● **BARIUM VS NUCLEAR SCINTIGRAPHY: WHICH IS BETTER FOR EVALUATING PATIENTS WITH ACHALASIA?**

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The treatment end-point of achalasia is usually based on symptoms improvement without objective data. More recently, post-treatment LES pressure < 10 mmHg was associated with a good response to treatment (Gastro 1992;103:1732). However, patients are often unaccepting of repeat manometry, and passing a catheter across the LES may be techniquely difficult. Therefore, other physiological studies are needed to evaluate the severity of disease and treatment response. **Aim:** We compared symptoms of untreated/treated achalasia patients to upright esophageal retention measured by barium swallow (liquid) and nuclear scintigraphy (solid). **Methods:** Pre- and post-treatment studies performed on 13 patients (7M, x age 68) as part of protocol comparing pneumatic dilatation to botulinum injection. Each patient underwent between one and three studies. Patients evaluated with: symptom score (SxScore) (scale: 0-15); upright barium column height (Ba C Ht) at 5 min after sufficient contrast given to show first evidence of esophageal emptying; and % upright esophageal retention of solid radiolabeled meal at 20 min by scintigraphy (NS-20). **Results:** Linear regression analysis.

Comparison	n	R	P
SxScore vs Ba C Ht (cm)	26	0.58	<0.01
SxScore vs NS-20 (% retention)	22	0.61	<0.01
Ba C Ht (cm) vs NS-20 (% retention)	22	0.79	<0.01

Ba. X-rays less expensive ($110) than nuclear scintigraphy ($628)

Conclusions: 1) Symptom scores show a significant relationship with both retained barium column height at 5 minutes (R=0.58) and % esophageal retention at 20 min by nuclear scintigraphy (R=0.61). 2) There is a strong correlation between these two physiological tests (R=0.79). 3) Considering the nearly 6 fold cost differential, test availability, and simplicity, we believe that upright barium retention studies should be routinely used to evaluate patients with achalasia pre- and post-treatment.

● **DIAGNOSIS OF *H. pylori* INFECTION BY THE LARA™ SYSTEM. Towards a simplified breath test.**

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INTRODUCTION: Noninvasive detection of *H. pylori* (HP) is accomplished either by serological methods or by urea breath tests (UBT). The main limitation of the former is the sustained antibody response that precludes assessment of eradication, while the latter is technically demanding and expensive. A novel methodology, LARA™, allows for a rapid and inexpensive UBT. The LARA™ system assesses expired stable isotopically labeled CO_2 by determining the $^{13}CO_2/^{12}CO_2$ ratios by laser optogalvanic spectroscopy. In this study, we compared the LARA™ to conventional invasive diagnosis of HP. **METHODS:** 103 pts (18-75 yo) were evaluated by UGI endoscopy with antral histology and CLO test, and also by the LARA™ UBT. For the UBT, 100 mg of ^{13}C urea were ingested by fasting pts, following the ingestion of a standardized meal (Ensure). Expired breath was sampled at baseline, 10, 20, 40, 60 and 90 min. Breath samples were collected in a proprietary device which retains mid-expiration air. $^{13}CO_2/^{12}CO_2$ ratios were assessed by LARA™. A UBT was considered positive when there was a change of $\geq$15 units in the 20 min or 60 min sample. **RESULTS:** Of the 103 pts, 69 had evidence of HP infection either by histology or by CLO test or by both. Of them, 68 were positive by the UBT and one (whose only the CLO test was positive) was negative; therefore the sensitivity of the test was 98.5%. 34 pts were negative for HP (both histology and CLO test negative for HP). Of them, 6 (18%) had a positive UBT and 28 (82%) a negative UBT. As it is documented that UBT is superior to either histology or CLO test or both, no specificity of the test can be calculated. **CONCLUSIONS:** These results demonstrate a) the feasibility of a non-radioctive, rapid UBT based on the LARA™ methodology, b) that this method has a 98.5% sensitivity, and that c) UBT was positive in 18% of the cases where both CLO and histology were negative.

