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**RADIONUCLIDE TEST FOR GASTROESOPHAGEAL REFLUX (GER) IN CHILDREN.** Dennis L. Christie, Thomas G. Rudd (Spon. by Vincent C. Kelley). The Mason Clinic, University of Washington School of Medicine, Department of Nuclear Medicine & Children's Orthopedic Hospital, Seattle.

A simple and reliable means of detecting abnormal GER in children is needed. A radionuclide method was evaluated in a series of 25 patients with documented GER (positive pH reflux test). Symptomatic patients with abnormal pH reflux test ( $\text{pH} < 4.0$ ) were studied. With the patient supine or semi-upright, 200 uci of TC-99m sulfur colloid in apple juice was given orally. Patients were allowed to drink until sated (volume 60-300cc). Gamma camera images were then made in the basal state, with hand pressure and at graded increments of external abdominal pressure (blood pressure cuff around abdomen) up to 100 mm Hg. Any visible activity in the esophagus was interpreted as an abnormal test. In addition, all patients had lower esophageal sphincter pressure (LESP) determinations as well as barium cine-gastroesophagograms.

The radionuclide study was easy to perform and well tolerated. Evidence of reflux was detectable in 20 patients using this method. The LESP was 12 mm Hg or less in 14 patients. The barium x-ray study was abnormal in 15 patients.

Radionuclide gastroesophagography is a sensitive technique for detecting GER in infants and children. Additional studies to further assess clinical utility and specificity appear warranted.

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**THE INTERACTION OF BOVINE CASEIN AND ENTEROTOXIN.**

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Secretory IgA in human breast milk can neutralize cholera enterotoxin in the rabbit ileal loop system. No similar protection by separate milk proteins could be demonstrated but one milk protein, casein, had a deleterious effect on intestine exposed to very small quantities of enterotoxin.

Rabbit intestinal loops were prepared by Sack's modification of the method of Kasai and Burrows. Highly purified *V. Cholerae* toxin (10 or 100 ng) was incubated with 10 mg of bovine protein solutions (lactalbumin, casein, lactoglobulin A, lactoglobulin B, gamma globulin) for 60 min. at 37° C. One ml aliquots were then injected into the rabbit ileal loops. Eighteen hours later the animals were sacrificed. The intestinal loop contents were aspirated and a volume/length of loop ratio was determined.

The activity of 100 ng of toxin was not affected by the majority of bovine milk proteins, but bovine casein caused a 40% increase in the fluid production ( $p < 0.001$ ). When the milk proteins were added to a low dose of enterotoxin (10 ng), all but casein nonspecifically inhibited the action of toxin. Bovine casein caused a 78% increase in fluid production by loops exposed to suboptimal toxin dose (10 ng). The exaggerated intestinal fluid response to low dose toxin and casein may result from mucosal damage similar to that caused by the higher dose of toxin alone.

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**INCREASED RISK OF PULMONARY EMBOLUS IN CHILDREN RECEIVING CENTRAL HYPERALIMENTATION.** T.D. Coates, H. Eigen, T.V.N. Ballantine, K.A. Rickard, J.L. Grossfeld and R.L. Baehner, Indiana University School of Medicine, Department of Pediatrics and Pediatric Surgery, Indianapolis, Indiana.

Pulmonary embolus (PE) is rarely observed in children but the incidence is increased in patients with a indwelling artificial prosthesis. The purpose of this study was to document the incidence of PE in newborns and children receiving central hyperalimentation (CHA) during a two year period. CHA was given to 131 patients employing a non-heparinized centrally-placed catheter. Five had clinical, radiologic or pathologic evidence of PE (incidence = 3.8%) and all had indwelling polyethylene catheters. Their ages ranged from 4 months to 19 years; three had cancer, one had regional enteritis and the other had total colonic aganglionosis. All developed sudden onset of tachycardia and 3 had respiratory distress. Ventilation perfusion studies in 3 were consistent with PE. Two expired with massive PE evident at autopsy. This study demonstrates the increased risk of PE in children receiving CHA and emphasizes the need for sensitive monitoring of changes in pulmonary function suggestive of this complication.

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**CHRONIC NON-SPECIFIC DIARRHEA: ROLE OF DIETARY MANAGEMENT IN CONTROL.** Stanley Cohen, Richard Mathis and W. Allan Walker, Massachusetts General Hospital, Pediatric Gastrointestinal and Nutritional Unit, Boston, Massachusetts 02114.

