LETTERS TO THE EDITOR

Why use Buscopan during diagnostic upper gastrointestinal endoscopy?

EDITOR,—In their audit of upper gastrointestinal endoscopy (*Gut* 1995; **36:** 462–7), Quine *et al* considered the issues surrounding the use of anticholinergic agents during upper gastrointestinal endoscopy. They found that although most procedures were performed without the use of either hyoscine butylbromide (Buscopan, Boehringer Ingleheim) or atropine these agents were still in use in certain centres and their findings indicate that Buscopan (dose range 10–40 mg) was used in 29% of procedures in East Anglia compared with 20·6% in the North West region and atropine (0·6 mg intravenously) was used in 11·2% procedures in the North West region and only 0·3% in East Anglia.

Buscopan is a spasmolytic or smooth muscle relaxant drug with anticholinergic activity. Its anticholinergic activity lasts for about 15–20 minutes and infusion experiments suggest that it is rapidly inactivated or excreted. It also has a sympathetic ganglion blocking action but this effect is unimportant in humans at the conventional dose of 20 mg commonly used. Its actions on the gastrointestinal tract include inhibition of motility in the stomach and colon, reduction of gastric acid secretion, and slowing transit through the small bowel. It also causes transient pylorospasm. Some studies have shown that oesophageal peristalsis is reduced by Buscopan[1] and that it relaxes the lower oesophageal sphincter.[2]

What is the rationale for the routine use of anticholinergic agents during diagnostic upper gastrointestinal endoscopy? The use of atropine to dry up secretions and for 'cardiac protection' is a benefit postulated[3] without there being any controlled data available. Reports have shown that anticholinergic premedication does not improve the quality of diagnostic endoscopy or reduce patient discomfort.[4 5] These investigators found no differences between groups with respect to gastric motor function or endoscopic quality as judged by the endoscopist or discomfort during endoscopy as judged by the patient.[4] Though atropine decreased both the amplitude and frequency of gastric peristalsis this objective effect of atropine did not have any effect on the outcome of the endoscopy.[5]

Anticholinergic premedication does not have any effect in reducing the incidence of cardiac arrhythmias during upper gastrointestinal endoscopy.[6] The audit by Quine *et al* reports on a total of eight patients who experienced significant cardiac arrhythmias that required treatment, including five patients who arrested. Four of these had been given Buscopan (two had been given doses of 40 mg). In another prospective study[7] comparing the use of Buscopan and Glucagon it was found that with intravenous Buscopan 20 mg the heart rate increased from a baseline of 94·4±11·1 to 126±19·5 beats per minute and there was a fall in the mean systolic, diastolic, and mean arterial pressure by 20–50 mm Hg in the Buscopan group. Four patients (aged 76–80 years) had hypotensive episodes immediately after intravenous Buscopan that lasted one to seven minutes. Thus Buscopan can cause both hypotension and a tachycardia.

Buscopan significantly reduces pressure in the lower oesophageal sphincter and in theory may facilitate gastro-oesophageal reflux.[2] The effect of an intravenous injection of 20 mg Buscopan on gastro-oesophageal reflux was evaluated in 112 consecutive patients undergoing barium meal evaluation.[8] This study concluded that the routine use of Buscopan was unlikely to spuriously increase the frequency or degree of gastro-oesophageal reflux seen during barium studies. However, radiological evaluation is an insensitive method of detecting gastro-oesophageal reflux and there are no data on oesophageal pH measurement after intravenous administration of Buscopan. Gastro-oesophageal reflux induced by Buscopan could predispose to the development of aspiration pneumonia. The audit by Quine *et al* reported 11 patients to have had pneumonia shortly after the procedure and 10 of these patients had received pharyngeal anaesthesia, which when combined with the presence of the fibreoptic endoscope interferes with glottic closure and swallowing and may cause pulmonary aspiration. We have no information on the use of anticholinergic agents in this group and wonder if these agents had any role in the development of pneumonia. Thus except for procedures such as injection of oesophageal varices and endoscopic retrograde cholangiopancreatography where the use of anticholinergic agents is clearly beneficial we would question their routine use during diagnostic upper gastrointestinal endoscopy.

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Reply

EDITOR,—The authors share Dr Chopra's concern about the use of anticholinergic medication for routine diagnostic upper gastrointestinal endoscopy and endorse the view that it should be reserved for therapeutic endoscopy where the benefit may outweigh the risks. Of the 11 cases of pneumonia reported six patients had received Buscopan and five had not. Therefore the use of Buscopan or Atropine did not seem to have an obvious effect on the risk of pulmonary aspiration though it is an interesting hypothesis.

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The nurse endoscopist

EDITOR,—As the advent of the nurse endoscopist seems ever more certain, I wish to raise my concerns about the provision of clinical information to the histopathologist who reports on the biopsy specimens.

We are already in a situation where most liver and gut biopsy specimens are sent by endoscopists or radiologists who are not primarily involved in the clinical care of the patient, and whose knowledge of their clinical history and medication is derived from a quick scan of the notes between appointments in a hectically busy department. Most pathologists will already be familiar with the terse statement 'raised LFTs', which is totally inadequate for a clinically useful assessment of a liver biopsy specimen. Two recent confusing samples received in this department were rendered interpretable only when histories of multiple myeloma and pelvic irradiation were eventually disclosed. Chasing clinicians and case notes is very time consuming and counterproductive.

Clearly the present situation, from the histopathologist's point of view, is not as good as it might be despite the fact that qualified medical practitioners are scanning the notes and entering clinical details on the request forms. But how will we fare when non-medical personnel are sending us specimens? Who will ensure the flow of accurate and relevant clinical information? Their training may encompass aspects of anatomy and physiology (*Gut* 1995; **36:** 795) but this is hardly sufficient training to rapidly assimilate and then distil the essence from a patient's medical case notes.

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Reply

EDITOR,—Dr Griffiths is understandably concerned about the provision of clinical information to the histopathologist by nurse endoscopists. Clinical information on histopathology forms is clearly of the greatest importance and this is one of many issues that will be covered in the training programme for nurse endoscopists.

However, I do not think he need fear that pathologists are suddenly going to receive a lot of incompetently filled forms with the advent of nurse endoscopists. Often forms are poorly filled in because of the 'hectically busy' life that most medical endoscopists lead. It is also well recognised that doctors are frequently poor form fillers. By contrast it is my experience that nurses are very diligent in this respect. I am sure that with the increasing numbers of gastrointestinal nurse specialists, obtaining appropriate clinical information from the notes will be well within the nurses' ability. I actually foresee a higher standard of