

the primary reason for transplantation in 113 patients (68%). Other indications included idiopathic pulmonary fibrosis, COPD, bronchiectasis, and alpha-1 antitrypsin deficiency. Exclusion criteria included patients with a prior diagnosis of gastric motility disorder and/or patients who died less than 30 days after transplantation. Following lung transplantation upper gastrointestinal endoscopy was performed on 11 CF patients for further evaluation of an abnormal abdominal plain film (n=4), early satiety/possible gastric outlet obstruction (n=3), abnormal medication levels (n=2), dysphagia (n=1), and jaundice with a dilated common bile duct (ERCP/EGD, n=1). RESULTS: A total of 165 patients underwent lung transplantation during the study period. 68% (113) of these patients were transplanted for CF. 28 patients died less than 30 days after transplantation and were excluded (10 CF patients). None of these patients had a prior history of gastrointestinal motility disorder or a known diagnosis of a bezoar. Post-transplantation, 11 CF patients (4 men, 7 women, mean age 28.2) were found to have gastric bezoars by endoscopy for an overall incidence of 10.6% for the CF population ($p=0.04$ compared to the non-CF patients by Chi square analysis). The mean time to endoscopic evaluation after transplant was 37.5 days. Of the patients who underwent lung transplantation for other reasons, none were found to have bezoars on any subsequent upper gastrointestinal endoscopy. CONCLUSIONS: Patients with cystic fibrosis who undergo lung transplantation are likely to form gastric bezoars within 30-45 days of transplantation. The pathophysiology is likely multifactorial and includes impaired gastrointestinal motility and altered secretion physiology. Further investigation is warranted as bezoars have been implicated in altered absorption of critical immunosuppressants and other medications. Consideration should be given to post-transplantation surveillance endoscopy in patients with CF.

T11835

Gastric Myoelectrical Activity Abnormalities in Patients with Chagas' Disease

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BACKGROUND: Chagas' disease (CD) is characterized by a variable degree of destruction of the neurons of the enteric nervous system along the digestive tract. CD may present gastric emptying disorders. Gastric dysrhythmias have been reported in patients with neurophatic pseudoobstruction with myenteric plexus lesions. It is not known if patients with CD may present gastric myoelectrical activity disturbances. **AIM:** To evaluate the gastric myoelectrical activity in patients with CD using cutaneous eletrogastrography. **METHODS:** Cutaneous eletrogastrography (EGG) was performed in 33 CD patients, presenting digestive symptoms (22 men, 27-65 years), chagasic group and in 15 healthy volunteers (8 men, 18-60 years), control group. The EGG was performed in two different periods: basal (in fasting) and post-prandial, after a yogurt and water meal (270 ml). After visual inspection and artifact elimination, a continuous spectral analysis was performed (Rapid Fourier Transform). The following parameters were evaluated: dominant frequency (DF), % DF in spectral bands: normogastria (2-4cpm), bradygastria (<2cpm), tachygastria (4-10cpm), duod/resp(10-15cpm), and the ratio of postprandial/basal EGG amplitude (AR). EGG was considered normal with: normogastria (>50%) and tachygastria (<10%) in both EGG periods, and a positive and persistent AR. **RESULTS:** The chagasic group showed % normogastria in fasting EGG significantly lower (88.6 ± 18.2 vs. 62.1 ± 33.2 , $p<0.01$); % bradygastria significantly higher in fasting EGG (10 ± 15 vs. 25.2 ± 40.8 , $p<0.05$); % tachygastria significantly higher in both fasting (0.2 ± 0.8 vs. 9.3 ± 19 , $p<0.01$) and post-prandial EGG (0.2 ± 0.9 vs. 3.6 ± 6.2 , $p<0.01$). EGG was considered normal in 14/15 (93.3%) control vs. 15/33 (45.4%) in chagasic patients ($p<0.05$). 10/33 chagasic patients presented an abnormal EGG response to the meal. 17/33 chagasic patients presented a dysrhythmic EGG: bradygastria(n=7), tachygastria (n=6), arrhythmic (n=3), duodenal (n=1). **CONCLUSIONS:** 1. Patients with digestive manifestations of Chagas' disease may present gastric myoelectrical activity abnormalities 2) Gastric dysrhythmias are one of the abnormalities presented in the chagasic gastropathy 3) EGG may be useful for the diagnosis of gastric myoelectric plexus involvement in patients with Chagas' disease.

