicarbonate, isotonic, buffers containing 124 mEq/L of Na, 20 or 40 mM glucose and 1 mM of either L-phenylalanine, L-lysine, glycine or α-aminoisobutyric acid. Tracer amounts of ^{14}C or ^{3}H- labelled glucose or amino acids were included, as well as 600 mg% polyethylene glycol, m.w. 3000-3700. The mean ± S.D. transport rates (in μEq/min/cm of jejunum) for Na were, controls: 165.76 ± 39.46; 1.5 mmoles/kg maleate: 93.97 ± 53.62 ($p < .001$) and 9.0 mmoles/kg maleate: 51.70 ± 22.31 ($p < .001$). There were no differences in the transport of glucose and amino acids between control and experimental animals, despite multiple tubular transport defects in the latter. These results suggest that maleate-induced Fanconi Syndrome affects only Na intestinal transport, but not that of amino acids or glucose.

EFFECT OF CHOLERA TOXIN IN Na TRANSPORT IN INTESTINE - Jehan-F. Desjeux, Y.H. Tai and Peter F. Curran, (Intr. by Charles D. Cook) - Yale Univ. Sch. of Med. Dept. of Physiology, New Haven, Conn. 06510.

Massive secretion of fluid and electrolytes into the intestinal lumen is observed in cholera but, in normal intestine, more than 50 per cent of Na transepithelial flux involves an extracellular route. This study was, therefore, undertaken to determine the role of the epithelial cells in the Na secretion observed in cholera. From measurements of flux as a function of electrical potential, the transcellular Na fluxes (Jc) were estimated, in vitro, on ileum treated with cholera toxin (CT) for 60 minutes and compared with Jc from unchallenged control (C) intestine from the same rabbit. In absence of electrochemical potential, in Ringer's solution, CT caused a net Na secretion (-1.35 ± 0.21 μeq/hr cm^2), without statistical change in short circuit current. The net secretion appears to be due to a decrease of transcellular Na flux from mucosa to serosa (Jcms) (-0.94 ± 0.35 μeq/hr cm^2) and an increase of flux from serosa to mucosa (Jcsm) (+0.75 ± 0.64 μeq/hr cm^2). The addition of 10mM glucose caused an increase ($p<0.01$) of J_{net}^{Na}, Isc and Jcms almost identical in C and CT intestine. However, Jcsm did not change significantly with glucose. These results indicate that CT caused an active cellular Na secretion which is most easily explained by postulating that CT stimulates an electrically neutral (Na+Anion) transport process from serosa to mucosa but does not change the "electrogenic" Na absorption.

INTESTINAL ABSORPTION OF LEAD; INFLUENCE OF AGE, CALCIUM AND MAGNESIUM. Burton P. Fine, Alexander Barth and Mahmoud Fathalla, Dept. of Ped., New Jersey Med. Sch., Newark, N. J.
(Intr. by Franklin C. Behrle)

Childhood lead poisoning is usually by gastrointestinal absorption. Calcium and iron ingestion have been shown to increase tissue lead levels and signs of lead intoxication. Lead balance studies were performed on four mature dogs (18 months old) and three 8 week old puppies while on a basal diet free of electrolytes except NaCl, KCl and $PbCO_3$ (0.1 mg Pb/kg/day). Fifteen day control periods were alternated with 15 day periods in which $CaCO_3$ or $MgCO_3$ was added to the diet. Other dogs fed Na_2CO_3 showed no change from controls in lead absorption. The control absorption for the mature dogs was 31.6 ± 6.4 % (m ± S.D.) and for the puppies 30.1 ± 8.2 % of the ingested lead. This difference was not significant. The percent absorption during the control periods of both groups combined was 30.9 ± 7.2 % which decreased to 27.1 ± 6.4 % (N.S.) when $CaCO_3$ was added to the diet and to 10.0 ± 3.4% ($p < .01$) when $MgCO_3$ was added. We conclude that in this experimental situation magnesium ingestion decreases the intestinal absorption of lead, and that age is not a significant factor.

Reaction to Bowel Injury in the Fetal Rabbit. John R. Esterly, Fred D. Haruda, Bjorn H. Thomasson, and Mark M. Ravitch. Departments of Pathology and Surgery. University of Chicago, Chicago, Illinois.

The fetal inflammatory response was observed following intestinal perforation made in utero at 20, 22, 24, and 28 days gestation (DG), and in newborn (31DG) and in adult New Zealand rabbits. In contrast to the newborn and adult, fetal peritonitis was characterized by a less focal reaction, a significantly retarded and less intense acute inflammatory response, and the absence of necrosis of the bowel wall. The response at 28DG showed an initial hyperplasia of the serosal mesothelial cells, a inflammatory response after 48 hrs., and granulation tissue by the 4th. day. At 24DG, fibrin and inflammatory cells were still meager by the 5th day and organization of the exudate was incomplete by 7 days. Animals operated on at 20 and 22DG showed only serosal reactions, limited fibrin, and a few inflammatory cells when examined at term. These observations suggest that gastric, hepatic and pancreatic secretions play an unimportant role in the injury responsible for peritonitis in the fetus. They also confirm the progressive role of the inflammatory response with increasing gestational age.

24 HOUR PATTERNS OF INSULIN & GLUCOSE DURING TOTAL PARENTERAL NUTRITION. Jordan W.Finkelstein, Michael I.Cohen, Scott J.Boley* Jacob Kream*, Leon Hellman*, Albert Einstein Col. of Medicine, Montefiore Hosp. & Med. Ctr., Depts. of Pediatrics, Surgery, and Oncology, Bronx, New York.

The concentration of glucose(G) and insulin(I) was measured every 20 min for 24 hrs in 3 pts receiving only 18% glucose and 3.3% casein hydrolysate in order to evaluate the G & I response to total parenteral nutrition. Pt 1 received 4L by gravity drip. G was 16-158mg% and I was 0-372μU/ml. Peaks of G and I occurred whenever a new bottle of infusate was started. In pt 2 who received 5L, G was 31-210, I:0-216 and rate of flow 100-240ml/hr. Peaks of G and I corresponded to peaks of the rate of flow. In pt 2 5L of 10% glucose alone was later administered using a pump. Flow was 140-180ml/hr, G:49-93 and I:0-25. Pt 3 received 3L during a 12 hr gravity drip and a 12 hr pumped period. During the gravity drip, flow was 30-180ml/hr, G:50-123 and I:0-78. During the pumped period flow was 120ml/hr, G:66-101 and I:0-24. Urinary excretion of G was < 2% of the amount infused in all pts. The main factor controlling the conc. of G and I seems to be the rate of flow. During gravity drips marked changes in flow rate cause parallel marked changes in G and I which were eliminated during pumped periods. When the conc. of G is held relatively constant, little I is secreted, and complete utilization of nutrients takes place. These data suggest that constant infusion of hypertonic fluids will minimize fluctuations in G and I and prevent both hyper- and hypoglycemia.