● **THE REGULATION OF pH BY *HELICOBACTER PYLORI* USING THE FLUORESCENT pH INDICATOR DYE, BCECF.**

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The regulation of both external and internal pH by *H. pylori* allows it to survive in the acidic milieu of the stomach. The bacterial urease alkalinizes the environment of the bacterium as well as the cytoplasm by the production of NH_3 which converts to NH_4^+ extracellularly or intracellularly. Morphology of the organism was monitored using acridine orange fluorescence. The pH of the bacterial environment or cytoplasm was measured by incubating *H.pylori* with 5 μM of the fluorescent intracellular pH indicator, BCECF-AM, in a medium containing 130 mM NaCl, 5 mM KCl, 1.3 mM $CaCl_2$, 10 mM glucose and 1 mM glutamine in 1 mM phosphate buffer, pH 7.0. The bacterium fluoresced strongly after loading with the dye, showing the presence of an effective esterase in the organism. The dye appeared to be bound by or to be within the organism. Dual wavelength excitation in a fluorimeter or in a video microscope were used to measure the pH sensed by BCECF. The absence of strong buffer in the medium allowed determination of the pH_o in the vicinity of the organism. In medium with external pH clamped with strong buffer, changes in cytoplasmic pH_i could be determined. The addition of 3mM urea in low buffer resulted in a rapid and large alkalinization in the immediate environment of the organism, reflecting the local production of NH_3 and reaction wtih H^+. At high buffer concentrations (20 mM tris-HCl, pH 7.0), a smaller change in fluorescence was observed due to a change in pH_i showing the presence of intracellular urease activity or entry of NH_3. The addition of 60 mM NH_4Cl in this buffer to *H. pylori* resulted in a rapid intracellular alkalinisation due to NH_3 diffusion across the cytoplasmic membrane. The pH_i rapidly returned to baseline with a $t_{1/2}$ of <1 minute indicating active NH_4^+in/H^+out exchange. Although the pH_i of BCECF loaded *E. coli* also increased with addition of 60 mM NH_4Cl, recovery was very slow with a $t_{1/2}$ >10 min, as is also characteristic of mammalian cells, which are unable to rapdily extrude NH_4^+. It is concluded that (a) BCECF-AM is able to monitor pH regulation by *H. pylori* (b) both extracellular and perhaps intracellular urease are important in pH regulation and (c) H. pylori may regulate internal pH in part by active extrusion of internal NH_4^+ in exchange for external H^+ to counteract NH_3 induced cellular alkalinization.

● **DOSE-RESPONSE COMPARISON OF LANSOPRAZOLE AND OMEPRAZOLE ON 24-HOUR GASTRIC ACIDITY AND PLASMA GASTRIN IN HEALTHY VOLUNTEERS.**

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The relative potencies of the proton pump inhibitors lanzoprazole and omeprazole on gastric acidity are not clear. This study was designed to compare the effects of lanzoprazole (LAN) 15, 30 and 60 mg and of omeprazole (OME) 20 and 40 mg on 24-hour intragastric acidity and 24-hour plasma gastrin profile. **Methods:** 16 healthy volunteers (8 males/8 females) participated in a randomized, double-blind, five-way crossover study. Each treatment period was followed by a wash-out period of at least two weeks. Intragastric acidity was measured prior to (day 0) and on day 5 of each treatment period with a glass electrode positioned in the fundic part of the stomach and connected to an ambulatory recorder. During each day of intragastric monitoring, blood for gastrin analysis was sampled at regular intervals for 24 hours. Plasma was stored at -20°C until assayed by RIA. Median intragastric 24 hour pH (MED pH), and nocturnal pH (11pm-7am), and $[H^+]$ every 10 minute was calculated using StatpHac II (Synectics). From 24 hour $[H^+]$ and gastrin concentration area under time curve (AUC), the per cent inhibition of intragastric acidity and per cent increase of gastrin concentration for each treatment period were calculated. **Results:** All treatments decreased gastric acidity and increased gastrin concentrations at day 5 ($p<0.01$). Effects on MED pH, acidity and gastrin concentration, and comparisons between the treatments are given in the table. Statistics: Median (range),Wilcoxon's sign-rank test.

Treatment dose (mg)	MED pH (day 5)	% inhibition 24 hr $[H^+]$AUC	% increase 24 hr gastrin AUC
a) LAN 15	2.90 (1.82-4.67)	60 (19-97)	16 (-20-206)
b) LAN 30	2.99 (1.22-5,45)	67 (0-98)	40 (10-191)**a
c) LAN 60	3.79 (2,29-6.40)*a,b	80 (61-93)*b,d	58 (11-168)**a,d/*b
d) OME 20	3.01 (0.83-6.78)	67 (0-86)	24 (2-157)
e) OME 40	4.15 (2.51-6,61)**a,b/*d	87 (0-96)*b,d	63 (2-217)**a/*d

* $p<0.05$ and ** $p<0.01$ indicate significant differences between treatments a-e

There were no statistical differences in nocturnal pH among the different treatments. No correlations between intragastric acidity and gastrin concentration profiles during the treatments (day 5) could be found. **Conclusion:** Effects of lansoprazole at 15 and 30 mg were equivalent to 20 mg of omeprazole on gastric acidity; the effect of lansoprazole 60 mg corresponds to 40 mg of omeprazole.

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