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HYPOXEMIA DURING UPPER GASTROINTESTINAL ENDOSCOPY: EFFECTS OF SEDATION AND ESOPHAGEAL INTUBATION

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**Introduction:** Patients undergoing outpatient endoscopy develop hypoxemia, which may predispose them to cardiac arrhythmias.[1] The influence of sedation on oxygen saturation ($S_pO_2$) is uncertain. Some studies show that hypoxemia occurs during endoscopy in the absence of sedation.[2] We compared $S_pO_2$ and EKG changes during upper GI endoscopy in patients receiving sedation, no sedation, or sedation with supplementary oxygen.

**Methods:** Fifty-eight patients were randomly allocated to 1 of 3 groups: Group 1 (n=18) received no sedation and breathed room air during endoscopy. Group 2 (n=20) received midazolam (mean dose) 4.7 mg before endoscopy and breathed room air during endoscopy. Group 3 (n=20) received midazolam (mean dose) 5.3 mg and 2 $L{\cdot}min^{-1}$ nasal oxygen during endoscopy. Topical anesthesia was with 120 mg of lidocaine. Monitoring included $S_pO_2$ by pulse oximetry and EKG (3 leads). Data were recorded at baseline, after topical anesthesia, insertion of mouth gag, insertion of endoscope, biopsy, endoscope removal, and 5 minutes after endoscopy. All endoscopies were performed by the same endoscopist. Statistical analysis included analyses of variance (repeated measures); data are expressed as mean (SEM), and $P < 0.05$ is significant.

**Results:** Weight, age, and duration of procedure were similar in the three groups. In group 2 significant decrease in mean $S_pO_2$ occurred after midazolam administration. In groups 1 and 3 mean $S_pO_2$ did not differ from baseline. Group 2 patients differed significantly from those in groups 1 and 3 at mouth gag insertion, endoscope insertion, biopsy, and endoscopy removal. Mean $S_pO_2$ values were lower in groups 2 and 3, 5 minutes after endoscopy when compared to group 1.

**Conclusion:** This study supports the hypothesis that hypoxemia during upper GI endoscopy is directly linked to the use of sedation. In this study, non-sedated patients or those who received oxygen developed fewer hypoxemic episodes. We recommend continuous monitoring of $S_pO_2$ and administration of oxygen during endoscopy.

**References:**

1. *Patterson KW et al*: Br J Anaesth 1991; 67:108-111
2. *Lavies NG et al*: Am J Gastroenterol 1988; 83:618-622

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CLARITHROMYCIN AS MONOTHERAPY FOR ERADICATION OF HELICOBACTER PYLORI. Walter Peterson, M.D., F.A.C.G., and members of the Clarithromycin/H. pylori Study Group, Dallas & Houston, TX, Nashville, TN, Charlottesville, VA & Abbott Park, Ill.

Antimicrobial agents used as monotherapy have been shown to eradicate H. pylori in no more that 10-20% of patients. Clarithromycin (C) is a new macrolide, more acid stable than erythromycin, with excellent in-vitro activity toward H. pylori. The **PURPOSE** of this double-blind study was to assess the ability of 3 dosages of C to eradicate H. pylori in human subjects. **METHODS:** 43 healthy volunteers (24M, 19W, mean age 40y) who were H. pylori positive by $^{13}C$-Urea breath test (UBT), histology, and culture were randomly assigned to C in dosages of 500mg bid, 1000mg bid, or 500mg qid for 14 days. Placebo dummy tablets were used to maintain the blind. UBT was repeated within 48h after completing medication. If the UBT was negative, follow-up endoscopic biopsies were obtained no sooner than 4 wk after completing medication. H. pylori eradication was defined as negative UBT, histology, and culture 4 or more weeks after the end of therapy. **RESULTS:** 37 subjects completed the study according to protocol. Eradication was achieved in 2/13 (15%) with 500mg bid, 4/11 (36%) with 1000mg bid, and 7/13 (54%) with 500mg qid. Eradication was not influenced by age, gender, ethnicity, or smoking history. Only 1/24 strains not eradicated became resistant to C. Taste perversion occurred in 31% with 500 mg bid, 64% with 1000mg bid, and 75% with 500mg qid but caused only 1 subject to drop out. **CONCLUSION:** 1) C alone in a dose of 500mg qid eradicated H. pylori in over 50% of subjects; 2) Resistance to C rarely developed; 3) Clarithromycin may become a valuable addition to anti-H. pylori therapy.

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**ENDOSCOPIC COMPARISON OF THREE ASPIRIN PREPARATIONS AND PLACEBO - LOW DOSE PROPHYLACTIC ASPIRIN EFFECTS ON GASTRODUODENAL MUCOSA.** **Donald Petroski, M.D., F.A.C.G., Rancocas Hospital, Willingboro, NJ**

**80 of 84 volunteers ranging in ages 22-70 successfully completed a 3 month single-blinded endoscopic study consuming single dose placebo, enteric coated aspirin, buffered aspirin, or plain aspirin. Upper endoscopy was performed at onset and after weeks 4, 8, and 12. The gastric and duodenal mucosa was scored endoscopically with the system adapted from Lanza et al. Ecotrin and placebo were not statistically different from one another in gastric damage produced but both were significantly different from Bayer and Bufferin. Significant damage to the gastric mucosa was seen with these latter two agents with no significant difference between them. Upon analysis of duodenal mucosa injury, Bayer aspirin caused significantly more damage than Ecotrin and placebo. Ecotrin and placebo were not significantly different as were the Ecotrin and Bufferin groups.**

**Gastric adaption occurred selectively in the volunteers consuming daily aspirin over 3 months. The relevance of the gastric adaptation phenomenon clinically in the chronic use of aspirin is unclear. Also, statistical significance of age-to-injury could not be proven.**

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EBROTIDINE PROTECTION AGAINST GASTRIC MUCOSAL INJURY INDUCED BY ETHANOL. J. Piotrowski, M.D., A. Slomiany, Ph.D., and B.L. Slomiany, Ph.D. Research Center, UMDNJ, Newark, NJ.

Ebrotidine is a new $H_2$-receptor antagonist with antisecretory potency comparable to that of ranitidine. In this study, we assessed the effect of ebrotidine on the physicochemical characteristics of gastric mucus gel as this agent is known to exhibit gastroprotection against ethanol injury. The study was conducted with groups of rats, with and without indomethacin pretreatment, receiving intragastrically either a dose of ebrotidine or vehicle followed by ethanol given at various intervals up to 4h. The gastric mucosa, 30 min after the ethanol challenge, was then subjected to macroscopic, histologic, and physicochemical assessment. The results revealed that ebrotidine, at doses of 50mg/kg and higher, effectively prevented the alcohol-induced mucosal injury, even in the presence of indomethacin. The protective effect was demonstrable already at 30 min, reached maximum at 1h, and persisted up to 3h. Physicochemical analyses established that ebrotidine elicited 30% increase in mucus gel dimension, caused 19-20% increase in glycolipids and phospholipids, and evoked 21% increase in sulfomucin and 18% increase in sialomucins. As a consequence, the mucus gel viscosity increased by 1.4-fold, $H^+$ retardation capacity by 16%, and hydrophobicity by 65%. The results demonstrate that ebrotidine is endowed with a remarkable mucosal strengthening capability.