T11836

Roles of nNOS and eNOS in Gastric Fundus Relaxation and Accommodation

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Background/Aims: Impaired gastric accommodation *in vivo* is noted in various disorders including functional dyspepsia and diabetic gastropathy. Nitric oxide is a major relaxatory mediator but its exact role and enzymatic source for gastric relaxation and accommodation remain unclear owing to limitations of pharmacologic agents. We examined the relative contributions of the constitutive nitric oxide synthases (nNOS and eNOS) in relaxation of fundic circular smooth muscle strips and in adaptive accommodation of isolated stomach using knockout mice. **Methods:** 1) Fundic smooth muscle strips from age-matched wild-type (WT), nNOS^{-/-} and eNOS^{-/-} mice were placed in vertical tissue baths and nonadrenergic (guanethidine 5 μM) noncholinergic (atropine 1 μM) responses to electrical field stimulation (EFS, 0.5-20Hz) were measured by Grass isometric force transducers. 2) For adaptive accommodation studies, whole stomachs excised from WT, nNOS^{-/-} and eNOS^{-/-} mice were cannulated for introducing Krebs and drug solutions (50 μL/10 sec). Intragastric pressures were measured with a solid state Millar Mikro-Tip catheter. **Results:** 1) In muscle strips, response to EFS in WT tissues consisted of a tetradotoxin-sensitive intra-stimulus relaxation (-0.75 ± 0.08 gm/mm², 30.5 ± 1.3 s at 5Hz), followed by a rebound contraction. L-nitroarginine (L-NA, 200 μM), which inhibits both nNOS and eNOS, abolished both the relaxation and rebound contraction. nNOS^{-/-} tissues lacked both the EFS-induced relaxation and rebound contraction, but revealed a prolonged relaxation (-0.47 ± 0.25 gm/mm², 261 ± 66 s). This relaxation was suppressed by L-NA, suggesting a role of eNOS. Like WT, eNOS^{-/-} stomach showed an intra-stimulus relaxation followed by a rebound contraction which were abolished by L-NA. 2) The WT and eNOS^{-/-} stomachs showed prominent adaptive accommodation which was completely abolished by L-NA. However, nNOS^{-/-} mice showed absence of accommodation. **Conclusions:** Use of knockout mice shows that L-NOS mediates gastric relaxation and rebound contraction and plays a pivotal role in adaptive gastric accommodation. eNOS is not involved in gastric accommodation, but mediates a prolonged EFS-induced relaxation.

T11837

Decreased Fundic Volume Response to Feeding is Associated with Greater Postprandial Antral Volume in Healthy Subjects

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Background: The stomach fundus normally relaxes postprandially. In some diabetics with dyspeptic symptoms there is decreased postprandial fundic relaxation and greater distribution of food to the antrum. The role of postprandial change in antral volume in the generation of symptoms is unclear. **Aims:** To assess antral volume response to feeding in healthy humans and its relationship with fundic volume response, satiation and postprandial symptoms. **Methods:** We assessed satiation (maximum Ensure® volume ingested at a constant rate) and postprandial symptom (nausea, bloating, fullness and pain) scores using a standard Nutrient Drink test. We measured gastric volumes during fasting and after 300mL Ensure® by means of ^{99m}Tc-SPECT imaging. This visualizes the gastric wall rather than the intragastric content and calculates the viscous volume. An algorithm that estimates the longest axis of the stomach and divides it into proximal 2/3 and distal 1/3 was used to calculate fundic and antral volumes, respectively. We used multiple linear regression to evaluate the relationship among variables. **Results:** We studied 43 healthy subjects (33 females and 10 males). Median age was 30y (range: 18-51) and median BMI was 28Kg/m² (range: 20-48). Table shows fundic and antral gastric volumes during fasting and postprandially. After meal ingestion, there were significant increases in postprandial fundic ($p<0.0001$) and antral ($p<0.0001$) volumes. Decreased fundic volume was associated with increased antral gastric volumes postprandially ($p=0.0046$), and this was not affected by gender or BMI. Increased fundic volume during fasting (not postprandially) was associated with decreased postprandial bloating score ($p=0.03$, adjusted for gender and BMI). We did not observe any association between antral gastric volumes (during fasting and postprandially) and either nutrient drink intake or postprandial symptoms. **Conclusion:** Gastric antrum increases its volume in response to feeding in healthy humans; this response is inversely correlated to the fundic volume response. The role of antral volume response to feeding in the genesis of symptoms in dyspeptic patients needs further study.

