53 COLONIC WATER ABSORPTION IS IMPAIRED DURING SMALL INTESTINAL SECRETION DUE TO CHOLERA TOXIN (CT)

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The effects of CT on small intestinal transport of water and electrolytes are well characterized. However, it is not known whether ion and water transport are altered in the colon when CT is present only in the small intestine. Aiming at defining the role of the colon in handling of water and electrolyte transport in such circumstances, we have measured water and ion transport in vivo in rat jejunal and colonic separate loops. Two groups of rats were investigated: in one, CT (1 ug/ml) was instilled in the jejunal loop two hours before separate perfusion of both loops for the ensuing 3 hours with a modified Ringer solution; the other (control) was likewise handled, but without any previous instillation of CT.
Results: In CT animals, water transport in the jejunum was significantly ($p<0.01$) shifted toward secretion (-0.77±2.0 ul/min g wet weight vs. 13.19±1.9 in the controls). Similarly, in CT animals, the simultaneously measured water transport to the colon was also significantly ($p<0.01$) shifted toward secretion (0.6±2,4 vs. 6.7±1.6 in the controls). Na net transport paralleled these changes.
In conclusion: our data shows that the colon contributes to the overall intestinal secretion seen in cholera infection, even when CT does not interact with its mucosa. It remains to be seen whether such observation relates only to CT-elicited small intestinal secretion or it represents a general pattern linking small and large intestinal transport.

54 NONINVASIVE URINARY INDICATORS OF CATABOLISM INVESTIGATED BY EXPERIMENTAL FOOD DEFICIENCY IN RATS

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We aim at measuring whole-body metabolic activity, especially metabolic imbalance caused e.g. by hunger (Schöch et al, ESPGAN 1988, Ped Res 24: 419). Particularly sensitive indicators of metabolic stress are the protein turnover marker 3-methylhistidine (m^3His; from actin and myosin) and the mRNA turnover marker 7-methylguanine (m^7Gua), both of which are quantitatively excreted in urine and measured by HPLC. Our results with young rats (Table) show that m^3His excretion rises from the first day of food restriction (negative r values), whereas m^7Gua excretion drops (positive r values) after a transient increase.

Table: Correlations between energy intake (MJ/kg BW/d), excretion of m^3His and m^7Gua (µmol/mmol creatinine) and N retention in young rats (n = 11 - 15; 32 d old on 1st day of experiment). Food quantity was varied in 3 steps (0.6 - 2.2 MJ/kg/d).

Parameter correlated to energy intake	Day of experiment and r values (* $P < 0.01$, ** $P \leq 0.001$)				
	1	2	3	4	5
m^3His	-0.642	-0.882**	-0.716*	-0.815**	-0.439
m^7Gua	-0.661*	0.771**	0.735**	0.915**	0.833**
N retention	0.972**	0.988**	0.984**	0.984**	0.989**

We conclude that in food deficiency muscle protein is broken down to ensure continued synthesis of vital proteins; concomitantly, after a transient breakdown of mRNA leading to increased m^7Gua excretion whole-body mRNA turnover is slowed down. Our results demonstrate that m^3His and m^7Gua excretion move in opposite directions with decreasing food intakes. Therefore increased m^3His/m^7Gua ratios, which can be measured in spot urine samples, may turn out to be a valuable noninvasive indicator of catabolism caused by food deficiency.

55 PERCUTANEOUS ENDOSCOPIC CONTROLLED GASTROSTOMY (PEG) FOR ALIMENTATION OF CHILDREN WITH CYSTIC FIBROSIS

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The benefit of a hyperalimentation in patients with cystic fibrosis is well known. Oral hyperalimentation is difficult to perform and gastric tubes are not well accepted by the patients. In a prospective study we investigated whether PEG might be an alternative in the application of hypercaloric diet. So far PEG was performed in 9 children aged from 7,2 to 17,9 years. In 8 patients we have a follow-up of at least 3 months. Those patients entered the study where there was 1. a percentage ideal weight for height (IWH) below 80% 2. and/or no improvement of the IWH above 3% despite optimal conventional nutrition in a 6 month period. PEG was performed under general anaesthesia according to the method of Keymling°. Except of two dislocations of the PEG-tube, which were easily corrected, we saw no complications. PEG was well accepted by the patients and their parents. In addition to normal nutrition the children received 1000 kcal bulkage-free diet over night. After 3 months there was a mean increase of IWH of 9% (3-12%), of triceps skin fold thickness of 2,91 mm (-0,3-6,9 mm) and of upper arm circumference of 1,88 cm (0,9-2,9 cm). From these data we conclude that PEG is a very effective and save procedure in the nutritional therapy in patients with cystic fibrosis.

