GASTRITIS IN THE RECTUM: CAMPYLOBACTER-LIKE ORGANISMS IN HETEROTOPIC INFLAMED GASTRIC MUCOSA. D.J. Pambianco, K.R. Dye, B.J. Marshall, H.F. Frierson, R.H. MacMillan, D. Franquemont, R.W. McCallum. Department of Internal Medicine, Box 145 University of Virginia, Charlottesville, Va. 22908.

A 35 y.o. white female underwent colonoscopy for evaluation of abdominal pain and weight loss. The exam was normal except for a 1 cm irregular clump of pale tissue 10 cm from the anus. Histology showed body-type gastric mucosa with parietal cell glands and overlying mucus secreting epithelium. Junctional areas where gastric and normal rectal mucosa joined were present. An active (2+) chronic (2+) inflammatory reaction was histologically present in the gastric mucosa but the adjacent rectal mucosa was normal. Mucus-secreting epithelial cells overlying the gastric glands appeared normal. At high power, large numbers of curved and "~" shaped spiral bacteria, with identical morphology to Campylobacter pylori, were observed adhering to the gastric-type mucus secreting epithelium but not to the rectal epithelium.

This is the first report of C.pylori infection distal to the duodenal bulb. The apparent presence of this organism on heterotopic gastric mucosa in the rectum emphasizes the organism's ability to selectively colonize the gastric epithelial cell. Although C.pylori has never been cultured from feces, this case suggests that viable C.pylori organisms do reach the rectum and that the organism may be spread by fecal-oral contamination.

EFFECT OF THE GARREN GASTRIC BUBBLE ON SOLID & LIQUID GASTRIC EMPTYING IN MORBIDLY OBESE PATIENTS. D. Pambianco, M. Plankey, K. Fisher, R.W. McCallum. Dept. of Int. Med., Univ. of Virginia, Charlottesville, VA 22908.

The Garren Gastric Bubble (GGB) is an inflatable device (200 cc) that may promote weight loss by causing a sensation of satiety. We investigated the effects of GGB on solid (S) and liquid (L) phase gastric emptying (GE) in morbidly obese subjects. Method: 14 morbidly obese patients (13F, 1M), mean age 36 (24-46) were studied before and 3 months after GGB implantation. A dual isotope GE method was used employing chicken liver labeled with Tc-99m sulfur-colloid as the S phase marker, and In-111-labeled water as the L phase marker. Subjective clinical evaluation of satiety was completed bi-weekly in all subjects. Results: The percent of isotope for S and L retained in the stomach over 2 hrs. (mean±SEM) in the GGB patients compared to normal subjects is summarized below (*p<0.05 compared to normals).

Percent Liquids (L)

		30 min	60 min	90 min	120 min
Pre	(n=11)	24±10.9*	11.0±6.3*	5.8±4.0*	3.4±2.4
3 mos	(n=11)	35.4±15.9	17.7±9.9*	11.2±8.2*	7.8±7.6
Normals	(n=11)	39.7±14.2	22.2±8.9	11.9±8.0	5.1±4.6

Percent Solids (S)

		30 min	60 min	90 min	120 min
Pre	(n=14)	90.5±10.4	73.1±18.7	57.7±23.5	43.2±25.8
3 mos	(n=11)	87.0±8.7	74.6±13.5	63.6±16.6	49.7±17.9
Normals	(n=21)	90.9±7.6	77.1±15.4	62.0±17.1	46.3±14.0

Pre-bubble GE was faster for patients compared to normals reaching significance ($p<0.05$) for the L. 3 months after bubble insertion GE was slower compared to the initial baseline reaching significance for GE of L. 8 of 11 patients subjectively sensed satiety for the entire 3 months of the GGB combined with a structured behavior modification program. Conclusion: 1) Morbidly obese patients had faster baseline GE, compared to normal subjects; 2) when re-studied 3 months after GGB insertion their mean GE was slower, and 3) the slowing of GE accompanying GGB may contribute to any weight loss attributed to the bubble by producing increased satiety.

