HOW IMPORTANT ARE GASTRIC PEPSIN, ACID AND DUODENAL CONTENTS IN GASTRO-ESOPHAGEAL REFLUX PATIENTS (GER) WITH OR WITHOUT EROSIVE ESOPHAGITIS (E).E.Malikova, G.Cadiot, M.Mignon, I.Sobhani, C.Flores, P.Ruszniewski, T.Vallot, C.Poitevin, J.Vatier. Department of Gastroenterology and INSERM U.10, Hopital Bichat, 75877 Paris-France.

In GER patients (pts), gastric contents studies are scarce or conflicting; none assessed gastric juice proteolytic activity while it is a major agressive factor through its mucolytic effect. Fifty one GER pts were studied : 28 M, 23 F, mean age : 55 ± 15 (SD) years; endoscopically proven E (no stenosis) existed in 35 pts (E+) and absent in 16 (E-). Groups were comparable for age and bw not for sex ratio. In E-pts, GER was confirmed by 24-hr esophageal pH-metry (De Meester criteria) except for 3 with hiatal hernia, heartburn and/or regurgitation. By gastric intubation after a 12-hr fast and withdrawal of antisecretory drug (≥3 days) were measured : a) 15-min emptying volume (EV, ml), b) basal and stimulated (pentagastrin 6 µg/kg/IM) outputs of pepsin (BPO, PPO, IU/hr) and acid (BAO, PAO, mmol/hr); c) maximal basal H+ concentration (mmol/l) d) stimulated outputs of sialic acid (PSAO in µg/hr), as marker of mucolysis and of choline as marker of duodeno-gastric (DG) reflux (PChO, µmol/hr).

Median (med.) and extreme values (ex.v.) were for E+ and E- pts respectively: 40 (14-135) vs 38 (16-120) for EV, 50 (0-90) vs 45 (0-95) for maximal H+, 4.1 (0-14) vs 3 (0-15) for BAO, 26.4 (12-65) vs 22 (7-45) for PAO. Although NS difference (non-parametric test) existed between groups, 29% E+, 25% E- had PAO above normal values (Biol Gastroenterol 1973, 6 : 187-196). Med. and ex. v. for BPO, PPO, PSAO, PChO were for E+ and E- respectively: 462 (0-1481) vs 270 (6-1392) for BPO, 1510 (672-2700) vs 1012 (336-2160) for PPO, 1773 (189-4537) vs 1650 (824-3712) for PSAO, 5 (0-136) vs 3 (0-130) for PChO; although NS difference existed between groups for those parameters, 30% E+ and 35% E- have BPO and PPO above normal limits as determined from 37 normal controls and in both groups 25% pts had increased DG reflux.

Twenty five to 35% E+ or E- GER pts, as in duodenal ulcer disease, have acid and pepsin hypersecretion; however the similarity between E+ and E- groups in terms of gastric components in this limited population supports the multifactorial character of GER disease.

GASTRIC SUCTION BIOPSY - A NON-ENDOSCOPIC METHOD TO DIAGNOSE HELICOBACTER PYLORI. B.J. Marshall, S.R. Hoffman, C.G. Antonesu, L.J. Barrett, H.F. Frierson, R.W. McCallum, University of Virginia, Charlottesville, VA 22908.

The gold standard for diagnosis of H.pylori (HP) is culture of a gastric mucosal biopsy, a process which also permits antibiotic susceptibility testing. In patients who did not otherwise require endoscopy, we wished to obtain culture and histology by a simpler and less expensive method. **AIM**: To study the efficacy of blind gastric suction biopsy as a means of diagnosing H.pylori. **METHODS**: A "Quinton" suction biopsy tube (diameter 3.5 mm) was passed on 13 consecutive patients attending the gastritis clinic in whom H.pylori status was known but in whom culture had not been performed. Patients sat during the procedure and the tube was passed through the mouth after applying local anesthetic to the throat. Location in the stomach was confirmed by measurement and by auscultation of injected air. Tissue was sent primarily for culture, then for histology and CLOtest, depending on the amount obtained. Suction biopsy samples were read blind by the microbiologist and pathologist. **RESULTS**: The tube was successfully passed without sedation in all 13 patients, but in 3 patients no biopsy material was retrieved. Of the 10 patients successfully biopsied, tissue was obtained for culture (10), histology (10) and CLOtest (7). From other studies (C-14 breath test or histology), we knew that 9 of the 10 patients were HP+. In the HP- patient all specimens gave negative results. In the 9 HP+ patients histology revealed H.pylori and gastritis in 9 (100%), and H.pylori was cultured in 8 (89%). CLOtest was positive in all 6 HP+ patients tested (100%). According to the histology, biopsy samples were obtained from the antrum in 3 patients (33%) and from body mucosa in 6 (66%). One week after the test, patients completed a mailed questionnaire to evaluate their impression of the procedure. Patient scores, representing grades from "no difficulty" to "great difficulty", ranged between 0 and 10, with an average score of 2.6. Of 10 patients who had undergone upper endoscopy in the past, 7 preferred the suction biopsy. **Conclusion**: In most patients, suction biopsy can obtain sufficient material to perform culture and histology for H.pylori. In this small series the sensitivity of the test was 100% in persons from whom tissue was obtained.

