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Colonoscopy: how was it for you? ►

▲ Heuss LT, Drewe J, Schnieper P, *et al*. Patient-controlled versus nurse-administered sedation with propofol during colonoscopy. A prospective randomized trial. *Am J Gastroenterol* 2004;**99**:511–18.

The short duration of action and rapid recovery with propofol make it attractive for endoscopy while patient controlled analgesia is a well established technique for analgesia. This trial compared nurse administered sedation (NAPS) with patient controlled sedation (PCS) to determine effectiveness, safety, and patient satisfaction. A total of 155 patients undergoing colonoscopy were eligible, of whom 114 entered the trial. Of these, 40 patients (35%) declined to be randomised, the majority (73%) because they did not want responsibility for their own sedation. This group tended to be younger with higher preprocedure anxiety scores. Thirty-six patients were randomised to PCS and 32 to NAPS. Risk factors for an adverse experience were evaluated, including visual analogue scales for anxiety and, afterwards, tolerability, satisfaction, and willingness to undergo the same method of sedation again. Endoscopists also rated technical difficulty and overall procedure satisfaction, and sedation complications were carefully studied. Both PCS and NAPS were well tolerated with no major complications. The control group (those declining randomisation and treated with standard nurse administered propofol) needed higher doses than the other groups and tended to find the procedure more uncomfortable. PCS resulted in a different drug profile, with 25% of the total being administered during withdrawal of the colonoscope, suggesting that it is not just instrument insertion that patients find uncomfortable. PCS did not prolong the procedure but was more expensive. There was a tendency for both patients and physicians to prefer the nurse administered route although this did not reach significance. Patient administered sedation is therefore suitable for some patients but is clearly not for everyone; the trick is in good patient selection.

Helicobacter pylori and oesophageal adenocarcinoma: association or causation? ►

▲ Ye W, Held M, Lagergren J, *et al*. *Helicobacter pylori* infection and gastric atrophy: risk of adenocarcinoma and squamous-cell carcinoma of the esophagus and adenocarcinoma of the gastric cardia. *J Natl Cancer Inst* 2004;**96**:388–96.

Population *Helicobacter pylori* screening and treatment may prevent mortality from distal gastric adenocarcinoma. This has not been recommended as *H pylori* may have protective as well as harmful effects and the risk-benefit ratio is uncertain. Patients with oesophageal and gastric cardia adenocarcinoma have been reported to be less likely to harbour *H pylori* than non-cancer controls although findings have been conflicting (Wu *et al*. *Int J Cancer* 2003). *H pylori* may have a protective role by causing gastric atrophy and reducing acid secretion. Ye *et al* studied 97 patients with oesophageal adenocarcinoma, 85 with oesophageal squamous carcinoma, 133 with gastric cardia adenocarcinoma and 499 controls. *H pylori* was associated with a reduced probability of oesophageal adenocarcinoma (odds ratio (OR) 0.3 (95% confidence interval (CI) 0.2–0.6)) with Cag A positive strains conferring no additional risk. Interestingly, gastric atrophy was not associated with oesophageal adenocarcinoma. Conversely, *H pylori* infection was associated with an increased risk of oesophageal squamous carcinoma (OR 2.1; 95% CI 1.1–4.0) and this appeared to be mediated through gastric atrophy. *H pylori* was not associated with gastric cardia adenocarcinoma. The important caveat to these findings is that association does not always imply causation. To help establish this, we need a better understanding of the carcinogenic mechanisms that lead to oesophageal cancer, particularly at the gastro-oesophageal junction.

Treat to test? ►

▲ Numans ME, Lau J, de Wilt NJ, *et al*. Short-term treatment with proton-pump inhibitors as a test for gastroesophageal reflux disease. A meta-analysis of diagnostic test characteristics. *Ann Intern Med* 2004;**140**:518–27.

Reflux symptoms often respond dramatically to proton pump inhibitors (PPIs) and one suspects that many clinicians use this response as a method of validating their diagnosis of gastro-oesophageal reflux disease (GORD). Numans *et al* analysed how accurate was the symptomatic response to a PPI in diagnosing GORD. Their meta-analysis included 15 studies that compared the symptom response to a short course of a PPI with an objective measure of GORD, such as endoscopic oesophagitis or abnormal 24 hour pH monitoring. Overall accuracy was disappointing with a sensitivity of only 78% and a specificity of 54% (that is, 46% false positive responses) in comparison with an abnormal pH. Comparison with endoscopic oesophagitis was no more accurate, and accuracy based on comparisons with different structured questionnaires was worse. The high false positive rate is perhaps not surprising given placebo responders and responders with other acid sensitive conditions. What is remarkable was the poor sensitivity in patients with oesophagitis, ranging from 50% to 80%. This might partly reflect the doses of PPIs used or the short (two week) assessment of response. None the less, their analysis has important implications for dyspepsia management in primary care given, current UK guidelines and the increasing evidence for rebound increases in gastric acid secretion after PPI withdrawal (Gillen *et al*. *Gastroenterology* 2004).

Proof of the pudding is in treating it ►

▲ Czaja AJ, Carpenter HA. Decreased fibrosis during corticosteroid therapy of autoimmune hepatitis. *J Hepatol* 2004;**40**:646–52.

A growing body of evidence suggests that liver fibrosis is a dynamic process that can both progress and regress. Although it is widely recognised that disease specific therapy could delay or arrest the progression of fibrosis, clinicians and pathologists alike have remained sceptical about the reversibility of fibrosis.

In the current study, 325 liver biopsies from 87 well characterised patients with autoimmune hepatitis were reviewed under code by one pathologist and scored using the Ishak method. All patients were treated with prednisolone and 64 (74%) received azathioprine in addition. During 63 (6) months of treatment and follow up, fibrosis scores improved in 53% of patients (by ⩾2 grades in 35%) and remained unchanged in 26%. Fibrosis resolved completely in 20% of patients who had fibrosis at diagnosis and the frequency of cirrhosis was reduced from 16% to 11%. Changes in fibrosis correlated well with those for histological activity index. Both histological indices reflected the clinical status (initial presentation, remission, or relapse) at the time of sampling. The majority (72%) of the biopsies were ⩾1 cm long and 87% had ⩾4 portal tracts. Reduction in fibrosis score persisted even after excluding the specimens with confluent necrosis (13%), and hence avoiding stromal collapse being misinterpreted as fibrosis.

These findings strengthen previous observations from small case series that optimum treatment of autoimmune hepatitis could lead to reversal of fibrosis. Recommendations to attempt complete withdrawal of immunosuppressive therapy during remission should be debated in the context of its potential to reverse fibrosis.