1475 Effect of Sodium-chenodeoxycholate on Basal and CCK-Induced Gallbladder Motility, Pancreatic Enzyme Secretion and Plasma PP Levels

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Sile salt diversion from the gut modulates gallbladder motility and pancreatic enzyme secretion by interactions with plasma cholecystokinin (CCK) release. To further elucidate the role of bile acids in the regulation of pancreatic-biliary function we studied the effect of intraduodenal (i.d.) perfusion of sodium chenodeoxycholate (CDCA) on basal and CCK-induced gallbladder motility, pancreatic enzyme secretion and plasma pancreatic polypeptide (PP) levels.

Methods: Two tests were performed in 7 healthy subjects (2 M, 5 F; 18-28 yrs.). Saline (6 mL/h) with or without CDCA (0.5 g/h) was continuously perfused i.d. for three hours followed by the last test hour in basal state (0.53 i.d. kg/h) and was infused in both tests. Plasma PP (RIA) and bile salt levels (chromatography), gallbladder volume (ultrasound) and amylase output (footprint sampling using PEG-4000 as a recovery marker) were measured at regular intervals.

Results: Plasma CDCA levels in the CDCA study were significantly (p < 0.01) increased when compared to the saline study (3.8 ± 0.9 vs 0.8 ± 0.2 mg/L; p < 0.05) and to control (3.8 ± 0.9 mg/L; p < 0.05). Basal PP levels were increased in both tests (1.4 ± 0.7 vs 0.7 ± 0.2 mg/L; p < 0.05). The only significant difference was observed in plasma PP levels in the saline study when compared to the CDCA study (1.4 ± 0.7 vs 0.7 ± 0.2 mg/L; p < 0.05).

Conclusion: Duodenal perfusion of CDCA decreases basal and CCK-stimulated gallbladder motility, abolishes the rise in CCK-induced plasma PP levels but is without significant effect on pancreatic enzyme secretion. These data indicate that CDCA inhibits the effects of CCK on gallbladder motility and PP release.

1476 Cost-Effective, European Approach to H. pylori Eradication

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Introduction. In recent years, the efficacy of treatments aimed at the eradication of H. pylori has improved significantly. The low dose, inexpensive, one-week Bologna regimen (imipenem 20 mg/kg daily + clarithromycin 250 mg b.i.d. + metronidazole 400 mg b.i.d) and the two week Boerse regimen (omeprazole 20 mg b.i.d. + amoxicillin 1 g b.i.d. + clarithromycin 500 mg b.i.d.) achieve eradication in 90-100% of cases. There is, as yet, no standardised approach to eradication therapy. A first treatment should be reliable, well-tolerated, inexpensive and efficacious.

Alm. 1) To assess the suitability of the Bologna regimen as a first line eradication protocol for H. pylori. 2) To determine the factors that lead to treatment failure. 3) To evaluate the Boerse regimen as a second line eradication protocol.

Patients and Methods. Subjects with H. pylori-associated duodenal ulcer (DU) or non-ulcer dyspepsia (NUD) were recruited at endoscopy. H. pylori status was assessed 4 weeks after treatment by histology (luminal + corpus x 2), culture (antral + corpus) and CLO-test (antral subjects were positive if 2 or more tests were +ve and negative if all tests were -ve). All subjects were treated with the Bologna regimen.

Results. 162 subjects were enrolled (79 male, 141 NUD and 21 DU, mean age 49 years (range 18-78). 150 patients completed the follow-up. H. pylori was eradicated in 121/150 (80.6%). Pre-treatment sensitivity were available in 20 of the 29 patients in whom treatment failure. 18/20 (90%) had primary metronidazole resistance, 1/20 had metronidazole and clarithromycin resistance and the remaining patient was sensitive to both antibiotics. 14 of the 29 subjects were subsequently treated with the Boerse regimen. H. pylori was eradicated in 17/19 (90%)

Conclusion. The inexpensive, Bologna regimen eradicated H. pylori in 80.6% of patients. Primary metronidazole resistance is an important factor in treatment failure. The more expensive Boerse regimen is a highly effective second-line treatment.

