Letters

Audit of upper gastrointestinal endoscopy

EDITOR,—In their audit of 14 149 upper gastrointestinal endoscopies (Gut 1995; 36: 462–7), Quine et al identified 104 deaths occurring within 30 days. The authors hope their findings will encourage endoscopists to examine their own practices, but we are concerned that the numbers done for bleeding. Such data are important as we have recently found that within our hospital, inpatients accounted for a significant proportion (38%) of new referrals for upper gastrointestinal endoscopy, and that the 30 day ‘all cause’ mortality in these inpatients was 12%.1 Inpatients tended to be older and have multiple medical problems. The death rate was even higher in patients who were already in hospital for other reasons and subsequently had upper gastrointestinal bleeding, but even in 251 established inpatients who were endoscoped for reasons other than haemorrhage, mortality was 8%.2

Regarding gastrointestinal bleeding, a recent national audit studying a population of 12.5 million found an incidence of 103 upper gastrointestinal haemorrhages/100 000 people-year, of which 75% were endoscoped.2 Forty percent per cent died within 30 days and again, established inpatients fared worse (death rate 33%) than acute admissions with gastrointestinal bleeding. Applying these figures to the population (5 533 225) covered by the audit of Quine et al, 1420 gastroscopies would have been done for upper gastrointestinal bleeding during the four month period. Assuming a similar death rate (14%) from gastrointestinal haemorrhage, 199 deaths would have been expected. This expected 30 day mortality is almost twice that reported by Quine et al, and yet only includes endoscopies done for gastrointestinal bleeding (approximately 10% of the total 14 149 endoscopies). This suggests that in the audit by Quine et al, gastrointestinal haemorrhage or its associated mortality was less than expected, or that the 30 day mortality was underestimated.

Thus, without data on numbers of inpatients and numbers of gastrointestinal bleeds, endoscopists may be unable to adequately compare their own figures for morbidity/mortality with those provided by this audit.

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Reply

EDITOR,—In our audit inpatients represented 26% of all referrals to the endoscopy unit (65% outpatients, 9% unstated) and likewise these patients were older and had higher comorbidity ratings. The ‘30 day all cause’ death rate for inpatients was only estimated to be 2-6% and this is definitely an underestimation. Similarly the death rate at 30 days for gastrointestinal bleeding was underestimated.

A validation process performed on seven per cent of all endoscopies showed that the reporting of deaths for up to 30 days was the only area of possible significant error. There is no argument that an audit such as that performed by Rockall et al,1 which looked specifically at gastrointestinal bleeding, will provide more accurate estimates of mortality associated with gastrointestinal haemorrhage. Our audit looked at many diverse aspects of endoscopy practice and it is hoped that our results will be of great interest and encourage further study. As regards looking at complications related to endoscopy we feel that the earthing of seven deaths directly caused by the gastroscopy is the most pertinent discovery especially as it is probable that stricter attention to the care and monitoring of the patient while on the unit may have prevented some of these deaths.

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1 Rockall TA, Logan RFA, Devlin HB, Northfield TC. Incidence and mortality of acute upper gastrointestinal bleeding in the UK. Gut 1994; 35 (suppl 5): $47.

Audit of upper gastrointestinal endoscopy

EDITOR,—The risk of gastric perforation following endoscopy, highlighted by Quine et al (Gut 1995; 36: 462–7), might be greatest when there is coexisting gastric outlet obstruction. Gastric distension resulting from trapped air could, in those circumstances, predispose to perforation at the site of weakness in the stomach wall— that is, at the ulcer site.1 The ulcer site could be even weaker in gastric ulcers resulting from coprescription of corticosteroid drugs and non-steroidal anti-inflammatory drugs. Because air insufflation can aggravate gas trapping, the duration of the endoscopic procedure should be reduced to a minimum in high risk subjects, and the stomach should be promptly deflated after gastric biopsy.

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Local anaesthetic sprays and pneumonia after gastroscopy

EDITOR,—The recent audit of upper gastrointestinal endoscopy by Quine and colleagues (Gut 1995; 36: 462–7) was both timely and useful. I am concerned, however, that a conclusion will be drawn that was not intended. It is stated that there was in particular a link between the use of local anaesthetic sprays and the development of pneumonia after gastroscopy. I suspect that the link was present and after a longer period of time rather than simply occurring at the time of the procedure. I believe that anaesthetic spray should not be used if intravenous sedation is also planned. Many endoscopists, like myself, use local anaesthetic sprays in the absence of intravenous sedation but do use local anaesthetic throat spray routinely in non-sedated patients. I am not aware of data showing a link between local anaesthetic spray and pneumonia in non-sedated patients. The endoscopist must be aware that the report is taken at face value endoscopists such as myself could be unjustly criticised. It would be helpful if the authors could say how many of the 11 patients with pneumonia after receiving local anaesthesia also had intravenous sedation.

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Gastric metaplasia and Helicobacter pylori infection

EDITOR,—In their paper (Gut 1995; 36: 193–7), Khulusi et al showed that the extent of gastric metaplasia in the duodenum declines 10 months, on average, after eradication of H pylori. The authors conclude that the pylori infection event with the reduction of duodenal inflammation led the authors to conclude that the bacterium itself rather than the commonly held increase in gastric acid production is, at least in part, responsible for the development of gastric type epithelium in the duodenum. This result is in contrast with the results of Nakao et al,1 who excluded any significant change in gastric metaplasia one year after pylori infection. As the phenomenon did not differ between patients with eradicated and persisting infection, these authors concluded that the pylori related inflammatory process is less important than myself, however, do not routinely use intravenous sedation may increase the risk of cardiorespiratory complications.

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