°Keymling M, Schlee P, Wörner W. Die perkutane endoskopisch kontrollierte Gastrostomie. DMW 112:182(1987)

56 COMPARISON OF MILK (M) AND YOGURT (Y) IN CHILDREN WITH PERSISTENT DIARRHEA (PD)

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PD denotes episodes of diarrhoea that begin acutely, but persist beyond 15 days. Although the pathophysiology of PD remains unclear, it has been suggested to be related to the composition of the food ingested. The main objective of this study was to compare the clinical outcome in children with PD fed M or Y. The sample size calculated on the basis of 30% reduction in clinical failure (weight loss) 5% and intestinal intolerance) was 30 children with M and 26 children with Y. The 2 groups were ientical at randomisation (age = 8.2 months : duration of diarrhea = 20 days; number of stools = 6.3/day; weight/height = 85%). Following observations were made. 1) Clinical failure 13 (43%) in group M and only 3 (11%) in group Y ($p < 0.01$). 2) Weight gain was 44 + 48 g (x + SE) vs 152 + 95 ($p < 0.02$) in M and Y respectively. 3) Duration of diarrhea was 73 + 9hr in M and 40 + 8 in Y ($p < 0.05$); 4) liquid stool output was 40 + 8 in M vs 22 + 4 in Y. 5) Fluid intake was 62 + 7 ml/kg/d in M vs 39 + 5 in Y. 6) The amount of food intake including milk product intake was the same in oth groups i.e. 150 ml/kg/d in group M and 142 ml/kg/d in group Y. These results demonstrate a clinical advantage of feeding yogurt rather than milk in children with persistent diarrhea.

57 DEFECTIVE JEJUNAL BRUSH BORDER Na^+/H^+ EXCHANGE IN LETHAL FAMILIAL PROTRACTED DIARRHOEA

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The clinical spectrum of disease associated with the recently described defect in jejunal brush border membrane (BBM) Na^+/H^+ exchange remains poorly defined. We describe a further, lethal case of protracted diarrhoea in a child from a family in whom 2 previous siblings died of protracted diarrhoea at 2 and 11 months. The patient, a boy, was born at term weighing 2.9 kg and was admitted at 6d with profuse watery diarrhoea, severe dehydration and metabolic acidosis. Parenteral nutrition was started but the diarrhoea persisted, and he developed severe necrotising enterocolitis requiring an ileostomy. A high-output secretory diarrhoea persisted during nil by mouth (ileostomy fluid mmol/l: Na^+ 128; K^+ 10; Cl 96) and reanastomosis. Parenteral Na^+ requirement was high (6-10 mmol/kg/d). Extensive investigation of gastrointestinal function was non-contributory, but transport studies on a jejunal biopsy at 3 mo, which showed a mild partial villous atrophy but normal electron microscopy, confirmed a specific defect in Na^+/H^+ exchange. 15 sec Na^+-stimulated glucose uptake by jejunal BBM vesicles was normal (patient: 107 pmol/mg protein; control mean: 213 (SD 111), whereas 15 sec Na^+ uptake under H^+ - gradient conditions was negligible (patient: 9 pmol/mg protein; control mean: 150 (SD 48)); Na^+ uptakes at equilibrium (120 min) were normal. The patient died of severe intercurrent infection at 18 mo. These data therefore extend the spectrum of disorders characterised by a specific defect in Na^+/H^+ exchange.

58 INTESTINAL MICROFLORA OF NEWBORN INFANTS IN ADDIS ABEBA AND STOCKHOLM

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AIMS: To compare the aerobic and anaerobic intestinal microflora of newborn infants with and without antibiotic treatment and also to describe any relationships between presence of anaerobic bacteria and colonization by potentially pathogenic gramnegative aerobic bacteria.
METHODS: Fresh faecal specimens were obtained from 61 infants in Addis Abeba and 21 infants in Stockholm. All infants were breastfed. The specimens were frozen for 0-20 days, thawed and transported in anaerobic athmosphere to the laboratory in Stockholm.
RESULTS: In Stockholm, the dominating aerobe was S.epidermidis and the dominating anaerobe was Bacteriodes fragilis. Bifidobacteria were only rarely isolated. In Addis Abeba E.coli and lactobacilli dominated. Antibiotic treatment produced no consistent change of the intestinal microflora of Addis Abeba infants, as opposed to the dramatic impact seen in Sweden. There was an inverse relationship between colonization by potentially pathogenic Gramnegative aerobic bacteria and occurrence of bifidobacteria but not of lactobacilli.
CONCLUSIONS: The intestinal flora of breastfed newborn infants in Stockholm differs from what has previously been reported, whereas in Addis Abeba it is more "classical". Bifidobacteria, but not lactobacilli, might produce some resistance to colonization by Gramnegative bacteria from the hospital environment.