1477 Different Effects of Medium-Chain Triglycerides and Long-Chain Triglycerides on Gastrin Stimulated Gastric Acid Secretion

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Fat in the small intestine stimulates cholecystokinin (CCK) and inhibits gastric acid secretion. In the present study we have investigated the role of fatty acid chain length and the role of circulating CCK in the inhibition of gastrin stimulated gastric acid secretion.

Methods. 8 healthy volunteers (8 M, 23 ± 2 yrs) were studied. 4 experiments were performed in random order on different days. In all experiments gastrin (17 nM) was infused for 150 min. During the last 90 min the duodenal perfusion was with equimolar amounts of fatty acids (124 mmol/l) of either corn-oil, mainly containing C18 fatty acids (LCT) or Ceres-MCT-oil, mainly containing C8 and C10 fatty acids (MCT). During the last hour 

1478 Effect of Intraduodenal Digestible and Non-digestible Fat on Gastrin Stimulated Gastric Acid Secretion


Fat in the small intestine stimulates cholecystokinin (CCK) and inhibits gastric acid secretion. It is not known whether intact or hydrolysed triglycerides are responsible for this enterogastric effect. We have investigated whether digestible fat (frying-oil) or non-digestible fat (ticroncose polymer; SPE) containing fatty acids of comparable chain length inhibits gastrin stimulated gastric acid secretion and stimulates plasma CCK.

Methods. 8 healthy volunteers (8 M, 23 ± 2 yrs) were studied. 3 experiments were performed in each volunteer in random order on different days. In all experiments gastrin (17 nM) was infused for 180 min in a dose of 10 pmol/kg/h. This dose results in plasma gastrin concentrations comparable to those after a meal. After one hour the duodenal perfusate was with equimolar amounts of fatty acids (62 mmol/l) of either digestible fat or sucrose polymer (SPE) for 60 min, at a perfusion rate comparable to the gastric emptying rate of fat after a meal. In the third experiment saline instead of fat was perfused. We have measured gastric acid secretion (phenoxy red recovery technique) and plasma gastrin and CCK concentrations (RIA) at regular intervals.

Results. Infusion of fat resulted in plasma gastrin concentrations ranging from 48 ± 4 to 65 ± 5 pmol/L. Digestible fat (62 ± 10 pmol/L) stimulated plasma CCK when compared with saline (5.4 ± 13.9 pmol/L; p = 0.0009). Gastrin-stimulated gastric acid secretion during saline perfusion (20.1 ± 1.6 mmol H+ /h) was inhibited (p = 0.0004) by fat (9.6 ± 2.7 mmol H+ /h) but not by SPE (17.8 ± 2.4 mmol H+ /h).

Conclusions. Intraduodenal perfusion of digestible fat but not of undigestible fat inhibits gastrin-stimulated gastric acid secretion. The inhibit of gastrin-stimulated acid secretion by undigestible fat does not signify the release of CCK. Our data demonstrate that hydrolysis of fat is important for the enterogastric effect of fat and for the release of CCK.

1479 The Importance of Saliva as a Sample for Diagnosis of H pylori Infections Under Various Sampling Conditions

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Experience has proven the usefulness of serum as a diagnostic sample. Saliva, however, would be superior to serum as a sample in a number of ways. Acquisition is simpler than venepuncture, is painless and non-invasive, and the sample itself presents less danger to those handling it than does blood. The usefulness of salmonpim immunoglobulin (Ig) as a diagnostic tool depends ultimately on its reliability as a source of information. One of the major and most basic advantages of serum in diagnosis is its reliability. The composition of saliva, however, is known to be extremely variable. Whether or not this variability can lead to the immune status of an individual to a particular organism being obscured under certain conditions is largely unknown. In order for salmonpim immunoglobulin to be of diagnostic use, the level of the specific immunoglobulin detected must not vary to such an extent that the response is obscured under a particular set of conditions.

We have investigated the effects of sampling. Brushing of teeth and circadian rhythm on the apparent salmonpim immune status of 35 individuals known to be serum and saliva anti-HAV positive, and from an equal number of anti-HIV negative individuals. Saliva samples were collected from the subjects before and after meals, before and after brushing of teeth, and at various times throughout the day. To date, samples from 20 anti-HAV positive and 20 anti-HIV negative individuals have been assayed for total IgG and for total anti-