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**JEJUNAL MORPHOLOGY AND DISACCHARIDASE ACTIVITIES IN CHILDREN WITH CHRONIC DIARRHEA.** R. Calvin, W. Klish, C. Garza, G. Daoud, B. Nichols. Dept. of Pediatrics, Baylor College of Medicine, Houston, TX 77030.

With the hypothesis that mucosal enzymes are a reflection of villous height, an investigation was carried out on 34 jejunal biopsy samples from children with mucosal injury. These were classified into 4 groups (B-E) according to villous to crypt ratio (V/C) and were compared with normal biopsies and those from cases of gluten sensitive enteropathy (GSE):

Variable	Group n=3 GSE	n=10 B	n=10 C	n=7 D	n=7 E <sup>1</sup>	n=48 Normal
V/C	<1.0	<1.0	1.0-2.0	2.0-4.0	>4.0	>4.0
Lactase	5.9 <sup>2</sup>	7.7	17.9	13.2	13.4	32.4
Sucrase	11.6	20.9	48.3	27.8	32.4	75.7
Maltase	33.7	128.5	161.1	177.0	179.9	265.5

1) Includes biopsies with enterocyte abnormalities; 2) Geometric means.

One way analysis of variance of the log transformed data indicated significant differences between groups. Significant differences between enzyme activities of the normal and all abnormal groups and between maltase activities of the GSE group and abnormal groups were found. All data from Groups C-E fell within the normal range.

The data suggest that GSE may be differentiated from other causes of mucosal injury on the basis of maltase activity, however, no significant differences in enzymes appear between groups divided according to villous height.

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**ZINC-BINDING LIGANDS IN DUODENAL JUICE AND SALIVA FROM NORMAL ADULTS AND ACRODERMATITIS ENTEROPATHICA PATIENTS.** Clare E. Casey, K. Michael Hambidge, Philip A. Walravens, Kenneth H. Neldner and Arnold Silverman. University of Colorado Medical Center, Department of Pediatrics, Denver.

Acrodermatitis enteropathica (AE) is an autosomal, recessively inherited disease, the pathogenesis of which is attributable to severe zinc deficiency. Before the introduction of oral Zn therapy some AE patients were treated effectively with breast milk. A zinc-binding ligand (ZBL) which may facilitate the absorption of Zn has been found in human milk (but not cow's milk) and in rat small intestine (Hurley et al., Proc. Nat. Acad. Sci., 1977). A similar ZBL may be present in human duodenal juice and an absence or defect in such a ligand could be one site of the molecular defect in AE. Samples of duodenal juice and mixed saliva from normal subjects were fractionated by gel chromatography on Bio-gel P-10. Most of the Zn eluted from the column in two peaks, one at the void volume and one at lower molecular weight. The second peak was further purified by electrophoresis on acrylamide gel and found to contain a ZBL similar in size to that in breast milk. Pancreatic juice and saliva from 3 AE patients also contained the ZBL. It is concluded that the ZBL is present in some cases of AE but it is not yet known if it is normal in quantity or quality. (Supported in part by NIH Postdoctoral Fellowship no. HD-07096 and USPHS Grant no. RO1-AM-12432.)

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**ZINC LEVELS IN CHILDREN WITH GROWTH RETARDATION.** M. Castro-Magana, P. J. Collipp, S. Y. Chen, S. Amin and V. T. Maddaiah. Nassau County Medical Center, SUNY, Stony Brook Health Sciences Center, Dept. of Pediatrics, East Meadow, New York 11554.

Concentration of zinc in hair and serum of children with short stature (familial or constitutional) was measured by atomic absorption spectrophotometry. Some of them were treated with methyltestosterone (MT), 10 mg. daily.

	Hair (µg/g)	Serum (µg %)
Untreated	161.8 ± 41.3 (37)	86.9 ± 18.4 (42)
Treated	197.0 ± 47.3 (19)	101.6 ± 19.1 (21)
"p" value	0.02	0.005

Our Zn values were similar to those reported for normal children. Nevertheless, children receiving MT had higher Zn levels than the children without medication. Since Zn deficiency is associated with growth retardation, these results raise the question of whether increased Zn retention may be one of the mechanisms by which androgens accelerate growth. We were unable to find any significant difference between the Zn levels from children with familial short stature and those from children with constitutional growth delay.

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**FATTY ACIDS AND PROSTAGLANDINS (PG) IN CHILDREN WITH CYSTIC FIBROSIS (CF).** H. Peter Chase and Jacqueline Dupont, Univ. of Colo., Dept. of Ped., Denver and Colo. St. Univ., Dept. of Food Sci. & Nutr., Fort Collins, Colo.

