1475 Effect of Sodium-chendexocholate on Basal and CCK-induced Gastric 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 choleschoktoin (CCK) release. To further elucidate the role of bile acids in the regulation of CCK release, we studied the effect of intraduodenal (i.d.) perfusion of sodium chendexocholate (DDCA) 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 DDCA (0.5 g/h) was continuously perfused i.d. for three hours. During the last test hour CCK (0.33 I.U. kg– 1 h–1) was infused in both tests. Plasma PP (RIA) and bile salt levels (chromatography), gallbladder volume (ultrasoanography) and amylase output (spot sampling using PEG-4000 as a recovery marker) were measured at regular intervals.

Results. Plasma DDCA levels in the DDCA study were significantly (p < 0.01) increased when compared to the saline study (3.8 ± 0.9 vs 0.8 ± 0.2 mM and 12.6 ± 2.6 vs 4.7 ± 2.7 μM after 2 h and 3 h of perfusion respectively). DDCA increased basal gallbladder volume from 26 ± 5 mL to 35 ± 7 mL (p < 0.05). Without significant effect on basal amylase and PP CCK 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 μg/mL 60 min to 136 ± 93 μg/mL 60 min (p < 0.05) and decreased incremental amylase output from 3.0 ± 1.5 to 1.6 ± 0.9 kU/h (NS).

Conclusion: Duodenal perfusion of DDCA decreases basal and CCK-stimulated gallbladder motility, abolishes the rise in CCK-induced plasma PP levels but without any significant effect on pancreatic enzyme secretion. These data indicate that DDCA 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 mg daily + clarithromycin 250 mg b.i.d. + metronidazole 400 mg b.i.d.) and the two week Bologna regime (omeprazole 20 mg b.i.d. + amoxicillin 1 gram b.i.d. + clarithromycin 500 mg 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.

Methods. We investigated the suitability of the Bologna regime as a first line eradication treatment. 1) To determine the factors that lead to treatment failure. 2) To eradicate the Bologna regime as a second line eradication treatment. Patients and Methods. Subjects were recruited at endoscopy. Clarithromycin resistant (CXR) or non-CXR duodenal ulcer (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 (78 male), 141 NUD and 21 DU, mean age 49 years (range 18–78). 150 patients completed the full follow-up. H. pylori was eradicated in 121/150 (80.6%). The treatment sensitivity was 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 Bologna 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 in treatment failure is an important factor. The more expensive Bologna 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 gastric 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 (110 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/l) of either corn-oil, mainly containing C18 fatty acids (LCT) or Ceres-CET-mix, mainly containing C24 and C16 fatty acids (MCT) with water. 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 = 8). 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 4.6 ± 0.4 to 56 ± 5 pmol/l in control rats. Ingestion of corn-oil resulted in plasma CCK concentrations from 108.8 ± 1.05 pmol/l. Gastrin stimulated gastric acid output was inhibited by LCT by 74 ± 6% (p = 0.0005) and by MCT by 43 ± 8% (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 ± 4%).

Conclusions. Intraduodenal CCK inhibit gastrin stimulated gastric acid secretion significantly more than LCT. But not MCT stimulate 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 digestive fat (frying oil)- or non-digestible fat (succrose polymer, SPE) containing fatty acids of different chain length inhibits gastrin stimulated gastric acid secretion and stimulates CCK production.

Methods. 8 healthy volunteers (6 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/l) of either digestible fat or sucrose polymer (SPE) for 90 min, at a perfusion rate comparable to the gastric emptying rate of fatty acids after a meal. In the third experiment saline instead of fat was perfused. In order to measure gastric acid secretion (bioluminescent technique) and plasma gastrin and CCK concentrations (RIA's) at regular intervals.

Results. Gastrin resulted in plasma gastrin concentrations rang- ing from 4.6 ± 0.4 to 68 ± 6 pmol/l. Digestible fat (68 ± 3 ± 10.9 pmol/l) did not significantly effect plasma gastrin levels but non-SPE (24.7 ± 14.5 pmol/l 60 min) stimulated plasma CCK when compared with saline (5.4 ± 13.9 pmol/l 60 min; p = 0.005). Gastrin-stimulated gastric acid output during saline perfusion (21.6 ± 1.6 mmol/l 60 min) was inhibited by SPE (0.0004) by fat (9.6 ± 2.7 mmol/l H2) but not by SPE (7.3 ± 2.4 mmol/l H2).

Conclusions. Intraduodenal perfusion of digestible fat but not of undigestible fat inhibits gastrin-stimulated gastric acid output but does not increase CCK production. 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 saliva as a diagnostic sample. Saliva, however, would be superior to serum as a sample in a number of cases. Acquisition is simpler than blood sampling, and the sample itself presents less danger to the handling it than does blood. The usefulness of salivary immunoglobulin (IgG) as a diagnostic tool depends ultimately on its reliability as a source of information. One of the major aids to the use of saliva in diagnosis is its reliability. The collection 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. The production of salivary immunoglobulin in the presence 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 fasting, brushing of teeth and circular rhythm on the parasitic 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. The samples from 20 anti-HAV positive and 20 anti-HAV negative individuals have been assayed for titol IgG and for total anti-