469 THERAPEUTIC INTRAVENOUS GALACTOSE IN GLUCOSE-INTOLERANT PREMATURES. John W. Sparks, Gordon B. Avery, Anne B. Fletcher, Gregory Buffone, and Walter H. Glinsmann. Children's Hospital National Medical Center, George Washington University School of Medicine, and National Institute Child Health and Development, Washington, D.C. and Bethesda, Maryland.

Small, sick prematures often require intravenous alimentation and are sometimes intolerant to concentrated glucose, with hyperglycemia and glycosuria. Animal studies have shown better glucose regulation in the presence of galactose, a normal product of lactose digestion. Possible benefit of IV galactose was studied.

Blood galactose levels on 58 newborns taking milk by mouth showed prompt utilization with peak levels $\leq$ 5 mg/dl. T1/2 was 45 min. with no significant effect of gestational age or time after birth, indicating necessary enzymes are present. Therapeutic infusions of equal conc. of glucose and galactose in 4 glucose-intolerant premies and 1 infant of a diabetic mother yielded promising results. All stabilized blood glucose in the normal range (70-120), 2 cleared significant urine glucose spills, 3 recovered from previous hyperglycemia, there was a tendency to tolerate higher CHO loads, and no clinical toxicity was noted. Blood galactose levels were monitored and remained below 20 mg/dl. These results suggest that IV galactose is feasible and may cause less hyperglycemia in sick prematures and less insulin stimulation in IDMs while allowing administration of additional calories as CHO.

470 TAURINE IN THE DEVELOPING KITTEN: NUTRITIONAL IMPORTANCE. J.A. Sturman, D.K. Rassin, K.C. Hayes & G.E. Gaull. Div. of Human Devel. & Genet., Inst. for Res. Ment. Retdn., Staten Is., NY: Dept. Nutr., Harvard Sch. Pub. Hlth., Boston, Ma.; Dept. Pediat., Mt. Sinai Sch. Med., CUNY, NY.

Kittens fed a purified-casein diet immediately after weaning develop retinal degeneration and blindness unless supplemented with taurine (tau). Supplementation with tau precursors (methionine, cystine or inorganic sulfate) does not prevent this. In contrast, the amount of tau in the form of taurocholate in these kittens does not change appreciably and tau concentration in brain tissue is more severely depleted than retina, but without apparent behavioral or pathological change.

Apparently the most important function of tau in kittens is bile acid conjugation and this function is maintained at the expense of tissues. Second in importance seems to be retinal function but the kitten is unable to maintain normal retinal tau conc because of: 1) its postnatal rapid rate of increase in retinal tau conc, 2) the high tau conc in cat retina, 3) the low rate of tau synthesis in cat, 4) the rapid growth rate of kitten, 5) the inability of cat to convert from tau conjugation to predominantly glycine conjugation of bile acids (as the human infant does).

These changes are likely to be more severe if the kitten is fed the synthetic diet immediately after birth, for cat milk contains a high concentration of tau and therefore can minimize the deficiency. The relevance of these results to the human infant will be discussed in light of the limited ability of man to synthesize tau, the low conc of tau in cow milk and formulas derived from it, and the high conc of tau in human milk.

471 CHYLOTHORAX FROM SPONTANEOUS AND IATROGENIC ETIOLOGY Nestor E. Vain, Chul C. Cha, Orville W. Swarner, (Spon. by James J. Quilligan). Loma Linda University School of Medicine, Department of Pediatrics, Loma Linda, Calif.

This is a description of seven consecutive cases of chylothorax in neonates. Chylothorax is known to occur spontaneously and is a recognized but infrequent cause of respiratory distress in neonates. It is, however, the most frequent cause of pleural effusion in the newborn. In four of the seven cases, the chylothorax was a spontaneously occurring event as has been previously described in the literature. But in three cases, it occurred in close temporal association with hyperalimentation. All three cases of chylothorax which were associated with the hyperalimentation and superior vena cava thrombosis died. In the four spontaneously occurring cases of chylothorax, a medium chain triglyceride diet was utilized to reduce the chylous accumulation and load in all four of these cases. The infants survived intact.

It is our report that chylothorax may be added to the list of possible complications of hyperalimentation via the superior vena cava route and that the needs of this form of hyperalimentation in the low-birth-weight infant must be weighed against this and other possible complications. It is further suggested that study is indicated on the use of medium chain triglycerides versus other formulae in cases of chylothorax to ascertain whether there is a significant difference in the amount of chylous accumulation which results.

