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Gastric emptying results from the co-ordinated contraction of antral, pyloric and duodenal smooth muscle. The rate of contraction is controlled by the electrical slow wave activity of smooth muscle cells which originates from a pacemaker area in the body of the stomach and controls the orderly aboral propagation of gastric contents. Such electrical control activity (ECA) can be detected by conventional mucosa or serosal electromyographic techniques but more recently attempts have been made to record ECA non invasively using bipolar skin Ag/Ag Cl electrodes. The resultant electrogastrogram (EGG) has a very low signal to noise ratio and requires a variety of signal processing methods to reveal the underlying signal. We have measured ECA frequency from a fasting EGG in control subjects and 5 patients with delayed gastric emptying due to chronic intestinal pseudo-obstruction (CIP). The EGG was recorded for 1 hour using low pass filtering at 0.33 Hz, digitalized at 1 Hz and stored on a floppy disc. The stored signal was then band pass filtered (0.01-0.25 Hz) and a running spectral analysis carried out using Zenith 248 Computer. In control subjects a consistent gastric ECA of 3 cycles per min (cpm) was found. In 4 of the patients with a myopathic CIP, ECA frequency varied between 1-6 cpm with time and no clear consistent dominant frequency could be seen. In 1 patient with a neuropathic CIP a persistent tachyarrhythmia at 6 cpm was seen. These data suggest that in smooth muscle disease ECA frequency wanders but alteration of the neurohumoral environment may result in an altered ECA frequency. Such brady or tachy-arrhythmias result in gastric atony and delayed gastric emptying and raise the question of whether anti dysrhythmic agent might be therapeutically more appropriate than conventional prokinetic drugs.

We evaluated GI motility by means of a perfused catheter system, DGR, as assessed by Bile Salts (BS) output in gastric aspirates, and GE of a milk labeled formula (% of emptying at 1 hr) in 11 patients (pts) with unexplained chronic vomiting (Apts), in 8 pts with protracted gastroesophageal reflux (GER) disease (Bpts) and in 7 symptomatic controls (Cpts). Mean \pm SD age (months) was respectively 44.2 \pm 37.7, 18.1 \pm 11.2, 20.4 \pm 14.4. In 9 Apts and 5 Bpts we found GI manometric abnormalities none of which were seen in Cpts: a) fasting and/or fed antral (and/or duodenal) hypomotility; b) abnormal propagation or configuration of interdigestive motor complexes (IMC); c) bursts of non propagated duodenal or jejunal motility; d) sustained fasting and/or fed phasic activity incoordinated with adjacent gut segments. Both A and B pts had BS fasting recovery significantly higher than Cpts during the various phases of IMC (mean group values (mg/ml): 1.52 (A), 1.12 (B), 0.36 (C), $p < 0.05$) and a significant delay of GE (A: 32.8 \pm 8.9%; B: 34.4 \pm 9.8%) as compared to Cpts (64.5 \pm 5.3%, $p < 0.05$, mean \pm SD). Highest degrees of gastric BS output and of delayed GE were associated with the most marked GI motility dysfunctions in both A and B pts. Conclusions: 1) children with chronic unexplained vomiting may exhibit, at GI manometry, disordered gut motility patterns; 2) the latter seem to be associated with increased DGR and delayed GE; 3) severe GER disease shows diffuse dysmotility of upper GI tract.

The introduction of feeds after correction of a midgut malrotation is usually uneventful but may be difficult in a small number of cases. The nature of the intra-uterine migration defect is unknown. We assessed small intestinal motor activity manometrically in five children (mean age 13.6 mths) with persistent feeding problems after correction at a mean time of 13.0 mths after surgery. Two groups were noted. Four children with persistent vomiting did not tolerate oral feeds. All of these had a second surgical procedure attempting to relieve an obstruction (2 a pyloromyotomy, 1 an ileocaecal resection, 1 an ileostomy) but continued to have symptoms. Three had abnormal small intestinal motility, one no activity, one slow Phase III pressure wave frequency with short Phase III duration, and one non-propagated Phase III complexes with a low motility index (MI).

