519 PROTEIN SPARING MODIFIED FAST IN OBESE ADOLESCENTS: LONG TERM EFFECT ON LEAN BODY MASS. Marilyn R. Brown, Gilbert B. Forbes, William J. Klish, Anita Gordon, Mary Ann Campbell, University of Rochester School of Medicine, Strong Memorial Hospital, Department of Pediatrics, Rochester, NY.

Protein sparing modified fasts have been studied in adults, but little data exist for adolescents. This study was designed to evaluate a high protein liquid (HPL) diet (Optifast-70^{R}, Delmark) in excessively overweight adolescents (>175% IBW). 7 subjects ages 14-18 were studied for 5 weeks as inpatients, and 4 for another 4-6 months as outpatients. After 1 week on a 1000 calorie mixed diet they were given HPL diet in amounts providing 7cal/kg/IBW and protein 1.5gm/kg/IBW. All patients tolerated the diet well and lost considerable amounts of weight. Frequent 24 hour ECG monitoring showed no significant abnormalities. Resting metabolic rate decreased 20% over the first 5 weeks and a further 5-10% over the next months. Nitrogen balance during the first 5 weeks was +1.65±2.45gN/d (SD). Lean body mass (K-40 counting) decreased during the first month and then levelled off, whereas body fat continued to decrease.

		Δ Kg($\bar{x}$±SD)		% of wgt Δ due to:	
	n	LBM	FAT	LBM	FAT
Initial month	7	-3.3±1.5	-3.6±2.7	48	52
Per mon thereafter	4	-.48±.27	-3.8±1.0	11	89

These data suggest that the high protein low calorie diet as used in this study spares lean body mass and maintains nitrogen balance in the excessively obese adolescent.

520 FOLLOW-UP OF ESOPHAGEAL FUNCTION IN REPAIRED ESOPHAGEAL ATRESIA. Marilyn R. Brown, Richard A. Lawrence, Mary Ann Campbell, Thomas C. Putnam, William J. Klish, Beverly P. Wood, Robert W. Emmens, (Spon. by Gilbert B. Forbes), University of Rochester School of Medicine, Strong Memorial Hospital, Departments of Pediatrics, Surgery, Radiology, Rochester, NY.

Recent studies in patients with repair of esophageal atresia and tracheoesophageal fistula show a high incidence of significant abnormalities of esophageal function. We have studied 20 patients operated on over the past 12 years. Ages ranged from 4 months to 12 years (av. 5.9 yrs). Studies included history, esophageal manometry, pH probe acid reflux test (Tuttle) and barium esophagram.

The only symptom present was occasional dysphagia for solid foods in 5 patients. Three had had a coin removed and 4 had required dilatation. Seven patients had a history of pneumonia.

Only 1 patient had a lower esophageal sphincter pressure less than 12mm Hg. In 5, peristalsis was abnormal by manometry and in 5 by esophagram. However, only 1 patient was in both groups. Minimal reflux was seen in 5 patients by esophagram and in 6 by Tuttle test. Only 1 patient was in both groups. As the patients became older, symptoms decreased.

This study reveals fewer severe abnormalities than previously described. Minimal reflux seems to occur frequently without sequellae and most problems in our patients were related to esophageal narrowing requiring dilatation.

521 INCREASED LACTASE ACTIVITY IN SUCKLING MICE TREATED WITH CORTISONE OR INSULIN. Sergio A. Bustamante, Robert Lindberg, and Otakar Koldovsky (Spon. by Vincent A. Fulginiti). University of Arizona, College of Medicine, Depts. of Pediatrics and Physiology, Tucson, Arizona.

Injection of cortisone is known to induce sucrase activity precociously in suckling rodents. A recent report (Develop. Biol. 69:661) indicated a similar effect of insulin in mice. To study the mechanism of this insulin induction, we have attempted to reproduce the experiments reported, using Swiss ICR mice and injecting insulin daily (12.5 mU/g BW/day, s.c.) starting on day 8 and sacrificing 24 and 72 hours later. No precocious induction of sucrase was observed, but interesting effect on lactase was seen. These data as well as data on littermates treated with cortisone acetate (25 µg/g BW/day) are summarized below [only values for midjejunum are given, similar results were observed in proximal jejunum and ileum; lactase activity (µmoles/60 min) is given per mg of protein (SA), or per segment (TA); mean ± SEM (N). Top: 24 hours, lower line: 72 hours].

CONTROLS		INSULIN		CORTISONE	
SA	TA	SA	TA	SA	TA
2.2±.21	12.6±.87(10)	2.5±.19*	15.7±1.2(11)	3.4±.08	20.7±.85(9)
3.6±.24	26.3±2 (6)	4.7±.23	34.8±2.4(9)	6.5±.40	41.7±2.5(8)

Treatment either with cortisone or insulin evoked significant increase of lactase (SA and TA) (exception *). Further studies have to elucidate the mechanism of action of these hormones on lactase; a possibility exists that the insulin effect is secondary due to the adrenal cortex stimulation by hypoglycemic stress.

522 QUANTITATIVE FECAL ALPHA-1-ANTITRYPSIN TO MEASURE GASTROINTESTINAL PROTEIN LOSS. Helen L. Butler, J. Nevin Isenberg, J. Scott Somerset, Donald R. Barnett (Spon. by Geraldine K. Powell), University of Texas Medical Branch Hospitals, Depts. of Pediatric & Human Biological Chem. & Genetics, Galveston, Texas.