Chronic non-specific diarrhea (CNSD) still represents the most common cause (85%) of chronic diarrhea in childhood. Although self-limited, this condition has been a therapeutic enigma for gastroenterologists. In an attempt to control this problem, we have assessed the association of CNSD with altered dietary intake due to either iatrogenic measures used to treat post-infectious diarrhea or due to dietary idiosyncrasies within families. Of 86 consecutive patients referred with chronic diarrhea, 48 were classified as having CNSD (Davidson's criteria) after a negative malabsorption workup including lactose tolerance test. Duration of symptoms ranged from 3 weeks to 6 months. When a complete dietary history was obtained on 23 patients, 19 reported a low fat (<25% of total calories) and high carbohydrate (>50%) intake. Fourteen additional patients gave histories suggestive of similar intakes. When placed on diets with increased levels of fat (>50%) and with lower carbohydrate levels (<20%), 19 of 23 patients demonstrated normalization of stool frequency and consistency within 5 days to 2 weeks. In addition, 4 patients with a normal dietary intake also responded to an increase in dietary fat. These observations suggest that iatrogenic changes in diet to treat diarrhea (low fat, high carbohydrate) may account for chronicity in numerous instances and an increased fat intake may decrease bowel motility.

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**HEMOCARPOPERFUSION IN CHILDHOOD HEPATIC COMA.**

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Fulminant hepatic failure and coma in children has a mortality rate of 60 to 70%. Hemocaropoperfusion (HCP) employing an acrylic hydrogel polymer coated charcoal column was used in three children ages 8, 11, and 13 years presenting with acute encephalopathy and hepatic failure. Two children were in stage IV hepatic coma and one in stage V Reye syndrome. Following HCP all patients showed significant improvements in deranged serum chemistries. Serum ammonia and amino acid concentrations decreased dramatically. Serial examinations revealed no evidence of hemolysis, thrombocytopenia, or microembolization secondary to HCP. One of three patients survived and had no evidence of cerebral edema. The other two patients developed posturing and pupillary changes suggestive of severe cerebral edema prior to HCP. Continuous intracranial pressure monitoring (employing Richmond screw) in one patient showed  $> 50 \text{ mmHg}$  (N:0-10) which transiently diminished with mannitol infusion but failed to respond to HCP. The patients who expired had nearly isoelectric EEGs which showed no improvement following HCP.

The patient who responded to HCP and had no evidence of cerebral edema is well without complications after a two year period. It is concluded that for effective HCP in children with hepatic encephalopathy, the therapy should be initiated before the onset of cerebral edema.

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**LIGHT MICROSCOPIC ABNORMALITIES OF PROXIMAL SMALL INTESTINAL BIOPSIES OF PATIENTS WITH CYSTIC FIBROSIS (CF).**

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In pancreatic insufficiency, a reduction of  $\text{HCO}_3^-$  secretion results in increased proximal small intestinal acidity. Injury to the mucosa by increased acidity may contribute to malabsorption. Multiple small intestinal biopsies (bx) from the region of the duodeno-jejunal junction were examined in patients with CF and controls (C) to determine if there was significant mucosal injury. The bx were read in a double blind fashion by 2 independent observers. 40 small intestinal bx from 10 patients with CF and 12 bx from 3 normal C were evaluated. Bx were evaluated for villus-crypt ratio, mitotic index, cell content of lamina propria and other morphologic abnormalities. Bx were categorized as normal, mildly, moderately or severely abnormal on the basis of specific morphologic criteria. The CF patients (mean age 15.2 years) had steatorrhea in spite of taking high doses of pancreatic enzymes. Mean fasting duodenal pH in 7 was 4.96. The bx from 8 CF patients were distinguished from the C by both observers. Bx from 2 were categorized as moderately, 2 as mildly-moderately and 4 as mildly abnormal. The bx from 2 CF patients could not be distinguished from the C by both observers. There was complete concordance between the observers. Proximal small intestinal bx of 8 of 10 CF patients demonstrated significant mucosal injury. This may have resulted from increased proximal small intestinal acidity due to pancreatic insufficiency or increased acid production and may contribute to malabsorption.