

LETTERS TO THE EDITOR

Nasal oxygen during endoscopy

EDITOR,—Recently, the possible value of nasal oxygen in endoscopic retrograde cholangiopancreatography was studied by Haines and colleagues (*Gut* 1992; 33: 973–5). They found, not unexpectedly, that arterial oxygen saturation was increased during oxygen treatment, but the effect of oxygen treatment on heart rate was not significant.¹ They concluded, on the basis of the measured values for arterial oxygen saturation that nasal oxygen should be routinely used in patients over the age of 60 undergoing endoscopic retrograde cholangiopancreatography (ERCP),¹ thus supporting the guidelines published by the British Society of Gastroenterology.² These recommendations, however, were made because of previous numerous reports of hypoxaemia during endoscopy, but were not based on prospective studies relating hypoxaemia to clinical outcome. Such large scale prospective studies have not yet been published.

Instead, we may look at studies relating arterial hypoxaemia during endoscopy to the development of myocardial ischaemia and infarction. There are no data available on the association of hypoxaemia and myocardial infarction, and only two studies have focused on arterial hypoxaemia and associated myocardial ischaemia during upper gastrointestinal endoscopy.^{3,4} Murray and colleagues found significant ST depression on ECG in three of 20 patients during gastroscopy with only one patient having simultaneous oxygen saturation of less than 90%.³ We found, studying 15 patients undergoing ERCP, that significant ST depression occurred in 10 of 15 (67%) patients during endoscopy.⁴ Coherent ischaemia and episodic hypoxaemia was found in five patients, isolated ischaemia in seven patients, and isolated episodic hypoxaemia in 13 patients.⁴ Coherent ischaemia and a tachycardia was found in 10 patients, ischaemia without a tachycardia in no patients, and an isolated tachycardia in one patient.⁴

These results suggest tachycardia is more important than hypoxaemia in the pathogenesis of myocardial ischaemia during upper gastrointestinal endoscopy. Making pulse oximetry or oxygen treatment part of the standards of care guarantees that the experiments necessary to document its efficacy will never be carried out because we have ruled out a control group, and efficacy cannot be measured in terms of frequency of oxygen desaturation, but only in clinical outcome. Thus, the role of hypoxaemia to precipitate cardiac or other complications remains to be shown. In Denmark, clinical recommendations for monitoring and oxygen treatment during endoscopy have not been

issued, as it seems premature considering the lack of scientific data in support.⁵

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Reply

EDITOR,—We note the above comments of Dr Rosenberg with interest. We would entirely agree that the success of any recommendation to improve the safety of gastrointestinal endoscopy must ultimately be judged by the criterion of clinical outcome. As we are dealing with (fortunately) relatively rare major adverse clinical outcomes (for example myocardial infarction, respiratory arrest or death), we agree that very large scale prospective studies are required for definitive proof. In the absence of such studies, we must draw conclusions from the limited data available and make appropriate recommendations to ensure that our current practice is as free of avoidable risks as we can achieve.

On current evidence, it seems reasonable to assume that occurrence of both arterial hypoxaemia and myocardial ischaemia (as evidenced by ST segment depression) may identify a subgroup of patients particularly at risk of a significant adverse outcome. It is not possible to be certain whether these are independent risk factors in an individual, or whether they are additive. It is possible to be certain that one of them, arterial hypoxaemia, is to a large extent preventable by a simple (and cheap) modification to current practice. We therefore entirely stand by the recommendations of the British Society of Gastroenterology¹ but agree that there is no cause for complacency. We would agree with Dr Rosenberg that the occurrence of significant tachycardia in our own series of patients is worrying and that the reduction of this additional risk factor to enhance patient safety is clearly needed.

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von Willebrand factor in upper gastrointestinal tract inflammation

EDITOR,—The endothelium is attracting attention in the study of diseases of the lower intestinal tract because it may be related to the pathogenesis of impaired immune responses in situ in ulcerative colitis.¹ Stephens *et al* recently reported raised values of von Willebrand factor antigen (vWFAg, a specific endothelial product) in inactive as well as active inflammatory bowel disease suggesting that vascular injury may be involved in its pathogenesis² and Sawyerr *et al* found anti-endothelial cell antibodies in Crohn's disease, but not in ulcerative colitis.³

Despite this interest in the endothelial cell in diseases of the lower intestine, little seems to be known about their possible role in inflammatory conditions of the upper intestinal tract. In order to investigate a possible role of vWFAg in upper gastrointestinal tract disease, serum was obtained from 44 patients presenting for endoscopy. vWFAg was measured by ELISA, statistics were by Mann-Whitney on Minitab, and Ethical Committee approval was obtained.

Endoscopic findings were normal in 16 patients (five smokers), whose mean (standard deviation) vWFAg was 118 (36) international units per decilitre (IU/dL). There was no change in vWFAg in 28 patients (12 smokers) whose endoscopic findings were abnormal at 97 (31). Subsequent analysis according to endoscopic finding (the locality of the lesion, (oesophageal, gastric or duodenal), the severity of the finding (mild, moderate or severe), or the presence of hiatus hernia) also failed to identify any subgroups with a raised vWFAg.

Hence although there appears to be evidence of vascular injury in inflammatory bowel disease such as ulcerative colitis or Crohn's disease,² normal levels in upper gastrointestinal tract inflammation suggest that the involvement of endothelial cells are not part of this disease process.

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