GASTROENTEROLOGY & NUTRITION

558

DELAYED IRON SUPPLEMENTATION IN PRETERM INFANTS.

Soraya Abbasi, Vinod K. Bhutani, Elizabeth Fong, Rosemary Dworanczyk, & Lois Johnson. Univ. Penn. Sch. Med. Pennsylvania Hosp., Sect. Newborn Pediatrics, Phila., PA.

Preterm infants, at risk for retinopathy of prematurity, need to be maintained Vitamin E sufficient (range: 0.8 to 1.2 U/ml) with appropriate Vitamin E supplements. Since iron is a potent pro-oxidant and free radical donor, it effectively increases the need for Vitamin E. Based on these considerations, iron supplementation has been delayed in our ICN until retinal vasculature matures. The iron status and need for supplementation was evaluated in 50 growing preterm infants (mean $\pm$ SD birthweight = 1019 $\pm$ 23.8 gm, range: 650-1370 gm). The only presumable source of Iron was packed red blood transfusion administered during the ICN stay (total mean blood received $\pm$ SD: 374 $\pm$ 255 ml). Serum Iron (SI) and transferrin saturation (SAT) were determined at birth and biweekly until eyes were mature. Mean $\pm$ SD values at 8, 10, 12 wks postnatal age appear below:

	8 wks	10 wks	12 wks
Serum Iron (mg %)	87.7 $\pm$ 27.7	37.7 $\pm$ 22.7	99.6 $\pm$ 25.5
SAT (%)	45.5 $\pm$ 15.3	45.7 $\pm$ 16.8	46.6 $\pm$ 13.6

These data indicate that infants in the ICN remain iron sufficient and the use of iron containing formulas may be delayed. In the occasional infant who requires little or no blood and therefore does not add to his iron stores in the nursery, the delay in providing an iron supplement can be compensated for by prescribing Poly-visol with Iron. A dosage of 1 ml/day for one month provides about as much iron as a daily intake of 24 oz. of iron-enriched formula over a six week period.

559

PROTEIN AND AMINO ACID CONTENT OF PRETERM (PT) AND FULLTERM (FT) MILK ARE THE SAME. Eugene W. Adcock and Eileen D. Brewer. Univ. of Tx. Med. Sch. at

Houston, Dept. of Pediatrics, Houston, Tx.

To determine if differences exist in the bioavailability of nitrogen-containing compounds in PT and FT milk, we measured both protein (micro-Kjeldahl) and amino acid (automated amino acid analyzer) content in serial samples collected during the first month of lactation. 149 samples of FT milk from 80 mothers were analyzed to construct a 95% normal limits plot for protein content (Fig.). 136 samples of PT milk from 48 mothers were superimposed on this FT plot. Analogous 95% normal limits and superimposition plots were made for amino acid content. Both protein and amino acid content of the PT samples fell within the FT 95% normal limits plot.

CONCLUSIONS:

- 1) FT milk has wide biological variation.
- 2) PT milk has similar wide variation.
- 3) PT and FT milk have the same nitrogen bioavailability.

560

CONTINUOUS PH MONITORING OF THE DISTAL ESOPHAGUS (CPHM); A COMPARISON OF 8 AND 12 HOUR VERSUS 18-24 HOUR VALUES. Nasrin O. Akbarnia, Kathleen B. Schwarz

(Spon. by Thomas Aceto, Jr.) St. Louis University School of Medicine, Cardinal Glennon Memorial Hospital for Children, Dept. of Peds., St. Louis, MO.

Most investigators recommend a period of 18-24 hours for CPHM for the diagnosis of gastroesophageal reflux (GER). The purpose of this study was to determine if a shorter period of CPHM would be a valid reflection of an 18-24 hour study. 20 patients (age 4 weeks-3 years) had CPHM for 21-24 hours. The method and scoring were according to Jolley, S.G., et al (Surgery 84:16, 1978). Scores at 8, 12 and 21-24 hours are presented below. In this scoring system a score of ≥ 64 = positive GER.

	8 vs 24 hr (n)		12 vs 24 hr (n)	
Scores agree				
+GER	277+47	218+30 (13)	261+33	194+30 (14)
-GER	61	46 (1)	51+1	47+1 (2)
Scores disagree				
+GER 24 h	17+8	91+11 (4)	31+8	86+21 (2)
-GER 24 h	68+3	55+8 (2)	71	64 (1)

When short duration scores were high (> 90 for 8 hours, > 114 for 12 hours) 100% of the 24 hour scores were diagnostic of GER.

Conclusion: Our data suggest that CPHM for only 8 hours is a valid reflection of the 24 hour pattern in those patients with high scores. If the score is below 90 the test should probably be repeated for more extended time.

