626 **NONNUTRITIVE SUCKING(NNS) DOES NOT STIMULATE THE SECRETION OF LINGUAL AND GASTRIC LIPASES IN PREMATURE INFANTS.** Yolande F. Smith, Judy Bernbaum, Margit Hamosh, Carla M. York, Christine J. Waring, Carol S Fink, Susan D. Bowers and Paul Hamosh. (Spon. by Pedro A. Jose) Georgetown Univ. Med. Ctr. Washington DC and Univ. of PA, Sch. of Med.-Childrens' Hosp.of Philadelphia, PA.

NNS improves weight gain and allows earlier hospital discharge of low birth weight (LBW) infants (*Pediatrics* 71:41, 1983). The improved weight gain could be due to more efficient nutrient digestion and absorption.Preduodenal lipases have a compensatory function in fat digestion during physiologic (developmental) or pathologic (cystic fibrosis, alchoholism related) pancreatic insufficiency (*J Clin Invest* 67:838, 1981; 73:374, 1984; *Pediatr Res* 18:402, 1984 and *Gastroenterol* 1987, in press). We have therefore investigated whether NNS stimulates the secretion of these lipases in LBW infants. Two separate studies were conducted:1) 9 infants (gest. age 30.5±0.8 wks, postnatal age 29±3 days) received gavage feeding (GF) with and without NNS, each infant being his/her own control. 2) 10 infants (gest. age 29.7±0.8 wks, postnatal age 32±5 days) were divided into two groups: 4 infants received GF without NNS and 6 infants received GF with NNS. Lipase activity was measured in gastric aspirates.

Lipase Activity (nmol FFA/ml aspirate/min)*			
STUDY 1	GF	446±168	(0-1513)
	GF + NNS	620±261	(0-1571)
STUDY 2	GF	410±156	(5-1443)
	GF + NNS	346± 98	(0-1450)

*Hydrolysis of ^{3}H Triolein; data are mean±SEM and (range)

These data suggest, that increased secretion of lipases is not the cause of improved weight gain in infants fed by gavage with nonnutritive sucking. (Support: NIH grant HD 10823.)

627 SMALL PERCUTANEOUS CENTRAL VENOUS CATHETERS IN INFANTS AND CHILDREN. Daniel B. Sobel, Susan A. Kecskes. (Spon. by Dharmapuri Vidyasagar) University of Illinois Medical Center, Department of Pediatrics, Chicago, Illinois

Surgically placed central venous catheters (CVC) are increasingly being replaced by percutaneous CVC (PCVC) for nutritional and fluid support of neonates. (Durand, Pediatrics, 1986) We report successful use of the same small silastic PCVC in infants and children aged 2 months to 12 9/12 years with weights between 4.4 and 21 Kilograms. Indications for use included post-operative short bowel syndrome, regional ileitis, intractable diarrhea, and meningitis. Although tiny (internal diameter 0.012 inch, outer diameter 0.025 inch) PCVC accommodated flow rates from 2-90 ml/hr and hyperalimentation fluid with concentrations of 25% Dextrose and 20% Intralipids.

Analgesia was used for the procedure which was performed in the treatment room or the patient's own room. Placement (in the Subclavian vein or Right Atrium) was confirmed by chest x-ray with injection of 0.3cc of Renografin. PCVC remained in place an average of 29 days (range 5-43 days). Difficulty advancing the catheter from peripheral entry site was noted. Complications included occlusion (treated with Urokinase), dislodgement of the catheter and infection.

Conclusion: PCVC is an alternative to surgically placed CVC and does not require ligation of major veins. They can be placed at the bedside, without general anesthesia, operate at high flow rates, with high viscosity fluids, and remain functional for long periods of time.

628 EFFECT OF EPINEPHRINE ON BASAL GASTRIN LEVELS OF NEONATAL PIGS, William S. Stevens and Bradley M. Rodgers, University of Virginia Hospital, Department of Surgery, Charlottesville, VA. 22908

The elevated serum gastrin of the neonate may be the result of maximum stimulation of the G-cell. Epinephrine is a stimulant of gastrin release in adult man.This study evaluated the effects of epinephrine on basal serum gastrin in neonatal pigs. Thirteen Landrace piglets less than fourteen days of age were administered halothane, nitrous oxide, oxygen mixture. After a 15 minute period, IV epinephrine was administered at 25 or 50ng/kg/min for 15 minutes. After a second 30 min stablization, epinephrine was administered at 100 or 200ng/kg/min. Blood samples for gastrin and epinephrine were obtained at 5,10, and 15 minutes of infusion. Samples were assayed for gastrin by radioimmunoassay and epinephrine by high performance liquid chromatography.

	BASAL	5 min	10 min	15 min
Group A: (25ng/kg/min)				
N=6	377.9±94	283.7±57*	297.5±76*	298.7±20*
Group B: (50ng/kg/min)				
N=6	489.3±145.6	382.7±88.4*	330.3±69*	358.7±77*
Group C: (100ng/kg/min)				
N=6	256.4±69.2	216.8±52	246.2±86.7	246.2±83.9
Group D: (200ng/kg/min)				
N=6	383.9±97.3	371.9±95	403.±113	396.2±135

(Gastrin pg/ml±SEM) *p <.05 compared to basal

1) Epinephrine in doses of 25 and 50 ng/kg/min result in a significant decrease in serum gastrin levels. Doses of 100 and 200ng/kg/min had no effect on basal gastrin levels. 2) Neonatal gastrin levels appear maximal and the G-cell cannot be stimulated by epinephrine.