472 DEMONSTRATION OF AN ADEQUATE BARRIER TO GASTROESOPHAGEAL REFLUX IN THE NEWBORN AND PRETERM INFANT. Jon A. Vanderhoof, Peggy J. Rapoport, and Charles L. Paxson, Jr., U. of Nebraska Coll. of Med., Omaha, (Spons. by G. C. Rosenquist).

Previous investigators have stated that newborn and preterm infants have an incompetent lower esophageal sphincter (LES) and are therefore susceptible to gastroesophageal reflux (GER) and aspiration pneumonia. We tested this hypothesis by comparing 5 "healthy" preterm and 10 fullterm infants to 10 infants under 1 yr. age with GER. GER was defined by free reflux of barium on barium swallow roentgenograms, or the persistence of acidic fluid (pH 4) in the lower esophagus following gastric infusion on 0.1N HCL in a dose of 173 cc/m^2 body surface area. LES pressure gradients were measured by withdrawing a single lumen side opening catheter perfused with H_2O at 1.3 cc/min. from the stomach into the esophagus. Pressure gradients across the LES were as follows:

	mmHg+SD
Preterm infants	49±9
Fullterm newborns	39±8
Infants with GER	15±4

All normal preterm and fullterm neonates exhibited LES pressure gradients strikingly higher than infants with GER ($p<0.001$).

Our data suggest that infants with LES pressure gradients of less than 25 mmHg may be at risk for GER, and that healthy preterm and fullterm neonates have a competent lower esophageal sphincter.

473 SINGLE LUMEN PERFUSED CATHETER MANOMETRICS: A SENSITIVE NEW TECHNIQUE FOR MEASUREMENT OF LOWER ESOPHAGEAL SPHINCTER PRESSURES IN YOUNG INFANTS. Jon A. Vanderhoof, Peggy J. Rapoport, and Charles L. Paxson, Jr., U. of Nebraska Coll. of Med., Omaha, (Spons. by G.C. Rosenquist).

Lower esophageal sphincter pressures (LESP) are generally measured during esophageal motility studies using multilumen water perfused catheters. Because of the large size of this assembly, gagging occurs, sedation is required and acceptable tracings may not be obtained. We have compared a large catheter assembly (three 1.2 mm single lumen catheters bound together with a 4.0 mm total diameter) with a 1.2 mm single lumen catheter with 1.0 mm side orifice. Triplicate studies were obtained by perfusing the catheters with water at 1.3 cc/min and slowly withdrawing from the stomach to LES. LESP were obtained in 10 fullterm newborns 3 hours after feeding; infants were then sedated and 90 min. later triplicate studies repeated. LESP were as follows:

	mmHg+SD
single lumen-sedated	42± 8
single lumen-unsedated	39± 10
triple lumen-sedated	22± 7

In two patients, gagging prevented the recording of adequate tracings from triple lumen catheters. Our data indicates that LESP can be successfully measured in the unsedated infant using a small single lumen catheter, and that higher pressures are obtained with the single lumen assembly.

474 ACRODERMATITIS ENTEROPATHICA (A.E.) : STUDIES OF ZINC METABOLISM. Philip A. Walravens, K. Michael Hambidge and Kenneth H. Neldner. Depts. of Ped. and Med. Univ. Colorado Medical Center, Denver.

The metabolic changes associated with Zn therapy were studied in 3 pediatric A.E. patients. Hypozincemia (plasma Zn: 30-39 µg/dl; N= 68-100), hypozincuria (urine Zn 37-57 µg/day; N= >100) and low levels of serum alk. phosphatase were present during relapse. Zn administration (1-3mg/kg/day) caused remission of symptoms and normalization of biochemical parameters. The Zn concentration of skin was low, prior to, and corrected with Zn therapy.Plasma copper levels were normal in these 3 subjects but a 4th patient, age 4 months, received excessive Zn medication and developed a copper deficiency syndrome. Two 3-year old subjects had height increments of 11.1 and 11.6cm in the 13 months following onset of Zn treatment.

Jejunal mucosal Zn content of one patient in relapse was 77 µg/g dry weight (control values: 2 subjects= 97 µg/g dry weight) and increased after Zn treatment to 321 µg/g dry weight. The Zn content of cultured fibroblasts was assayed in 7 controls (mean+SD= 232 ± 96 ng Zn/mg protein) and in 2 A.E. patients (310 ± 83 ng Zn/mg protein). The clinical and biochemical findings, and the response to Zn therapy are consistent with a severe Zn deficiency state in untreated A.E. Present evidence indicates that A.E. results from an inherited defect in Zn absorption, which can be compensated by administration of supplemental oral Zn.