● IDENTIFICATION AND LOCALIZATION OF GTP-BINDING PROTEINS IN RABBIT ILEAL ENTEROCYTE MEMBRANES. R. Pamukcu and E.B. Chang, Dept. of Medicine, Univ. of Chicago, Chicago, IL.

Various electrolyte transport and other cellular processes in intestinal epithelia are regulated by neurohormones. Studies in other tissue systems have documented the role of GTP-binding membrane proteins as intermediaries in the signal transduction process initiated by the neurohormones. These proteins, heretofore, have been poorly characterized in intestinal mucosa. The objective of this study was to identify and localize GTP-binding proteins in various sub-cellular membrane components of the enterocyte.

Isolated enterocytes were obtained through a modified Weiser technique. Crude and enriched subcellular membrane fractions were achieved through differential density centrifugation and divalent cation precipitation. Enrichment and purity of apical (AP) and basolateral (BL) fractions were confirmed by assays of sucrase and K-stimulated phosphatase activities, respectively. The golgi/endoplasmic reticulum (GER) fraction was confirmed by assays of galactosyl transferase and arylesterase activities. GTP-binding proteins were identified by their electrophoretic migration on SDS-PAGE after their specific binding of an 8-azido-GTP photolabel or ADP-ribosylation (ADP-R) in the presence of pertussis (PT) and cholera toxins (CT).

ADP-R stimulated by CT revealed two major substrates of M_r 47 Kd and 43 Kd in the crude, AP, BL, and GER fractions. These substrates, which specifically bound the 8-azido-GTP photolabel, were presumed to be G_s. PT ADP-R revealed a 41 Kd substrate which also bound GTP^s in crude, BL, and GER fractions. This protein, presumably G_i, was absent in the AP fractions. However, the crude and AP fractions demonstrated a relatively weak PT substrate of 44 Kd. Freeze-thawing of the fractions yielded a prominent PT substrate at 39 Kd in all membrane components with the loss of the 41 Kd substrate. In addition, several other GTP-binding proteins were present which are not PT or CT substrates.

In summary, this study demonstrates the presence of several GTP-binding proteins, which may include G_s and G_i, in rabbit ileal enterocytes. The asymmetrical distribution of these proteins among the various membrane components suggests differences in their intracellular sorting.

● AZODISALICYLATE INDUCED SECRETION IN RABBIT ILEAL AND COLONIC MUCOSA. R Pamukcu, SB Hanauer, EB Chang, Section of Gastroenterology, University of Chicago, Chicago, IL.

Azodisalicylate (ADS), a dimer of 5-aminosalicylic acid (5-ASA) used to treat ulcerative colitis, causes diarrhea in ~4-13% of patients. To explore this further, the in vitro effects of ADS, sulfasalazine (SASP), and 5-ASA on rabbit intestinal electrolyte transport were compared.

Distal ileal mucosa mounted in Ussing chambers were exposed to varying concentrations of ADS, SASP, and 5-ASA. Maximal changes in short-circuit current (Isc), a measure of active anion secretion, were determined. Mucosal addition of ADS (>5mM) caused the greatest increase in Isc of 83uA/cm2 (ED50=0.3mM). Removing the Cl and/or HCO3 from the bathing medium significantly attenuated this effect. Isotope flux measurements under short-circuited conditions suggest that ADS stimulates electrogenic HCO3 (86%) and Cl (14%) secretion, and inhibits neutral NaCl absorption. Serosal addition of ADS and SASP (>5mM) producted much smaller effects; 5-ASA had no secretory effects. ADS did not affect ion transport in distal colon. To explore possible mechanisms of action, cyclic AMP and cyclic GMP levels in ileal mucosa were measured before and after addition of ADS (5mM). No significant differences could be detected. Similarly, the response to ADS was unaffected by the cyclooxygenase inhibitor piroxicam.

In conclusion, ADS is a potent and unique secretagogue in distal ileum at clinically relevant intraluminal levels. These findings may account for the diarrhea seen with this drug.