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

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Bile salt diversion from the gut modulates gallbladder motility and pancreatic enzyme secretion, possibly by interference with plasma cholecystokinin (CCK) release. To further elucidate the role of bile acids in the regulation of pancreatico-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 (5 mL/min) with or without CDCA (0.5 g/h) was continuously perfused i.d. for three hours. During the last test hour CCK (0.33 I.D.U. kg $^{-1}$ h^{-1}) was infused in both tests. Plasma PP (RIA) and bile salt levels (chromatography), gallbladder volume (ultrasonography) and amylase output (spot 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 μM and 12.6 ± 2.6 vs 4.7 ± 2.7 μM after 2 h and 3 h of perfusion respect.). CDCA increased basal gallbladder volume from 28 ± 5 mL to 35 ± 7 mL ($p < 0.05$), but was without significant effect on basal amylase and PP. CDCA diminished CCK-stimulated values for integrated gallbladder contraction from 2365 ± 309%·60 min to 1133 ± 178%·60 min ($p < 0.05$), integrated plasma PP from 787 ± 300 pM·60 min to 138 ± 93 pM·60 min ($p < 0.05$) and tended to decrease incremental amylase output from 3.0 ± 1.6 to 1.6 ± 0.9 kU/h (NS).

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 regime (omeprazole 20 mgs daily + clarithromycin 250 mgs b.i.d. + metronidazole 400 mgs b.i.d) and the two week Bordeaux regime (omeprazole 20 mgs b.i.d. + amoxicillin 1 gram b.i.d. + clarithromycin 500 mgs b.i.d.) achieve eradication in 90–100% of cases. There is as yet, however, no standardised approach to eradication therapy. A first line treatment should be reliable, well-tolerated, inexpensive and efficacious.

Aim. 1) To assess the suitability of the Bologna regime as a first line eradication treatment. 2) To determine the factors that lead to treatment failure. 3) To evualate the Bordeaux regime as a second line eradication treatment.

Patients and Methods. Subjects with H. pylori-associated duodenal ulceration (DU) or non-ulcer dyspepsia (NUD) were recruited at endoscopy. H. pylori status was assessed before and 4 weeks after treatment by histology (antral + corpus × 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 regime.

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 sensitivities were available in 20 of the 29 patients in whom treatment failed. 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 Bordeaux regime. H. pylori was eradicated in 13/14 (92.9%).

Conclusion. The inexpensive, Bologna regime eradicated H. pylori in 80.6% of patients. Primary metronidazole resistance is an important factor in treatment failure. The more expensive Bordeaux regime 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 (10 pmol/kg.h) was infused for 150 min. During the last 90 min the duodenum was perfused with equimolar amounts of fatty acids (124 mmol/h) of either corn-oil, mainly containing C18 fatty acids (LCT) or Ceres-MCT-oil, mainly containing C8 and C10 fatty acids (MCT) or with saline (n = 8). In the fourth experiment CCK-33 was infused i.v. for the last 90 min of the experiment in amounts that resulted in plasma levels that were somewhat higher than during perfusion of LCT (n = 6). At regular intervals we have measured gastric acid secretion and plasma gastrin and CCK concentrations.

Results. Infusion of gastrin resulted in plasma gastrin levels ranging from 46 ± 4 to 55 ± 5 pM comparable to postprandial values, and in gastric acid outputs from 8.6 ± 1.5 to 12.0 ± 2.9 mmol/30 min. LCT (+19.0 ± 4.1 pM.30 min) but not MCT (−4.8 ± 4.2 pM.30 min) stimulated plasma CCK when compared to saline (+1.3 ± 5.3; $p = 0.002$). CCK infusion increased plasma concentrations by 108.8 ± 10.5 pM.30 min. Gastrin stimulated gastric acid output was inhibited by LCT by 74 ± 6% ($p = 0.0003$) and by MCT by 43 ± 9% ($p = 0.043$) compared to saline (17 ± 4%). LCT inhibited gastric acid output significantly more than MCT ($p = 0.05$). CCK failed to inhibit gastrin stimulated gastric acid output (18 ± 6%).

Conclusions. Intraduodenal LCT inhibit gastrin stimulated gastric acid secretion significantly more than MCT. LCT but not MCT stimulated the release of CCK. However, infusion of CCK to plasma concentrations somewhat higher than during perfusion of LCT did not inhibit gastrin stimulated gastric acid secretion.

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

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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 enterogastrone effect. In the present study we have investigated whether digestible fat (frying-oil) or non-digestible fat (sucrose polyester, 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 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 duodenum was perfused with equimolar amounts of fatty acids (62 mmol/h) of either digestible fat or sucrose polyester (SPE) for 90 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 (phenol red recovery technique) and plasma gastrin and CCK concentrations (RIA's) at regular intervals.

Results: Infusion of gastrin resulted in plasma gastrin concentrations ranging from 46 ± 4 to 55 ± 5 pM. Digestible fat (+66.3 ± 10.9 pM. 60 min) but not SPE (−24.7 ± 14.5 pM. 60 min) stimulated plasma CCK when compared with saline (−5.4 ± 13.9 pM. 60 min; $p = 0.0092$). Gastrin-stimulated gastric acid secretion during saline perfusion (21.0 ± 1.6 mmol H^+/h) was inhibited ($p = 0.0004$) by fat (9.6 ± 2.7 mmol H^+/h) but not by SPE (17.9 ± 2.4 mmol H^+/h).

Conclusions. Intraduodenal perfusion of digestible fat but not of undigestible fat inhibits gastrin-stimulated gastric acid secretion. Digestible but not undigestible fat stimulates the release of CCK. Our data demonstrate that hydrolysis of fat is important for the enterogastrone effect of fat and for the release of CCK.

1479 The Reliability of Saliva as a Sample for Diagnosis of Hepatitis a Infection 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 salivary 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 salivary 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 eating, brushing of teeth and circadian rhythm on the apparent salivary immune status of 35 individuals known to be serum and saliva anti-HAV positive, and from an equal number of anti-HAV negative individuals. Saliva samples were obtained from the subjects before and after meals, before and after brushing of teeth, and at various timepoints throughout the day. To date, samples from 20 anti-HAV positive and 20 anti-HAV negative individuals have been assayed for total IgG and for total anti-