● A PLACEBO CONTROLLED CLINICAL TRIAL OF BISMUTH SUBSALICYLATE FOR THE TREATMENT OF HELICOBACTER PYLORI-ASSOCIATED GASTRITIS. B.J. Marshall, J.E. Valenzuela, R.W. McCallum, C.P. Dooley, R.L. Guerrant, H. Cohen, H.F. Frierson, L.G. Field, G.R. Jerdack and S. Mitra, University of Virginia, University of Southern California, Los Angeles, CA, and Procter & Gamble Co., Cincinnati, OH.

H.pylori (HP) associated gastritis is common in patients with non-ulcer dyspepsia (NUD) but an etiologic relationship is unproven. HP is inhibited by bismuth salts such as bismuth subsalicylate (BSS). **The AIM** of this study was to assess the short and long term effect of BSS on HP, gastritis, and symptoms, in patients with NUD. **METHOD**: Patients with NUD who were shown to be both HP+ and to have histologic gastritis were enrolled. Patients with continuing symptoms at the end of a two week placebo washout period were randomly assigned to 3 weeks therapy with either 525 mg QID BSS liquid or a matched placebo. Patients with severe symptoms at the end of the therapy period (week 5) completed the study as treatment failures. Those who improved were followed for an additional 4 weeks on no therapy. EGD, biopsy, and clinical evaluations were performed at entry, Week 5 (post-therapy) and at Week 9 (4 weeks post-therapy), or at time of symptomatic relapse. **RESULTS**: 89 patients entered the placebo phase; 10 violated the protocol and 33 others (37%) were removed as placebo responders; 46 were randomized to therapy but 11 were later disqualified leaving 35 evaluable patients. Of these, 17 received BSS and 18 received placebo. Based upon 35 randomized patients, BSS provided short-term clearance of HP in 70% of patients. Histologic gastritis resolved in 75% of the BSS group. No patient on placebo showed short-term clearance of HP, or histologic improvement. At biopsy 4 weeks after BSS therapy, all patients initially cleared of HP were found to still have the organism, and histology had returned to baseline levels. No significant differences in symptoms were seen between the active and placebo groups during the treatment period. **CONCLUSIONS**: 1) BSS given for 21 days suppresses H.pylori but does not eradicate the organism. 2) Short term suppression of H.pylori (3 weeks) heals gastritis but does not result in clinical improvement. 3) This study does not support or refute a role for H.pylori in non-ulcer dyspepsia since H.pylori was not eradicated and only transient histologic improvement occurred.

GASTROESOPHAGEAL REFLUX AS A PATHOGENIC FACTOR IN THE DEVELOPMENT OF SYMPTOMATIC LOWER ESOPHAGEAL (SCHATZKI) RINGS. JB Marshall, JM Kretschmar, AA Diaz-Arias. Depts of Medicine and Pathology, University of Missouri School of Medicine, Columbia, MO.

PURPOSE. Gastroesophageal reflux (GER) has been suggested as a cause of the lower esophageal ring (LER). We prospectively looked for the presence of GER and reflux injury in a series of patients with LER and dysphagia.

METHOD. Twenty patients with LER and dysphagia underwent 24-hour esophageal pH monitoring, upper GI endoscopy with esophageal biopsy, and esophageal manometry. We established lab normals for 24-hour esophageal pH monitoring by studying 15 asymptomatic controls. Lab normals for esophageal manometry were established by studying 25 asymptomatic subjects.

RESULTS. The 20 patients with LER included 18 men and 2 women with a mean age of 61 years (range 41-74 years). Intermittent dysphagia to solids had been present for a mean of 4.5 years. Seventeen of the patients experienced heartburn at least once per month. All 20 patients had a sliding hiatal hernia, mean length of 3.8 cm. Abnormal GER was documented in 13 of the 20 patients (65%) by ambulatory pH monitoring, ten of whom had erosive reflux changes in the distal esophagus. Seven patients (35%) showed no evidence of pathologic GER or reflux esophagitis. Nonspecific esophageal body motor dysfunction may have contributed to dysphagia in 5 patients, two of whom had no evidence of abnormal GER.

CONCLUSION. GER disease is a frequent cause of the gradually progressive ring stricturing and dysphagia seen in patients with LER. Anti-reflux therapy, as an adjunct to esophageal dilatation, may be appropriate for many symptomatic LER patients.