Excessive protein loss through the gastrointestinal tract is difficult to diagnose and quantitate because of the nonavailability of radiolabelled proteins and the disadvantages of their administration to children. Alpha-1-antitrypsin (α_1AT) is an endogenous protein which is resistant to proteolytic digestion in the intestine, not found in the diet and as suggested by others should reflect plasma protein loss into the GI tract. We investigated its usefulness by measuring the fecal α_1AT concentration in 24 patients with no malabsorption, 12 patients with cystic fibrosis (high fecal nitrogen), 10 patients with fat malabsorption only and 3 patients with protein-losing enteropathies. Stool aliquots were taken from 48-72 hour homogenized stool collections which had been evaluated for fecal fat. Five lambda samples were directly applied to and quantitated by radial immunodiffusion plates specific for anti-human α_1AT (Boehringer-Mannheim). "Normal" daily α_1AT loss expressed in mg/kg body wt/day was 1.70 ± 1.32 with similar values for cystic fibrosis (0.99 ± 0.68, $p>0.05$) and isolated fat malabsorption (1.51 ± 1.03, $p>0.05$). The α_1AT loss for the 3 patients with protein-losing enteropathies (two with lymphangectasia, one undiagnosed) varied from 4.4-48 mg/kg per day and correlated with clinical criteria for protein loss. Conclusion: Fecal α_1AT quantitation may be specific for measuring protein loss into the gastrointestinal tract.

523 GASTRIC ACID SECRETION IN CHILDREN: A COMPARISON BETWEEN NORMALS AND THOSE WITH PEPTIC ULCER DISEASE (PUD). William J. Byrne, Arthur R. Euler, Arkansas Children's Hospital, Little Rock, Arkansas.

Basal acid output (BAO) and pentagastrin (6 µg/kg SC) stimulated maximal acid output (MAO) were measured in 42 normal (NL) children (age range 2-144 mo; $\bar{x}$ = 19.4 mo). Similar measurements were made in 16 patients with PUD (age range 3-216 mo; $\bar{x}$ = 96.6 mo) including 7 evaluated both before and after 6 weeks of therapy. BAO and MAO were calculated by conventional technique with hydrogen ion concentration determined by titration to pH 7.0. The BAO and MAO in the NL children were ($\bar{x}$ ± SEM) 0.17 ± 0.22 meq/kg/hr and 1.38 ± 0.61 meq/kg/hr respectively. Although older heavier children produced more total acid than younger smaller ones, on a per kg basis no correlation was found between acid output (BAO/MAO) and increasing weight. Neither was there a correlation found between acid output (BAO/MAO; meq/kg/hr) and either height, age or sex. There was no significant difference between either the BAO (0.08 ± 0.02 meq/kg/hr) or the MAO (0.90 ± 0.14 meq/kg/hr) in the PUD patients and the values obtained in NL children ($p > 0.05$). There was also no significant difference between the groups for either BAO or MAO when comparisons were made on a meq/M^2/hr basis. Pre and post treatment BAO and MAO values in PUD patients were not statistically different ($p > 0.5$). In conclusion: 1) Since neither the BAO or the MAO in children correlated with weight, height, age or sex, gastric acid secretory data should be interpreted on a meq/kg/hr basis. 2) Gastric acid hypersecretion was not found in children with PUD either before or after treatment.

524 AN EVALUATION OF THE PARENTERAL MAGNESIUM (Mg) LOAD TEST IN WEANLING RATS FED FIVE LEVELS OF Mg. Joan L. Caddell (Spon. Thomas Aceto, Jr.), Dept. Pediat.-Adolesc. Med., St. Louis U. Sch. Med. and Cardinal Glennon Hosp., St. Louis, Mo.

A test is needed to evaluate the adequacy of a mammal's nutritional status of Mg during life. A normal kidney avidly retains Mg during deficiency and clears excessively high blood levels, but renal handling of intermediate levels is not known. To test the reliability of a parenteral Mg load test, groups of weanling (40.3 ±0.4 g) rats were fed purified diets containing 20% casein with five levels of Mg (Table) for one week. The rats were then tested using an intraperitoneal dose of 15 mg of magnesium/kg body weight (Fed. Proc. 39: Abst. 4086, 1980). Analyses for Mg were made using an atomic absorption spectrophotometer.

DIETARY Mg mg/100g (N=3)	WT GAIN g/1 wk	PLASMA Mg mg/100 ml	URINARY Mg mg/6 hr pre	FEMUR Mg mg/kg dry	%RETENTION Mg Load
A* 1.9±0.0	22.5±2**	0.36±0.03	21.4± 5	1226±26	86.9±1.9
B* 10.9±0.0	36.1±2	0.73±0.04	32.6± 9	2707±54	46.3±8.0
C* 15.8±0.6	43.5±2	0.92±0.06	24.8± 4	1959±77	48.5±1.1
D* 41.6±0.3	40.5±1	1.98±0.04	166.2±14	3237±48	12.0±3.9
EΨ 150.7±1.0	43.4±1	2.18±0.06	1084.3±59	3549±105	4.8±2.5

*N=6, ΨN=12, ** Mean±SEM. Analysis of variance testing for C,D,E showed NS for weight gain, but $P<0.001$ for other parameters. The test appears to be valid, but is not as exact a guide to the Mg-nutritional status as data found by chemical analysis of bone. The recommended dietary level of Mg for growing rats, 40 mg/100 g diet, appears to be suboptimal. Support: Life Seekers; SLU Dept Ped.-Adolesc. Med; Cardinal Glennon Hosp. for Children Research Fnd.