Patient	PIII Freq. (cpm)	PIII dur. (min)	PIII MI	Non-prop.	Cycle length (min)
Control	12.3 \pm 0.5	5.3 \pm 2.1	161 \pm 116	-	77.7 \pm 39.8
1	13.05	3.14	52	+	30.3
2	0	0	0	-	—
3	9.27	2.43	299	-	32.1
4	12.76	3.75	172	-	52.7
5	10.86	4.96	87	-	43.6

One child (number 5) had severe diarrhoea after feeds due to a short bowel syndrome following an ileal resection after a volvulus. This child had a low motility index in Phase III. These data show that a pattern of motor activity similar to that seen in intestinal neuropathic disorders is present in these patients. We speculate that the malrotation is secondary to a neuropathic disorder present during early foetal development in some patients.

Colonic EMG has not yet been reported in children. 5 children (3 boys, 2 girls, mean age 5.3 \pm 3.4 y) with severe irritable bowel syndrome (IBS) underwent a colonic bipolar EMG with a probe with annular electrodes positioned in left colon by colonoscopy. After an overnight fast, a two hour pre- and post-prandial recording was performed (meal : 30 Kcal/kg). Results : the tracing showed the typical colonic patterns : Short Spike Bursts (SSB), Long Spike Bursts (LSB), Migrating Long Spike Bursts (MLSB). As in adults, different patterns were observed, associated with different clinical features of IBS :
- in one case with watery diarrhea, low electrical activity with reduced number of SSB during the preprandial period, and a normal postprandial increase of colonic motility.
- in two IBS with abdominal pains, intense preprandial spiking activity with repetitive LSB, and decreased activity after feeding.
- in two cases with alternate diarrhea and constipation the preprandial electrical activity was normal and the postprandial spiking activity was lowered. These preliminary results individualize several groups of IBS children. Colonic EMG may contribute to diagnostic and therapeutic progress in IBS, and other intestinal dysmotilities in children.

In healthy adults, plasma Mo and PP levels increase significantly during ICMA at the end of phase 2 (P2) and the onset of phase 3 (P3) as compared to phase 1 (P1). Our aim was to study the correlation between plasma Mo, PP and So levels variations and the ICMA 3 phases in children with CIPS (visceral neuropathies). 6 children (4 boys, 2 girls ; mean age : 22.6 months) were studied. Intestinal manometry was recorded in each child during 6 hours and blood samples were taken at P1, at the end of P2 and the onset of P3 for Mo, PP and So radio-immunoassay. In 5 children, ICMA P1, P2 and P3 appeared spontaneously and in one case a P3-like after trimebutine intravenous injection. Plasma levels (pg/ml) of Mo (274 \pm 137) and PP (45 \pm 17) at the end of P2 and at the onset of P3 : Mo (230 \pm 61) and PP (39 \pm 13) did not increase as compared to P1 values (Mo : 321 \pm 188 and PP : 49 \pm 31). Moreover, plasma Mo P1 levels were statistically higher and PP levels lower as compared to healthy adults values (n = 12, Mo : 157 \pm 46 ; PP : 132 \pm 45). Plasma So levels did not vary significantly during the ICMA phases. These results suggest that in CIPS, a dysregulation of hormonal control (Mo, PP) of ICMA exists. Plasma Mo and PP determination could be of interest for CIPS diagnosis.

Although changes in mucosal function are relatively well documented in the human newborn there are no corresponding data on gut blood flow velocities during the first week of life. Using duplex pulsed Doppler ultrasound we have therefore prospectively studied, averaged over 6 cardiac cycles (CC), the peak systolic velocities (FSV), area under the peak velocity envelope (AUPVE), and the time averaged mean velocity (TAMW) of blood in the coeliac axis (CA) and superior mesenteric artery (SMA) in 9 normal term babies (7 bottle and 2 breast fed). Subjects were studied daily, immediately before and for 2 hrs following a feed. The mean fasting FSV in the SMA rose from 56 (SD 24) cm/sec on day 1 to 80 (SD 24) cm/sec on day 2 ($p < 0.006$). There was no further significant rise in fasting velocities between days 2 and 7. In contrast there were no significant changes in the CA during this time.

Feeds led to an increase, peaking at 50 mins, in FSV, TAMW and AUPVE as shown:

	SMA		Vel at Max Inc	p < 0.0001
	Mean \pm SD	Pre-Feed		
FSV cm/sec	71 \pm 21	110 \pm 33		
TAMW cm/sec	33 \pm 11	52 \pm 16		p < 0.0001
AUPVE cm/CC	16 \pm 5	26 \pm 8		p < 0.001

Similar, but much attenuated, changes were seen in the CA suggesting that this increase in velocity is specific to gut perfusion and represents a physiological response to feeds.