561

APNEA: DOES LARYNGEAL MICROASPIRATION PLAY A ROLE? Steven M. Altschuler, John T. Boyle, Todd E. Nixon. (Spon. by John B. Watkins) Univ. of Penn. School

of Med., Children's Hosp. of Philadelphia, Dept. Peds., Phila., PA

To examine physiologic mechanisms whereby gastroesophageal reflux might cause apnea, we examined the breathing pattern and airway response after intralaryngeal infusion of 0.1 and 0.5cc each of saline, cow's milk formula, and 0.1N HCl in 9 adult cats. Anesthetized animals breathed spontaneously through a tracheostomy tube. A second tracheal cannula directed rostrally allowed direct stimulation of the larynx from below. Pulmonary function was determined from simultaneous measurements of intrathoracic pressure by intrapleural catheter, air flow by pneumotachograph, and lung volume by flow integration. Results: 1) None of the stimuli was followed by apnea defined as cessation of effective tidal volume $\times$ 15sec. 2) A cough response was elicited in 6 of 9 animals following 0.1N HCl only. 3) A short latent, rapidly adapting (20sec) decrease in respiratory rate from control (25.9 $\pm$ 4.9 br/min) was noted following milk (19.4 $\pm$ 3.7 br/min) and acid (13.5 $\pm$ 1.2 br/min) infusion ($p < .01$) despite continued presence of stimuli in the larynx at both volumes tested. 4) 0.1N HCl alone produced a short latent, rapidly adapting (20sec) increase in total lung resistance (47.6 $\pm$ 10.6 cm H₂O/L/sec vs control of 23.3 $\pm$ 1.7, $p < .01$). 5) Pretreatment of laryngeal mucosa with Benzocaine abolished the response to all stimuli.

CONCLUSIONS: 1) Apnea is not the major response following laryngeal aspiration. 2) Aspiration of microvolumes of acid produces a more significant pulmonary response than milk aspiration. 3) Responses to both stimuli are mediated by rapidly adapting mucosal receptors. 4) Thus, in infants with apnea, combined respiratory and intraesophageal monitoring should include recording of respiratory rate and cough.

562

MANNAN PREVENTS THE IN VITRO TOXICITY OF GLIADIN PEPTIDES (gp) ON CULTURES OF CELIAC MUCOSA. Salvatore

Auricchio, Giorgio de Ritis, Massimo De Vincenzi, Elena Mancini, Maurizio Minetti, Orazio Sapora, Vittorio Silano. (Spon. by F. Sereni). University of Naples, II Faculty of Medicine, Dept. of Clinical Pediatrics, Naples. Istituto Superiore di Sanità, Dept. of Comparative Toxicology and Ecotoxicology and Dept. of Cell Biology, Rome, Italy.

We have demonstrated that the lectin-like activity of prolamins peptides from cereals toxic for celiac patients is inhibited by N-acetylglucosamine, mannose and mannan. Peptic-tryptic-pancreatic gp prevented the recovery of enterocytes which occurred when atrophic mucosa of celiacs is cultured in

ENTEROCYTE HEIGHT (mean $\pm$ SD)

Uncultured	After 48 h culture		
	Without gp	With gp (0.5mg/ml) alone	+mannan (5 mg/ml)
17.50	20.16°	17.57	19.04°
+0.52	+1.97	+0.41	+1.36
(n= 10)	(n= 4)	(n= 6)	(n= 10)

°difference from uncultured mucosa $p < 0.01$

the absence of toxic peptides. Mannan allowed a significant improvement of celiac mucosa cultured with gp. Mannan is therefore able to detoxify gp in this in vitro model of celiac disease.

563

LECTIN-LIKE ACTIVITY OF CEREAL PROLAMIN-DERIVED PEPTIDES. Salvatore Auricchio, Giorgio de Ritis, Massimo

De Vincenzi, Elena Mancini, Maurizio Minetti, Orazio Sapora, Vittorio Silano. (Spon. by F. Sereni). University of Naples, II Faculty of Medicine, Dept. of Clinical Pediatrics, Naples. Istituto Superiore di Sanità, Dept. of Comparative Toxicology and Ecotoxicology and Dept. of Cell Biology, Rome, Italy.

It has been suggested that cereal peptides may damage the celiac intestinal mucosa by a cytotoxic action on immature or immature-like enterocytes (Auricchio et al. Ped. Res. In press). Peptic-tryptic peptides (PTP) of prolamins from bread wheats, rye, barley and oats and of bread wheat A-gliadin (gift of Dr. D. Kasarda) were very active in agglutinating undifferentiated in vitro cultured K 562 S cells (Lozzio et al. Blood 45, 321, 1975). The minimal concentrations required to agglutinate 30% of total cells (MAC) were: 3.2mg/l of PTP of wheat gliadins, 0.3mg/l of PTP of A-gliadin, 4mg/l of concanavalin A and 20mg/l of wheat germ agglutinin. The MAC of PTP of wheat gliadins was much higher (412mg/l) when tested on erythroid differentiated K 562 S cells. The agglutinating activity was inhibited by N-acetylglucosamine, mannose and mannan and was reversed by mannan. PTP of rice and maize prolamins did not agglutinate undifferentiated K 562 S cells. There is therefore a good correlation between toxicity for celiacs of cereal prolamins and their lectin-like activity on undifferentiated cells.