629 ONE-YEAR FOLLOW-UP OF MENTAL/MOTOR DEVELOPMENT AND GROWTH IN CHILDREN AFTER SUCCESSFUL LIVER TRANSPLANTATION. Sunita M. Stewart, Ricardo Uauy, William Belknap, Betsy Kennard, David Waller, Walter Andrews (sponsored by J. Warshaw). Univ. of Texas, Depts. of Pediatrics and Psychiatry, Dallas, TX

We measured anthropometrics, intellectual and motor functioning in 18 children (8 months - 15 years) before and one year after liver transplant. Depending on age at testing, we obtained Bayley Mental (MDI) and Psychomotor (PDI) Development Indices, or Stanford Binet IQ and Motor Age (MA), or Wechsler Verbal (VIQ), Performance (PIQ), and Full Scale (FSIQ) IQs on each subject. Weight (W), height (H), and head circumference for age (H/A, HC/A), and W for H (W/H) were expressed as percent of median (NCHS standards). Relevant findings were (Means):

TRANSPLANT	H/A	HC/A	W/H	VIQ	PIQ	IQ-MDI	MA-PDI	SCORES<80
Before	92	98	102	94	96	80	78	68
After	92	100	112	94	96	79	73	74
p (paired t)≤	1.0	.005	.002	1.0	.91	.98	.59	.10

No significant differences were found in mean development scores before and after transplant. However, for subjects with scores <80 before transplant, there was a trend towards improvement after transplant. Older subjects consistently improved in visual attention and fine motor speed. We conclude that there were significant increments in weight and head growth but no increase in linear growth. Intellectual and motor development remain unchanged for the group as a whole, although the most delayed made meaningful improvement. Long-term follow-up studies are indicated to determine ultimate intellectual outcome.

630 MENTAL FUNCTION AND GROWTH IN CHILDREN WITH EARLY (E) VS. LATE (L) ONSET END-STAGE LIVER DISEASE (ESLD). Sunita M. Stewart, Ricardo Uauy, William Belknap, Betsy Kennard, Margareta Benser, David Waller, Walter Andrews (spon. by J. Warshaw). Univ. of Texas, Depts. of Pediatrics and Psychiatry, Dallas, TX

We compared mental function and growth in 11 children with onset of liver disease in the first year of life (range:1-12 months) with 16 patients with late onset (18 months-11 years). Age ($\bar{x}$±SD) at testing was: E:8±3; L:11±4 years. Wechsler Scales of Intelligence provided Verbal (VIQ), Performance (PIQ) and Full Scale (FSIQ) IQs. Percent of median (NCHS standards) for weight (W) and height (H) for age (W/A, H/A) and W for H (W/H) were also obtained. E and L groups were similar in severity of liver disease as measured by serum bilirubin (range, E:1-37; L:1-46 mg/dl), albumin (E:2-4; L:2-4 gm/dl), γGT (E:30-258; L:54-512 IU) and vitamin E (Mean±SD, E:7±8; L:8±10 µg/ml). Duration of disease ($\bar{x}$±SD) was: E 8.0±2.7, L 4.4±3.5 years. RESULTS ($\bar{x}$±SD):

GROUPS	VIQ	PIQ	FSIQ	W/A	H/A	W/H
Early (E)	81±15	83±13	80±14	85±25	87±13	114±22
Late (L)	98±11	105±16	102±13	96±19	96± 6	99±12
p (t test)	.002	.002	.0006	ns	.008	.004

Mental function and growth were significantly delayed in the E group; height deficits suggested stunted growth from severe early malnutrition. We conclude that liver disease during early brain growth has pernicious effects on ultimate intellectual function and linear growth; duration of disease prior to ESLD may also play a role. We speculate that aggressive nutritional support must begin at diagnosis in early onset liver disease to prevent delayed growth and development.

●631 THE KINETICS OF NA^+/H^+ EXCHANGE BY BASOLATERAL PLASMA MEMBRANE VESICLES FROM NEONATAL RAT LIVER. Frederick J. Suchy and Anita L. Goodrich, Children's Hospital Research Foundation, Cincinnati, Ohio.

The earliest known cellular response to transmembrane signalling by polypeptide growth factors is to stimulate a plasma membrane Na^+/H^+ exchanger which facilitates mitogenesis through alkalinization of the cytoplasm. We recently reported (Hepatology 6:1175, 1986) that Na^+/H^+ exchange activity was markedly enhanced in basolateral plasma membrane vesicles (BLMV) isolated from neonatal (7 days) rat liver. The purpose of this study was to define the mechanism for this increase by determining the kinetics of Na^+/H^+ exchange in BLMV prepared from neonatal and adult liver by a Percoll gradient method. The initial rate of $^{22}Na^+$ uptake (7 sec) was assayed in the presence of an outwardly directed transmembrane proton gradient (pH 5.5 inside, pH 7.5 outside) using a rapid-quench Millipore filtration technique. Total Na^+ uptake measured as a function of extravesicular $[Na^+]$ (0.5 to 30 mM) was curvilinear and significantly higher in neonatal compared with adult BLMV. The passive component of uptake, estimated in the presence of the inhibitor amiloride, increased linearly with the $[Na^+]$ concentration and was similar at each age. Computer analysis of the amiloride-sensitive portion of Na^+ uptake (via Na^+/H^+ exchange) revealed saturable kinetics with a similar Km (8.6 ± 1.6 vs. 8.1 ± 1.5 mM) but a 2.5-fold higher Vmax (8.06 ± 0.67 vs. 3.20 ± 0.40 nmol/mg protein/min, p< 0.01) in neonatal vs. adult vesicles. We conclude from these studies that enhanced Na^+/H^+ exchange activity in neonatal liver, which may be essential for regulation of cell proliferation and growth, results from an increased incorporation of functional carriers into the plasma membrane or an increased translocation rate of existing carriers.