Fatty acids (FA) in plasma and red blood cells (rbc) have been measured once monthly and PG's every 3 months, over an 18 month period in 10 children with CF. Infusions of Intralipid (group A) or glucose (group B) were given every 2 weeks in the first 12 mos and oral linoleic acid (to group B) or a placebo (to group A) in the final 6 months. The percent of linoleic acid in plasma and rbc's was initially lower in the CF compared to control children without CF (p<.01) as was rbc arachidonic acid (5.7 + 3.8 vs 19.8 ± 1.7, p<.001). At the end of the 12 mos of IV infusions, there were no significant differences between fatty acid levels in children receiving Intralipid compared to glucose, with both groups improving in rbc linoleic and arachidonic acid levels.

The rate of PGE<sub>2</sub> production in whole blood incubated at 37°C for 60 mins was significantly higher in all 10 children with CF (2.83 ± 2.11) compared to normal controls (1.85 ± 1.21) (p<.02) during the first year. At 18 mos, PGE<sub>2</sub> levels in CF children receiving oral linoleic acid were similar to controls, but were even higher (6.13 ± 2.92) in the CF children not receiving linoleic acid (p<.01). Production of PGE<sub>1</sub> and PGE<sub>2</sub> were similar in the controls and the children with CF. PGE<sub>2</sub> levels are associated with bronchoconstriction and the elevated levels or altered PG ratios may be associated with the pathophysiology of CF.

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**USE OF HYDRAULIC CAPILLARY INFUSION SYSTEM IN STUDY OF ESOPHAGEAL MOTILITY IN CHILDREN.** Dennis L. Christie, Donald V. Mack (spon. by Ronald J. Lemire), The Mason Clinic, University of Washington & Children's Orthopedic Hospital, Seattle.

One hundred sixty-one esophageal manometric studies were performed on 106 pts using infusion system previously described for adults. (1) Flow rate was 0.6 cc/min. Three fused esophageal catheters with outside diameter of 3.1 mm and internal diameter of 0.8 mm/catheter were used. Sixty-three pts had gastroesophageal reflux, 29 normal, 14 other esophageal diseases. Amplitude (A) and duration (D) of peristaltic contractions and velocity (V) of contraction wave down esophagus were determined on 13 pts without identifiable esophageal disease (6 < 2 yrs, 7 > 2 yrs). Data expressed as mean ± SD.

ESOPHAGUS	upper 1/3	middle 1/3	lower 1/3
Amplitude (mm Hg)			
<2 yrs	71.7 ± 22.06	79.8 ± 18.60	56.5 ± 17.50
>2 yrs	83.6 ± 11.80	85.3 ± 22.33	71.4 ± 23.75
Duration (sec)			
<2 yrs	3.3 ± 1.09	3.3 ± 1.00	2.8 ± 0.77
>2 yrs	3.0 ± -.76	2.9 ± 0.66	3.3 ± 1.37
Wave Speed (cm/sec)			
<2 yrs	----	2.4 ± 0.54	----
>2 yrs	----	2.7 ± 0.81	----

Conclusions: (1) No difference in A, D or V in pts <2 or >2 yrs. (2) Infusion system allows for use of low flow rates and small catheter size. (3) Abnormalities in peristalsis can be quantitated in pediatric patients. (1) Arndorfer RC, Gastro 73, 1977.

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**USE OF INTRAOPERATIVE ESOPHAGEAL MANOMETRICS IN SURGICAL TREATMENT OF GASTROESOPHAGEAL REFLUX IN PEDIATRIC PATIENTS.** Dennis L. Christie, Donald V. Mack (spon. by Ronald J. Lemire). The Mason Clinic, University of Washington & Children's Orthopedic Hospital, Seattle.

Ten consecutive pts (7 < 2 yrs) had Nissen fundoplication performed because of gastroesophageal reflux (GER). Indications for surgery were (1) stricture-2; (2) failure to thrive 2<sup>o</sup> to vomiting-3; (3) chronic esophagitis-2; (4) recurrent pneumonia-2; (5) respiratory arrest-1. All had GER demonstrated by positive pH reflux test. Esophageal motility was performed using catheters infused with water by hydraulic capillary infusion system. Measurements of lower esophageal sphincter pressure (LESP) and length (LESL) were obtained before, intraoperatively and after surgery.

TABLE I (mean ± SEM)	LESP (mm Hg)	LESL (cm)
Preoperative	15.6 ± 2.64	1.3 ± 0.13
Anesthesia, post-induction	14.7 ± 2.20	----
Pre-incision		
Anesthesia, pre-repair	9.8 ± 1.51	----
Incision open		
Anesthesia, post-repair	32.8 ± 2.68	2.7 ± 0.16
Incision open		
Postoperative, 1 week	26.4 ± 1.74	2.8 ± 0.26

Conclusions: (1) All pts were relieved of symptoms post-fundoplication. (2) Postoperative LESP and LESL significantly greater than preoperative LESP (p<0.01) and LESL (p<0.001). (3) There was significant difference between pre-repair LESP compared to post repair LESP (p<0.001).