Gastric Volume (mL)	Fundus	Antrum
Fasting	110 (88-150)	105 (84-134)
Postprandial (PP)	398 (288-504)	289 (208-347)
Difference (PP-Fast)	279 (204-368)*	174 (108-238)*

Data are median (interquartile range). * $p<0.0001$ (Wilcoxon Rank Sum test: testing different from 0)

T11838

Lactate Colonic Infusion Delays Liquid Gastric Emptying in Humans, Through an Intraluminal Pathway Which Is Independent of Pyy, Glp-1 and Oli

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Colonic fermentation of lactose produces Short Chain Fatty Acids (SCFAs) which are metabolized into lactate. Colonic fermentation of lactose inhibits gastric tone in humans, but its effect on gastric emptying is unknown. Different mechanisms may be involved in such feedback phenomenon eg intraluminal regulation via chemoreceptors, pH-sensitive receptors, and humoral regulation via the enterogastrone candidates ie peptide YY (PYY), Glucagon-Like Peptide 1 (GLP1) and Oxyntomodulin (OL1). The aim of this study was to determine the effect of intracolonic infusions which differ in their pH and contents on 1/ liquid gastric emptying and, 2/ PYY, GLP1 and OL1 plasma levels in humans. Four 200 mL solutions were randomly infused at a rate of 2 ml/min during 4 separated days in the proximal colon of eight volunteers: a/ 0.15 M NaCl, pH 6.5, b/ 0.15 M NaCl, pH 5.5, c/ 36 mM SCFAs, pH 6.5, and d/ 30 mM Lactate, pH 6.5. The time-course of plasma concentrations of PYY, GLP 1 and OL1 was determined during and 3 hours after each infusion. The gastric emptying of a 200 mL liquid acaloric meal (0.15 M NaCl) was studied by the 13C acetate breath test. Results are expressed by mean +/- SEM. All infusions were responsible for a significant release of PYY, especially the SC FAs infusion. There was no correlation between any of the peptide release and the gastric emptying rate. GLP 1 and OL1 were not released during the four infusions. Only the lactate infusion induced a significant delay of liquid gastric emptying ($t_{1/2}$ Lac t ate = 66.1 ± 11.4 min vs $t_{1/2}$ NaCl6.5 = 48.3 ± 2.4 min, $P=0.03$, T-Wilcoxon test). Neither the low pH infusion nor the SCFAs infusion significantly modified the gastric emptying rate. In conclusion, 1/ only lactate colonic infusion but not SCFAs delay s the liquid gastric emptying in humans, and 2/ this effect is independent of PYY, OL1 and GLP 1 release. These results are consistent with an intraluminal regulation of the so-called colonic brake, via a chemoreceptor rather than through a pH-sensitive receptor pathway. This study was supported by grants from Astra Pharmaceuticals, Issy-Les-Moulineaux- France..

T11839

The Effect of Subcutaneous Pegylated Recombinant Native Human Leptin on Gastric Emptying in Man

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Introduction: Leptin, the ob gene product, is primarily produced by adipocytes but also at a lower level by gastric tissue. Recently, it has become clear that the stomach contains leptin receptors. Leptin plays an important role in the regulation of food intake and energy expenditure. It may reduce appetite, which could be related to changes in gastric emptying. However, it is not known whether leptin has a direct action on gastric emptying. **Methods:** 10 healthy non-obese males (age 19-36 y) were included. Gastric emptying was measured in the morning at baseline, and 3 days after subcutaneous administration of a single dose of 80 mg long-acting pegylated human recombinant leptin (PEG-OB). It is known that peak serum levels are reached three day after administration*. Gastric emptying of a standardised solid test meal was measured using the 13C-octanoic acid breath test. Serum leptin levels