LETTERS TO THE EDITOR

Towards safer endoscopic retrograde cholangiopancreatography (ERCP)

EDITOR,—Diagnostic and therapeutic ERCPs are the most dangerous procedures regularly performed in the United Kingdom. A recent series of cases involving 25 expert erectors in ERCP.1 Reported data vary according to the completeness of their collection. Most retrospective (certainly multicenter) studies minimize the problems, and prospective analyses always show higher rates. Another point is that the risk of a bad outcome is certainly influenced by the severity of the patient’s presenting illness and burden of concomitant diseases. We cannot assess our results, or compare them with others, without being able to describe the risk factor spectrum of our patient material.2 Furthermore, the significance of any risk of complication must be judged against the available alternative techniques in that specific clinical context.

Pancreatitis is the commonest complication of ERCP. Thornton and Axon give it only a few lines, stating that ‘clinically significant’ pancreatitis occurs in only about 2% of procedures.1 It depends what you mean by ‘clinically significant’. Much higher figures have been published.2 We are still seeing this complication with distressing frequency at Duke University Medical Center. In a strict prospective computer-based study using agreed definitions, we have recorded a total of 160 complications in 3001 ERCP procedures performed over the last three years. One hundred and sixty-two patients had pancreatitis, an incidence of 3.7%; most cases (55%) were graded as mild (less than three days in hospital). Sphincter of Oddi manometry carries a 12% pancreatitis rate at this institution.

The whole problem of pancreatitis after ERCP has been discussed exhaustively by Sherman and Lehman recently in an important review article, with 181 references.3 Unfortunately, there have been too many breakthroughs in understanding or prevention. The hope that non-ionic contrast materials might be safer has not been realised in a large randomised controlled trial.4 Many studies have failed to show any protective value of drugs given before ERCP; the latest showed that prophylactic somatostatin actually increased the risk of pancreatitis.5

It is surprising that the leading article has nothing to say about training, as emphasised by my colleagues in the accompanying letter. Although difficult to prove, it is probable that quality training and substantial ongoing experience have some effect on the results of our interventions. Indeed, there is a danger that widespread application of these potentially dangerous techniques in inexperienced hands will cause them to fall into disrepute.

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Location of superoxide anion in the human colonic mucosa

EDITOR,—We read with interest the article by Oshinati et al (Gut 1993; 34: 936-8) regarding the location of superoxide anion generation in the human colonic mucosa. We have some concern about the interpretation and the significance of the data, which is probably led the authors to characterise superoxide as the oxygen radical generated in the colonic mucosa especially in ulcerative colitis. The methodological approach used in the study was based on the morphological evaluation of nitroblue tetrazolium (NBT) reduction by endothelial, epithelial, and infiltrating mononuclear cells in the colonic mucosa. Undoubtedly, NBT is reduced by superoxide anion4; however, the reduction of NBT is not so specific, and other molecules can favour its reduction in the cell environment. Accordingly, xanthaiox (which is localised in the endothelium) readily reduces NBT also by a superoxide-independent way, which is probably related to a direct NBT electronic transfer with a bypass of the superoxide forming enzyme flavin cent. Furthermore, cell debris and the enzyme systems that reduce NBT physiologically, so that morphological techniques based on tetrazolium dye reduction precipitation have been largely used to quantify myocardial infarct size, because necrosis areas lack dehydrogenase activity and therefore fail to reduce NBT and to stain.5 There is evidence that the spontaneous reduction of NBT mediated by tissue homogenates can be inhibited by iron and copper chelators, as well as by mitochondrial electron transport chain blockers,6 thus pointing to a role for transition metals and mitochondria in tissue NBT reduction. In this context, it is noteworthy that the radicals generated by inflammatory cells may increase tissue ‘free’ iron and copper concentrations, as a result of ferritin iron mobilisation.

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Reply

EDITOR,—We are grateful to our colleagues from Duke University for drawing attention to our leading article. We agree with the points that they have made. We concentrated mainly on the areas in ERCP where innovations have led to greater safety and as Dr Cottle points out few advances have been made recently where pancreatitis occurs after ERCP.

The question of endoscopic training is of particular relevance in preventing complications, not just in ERCP but in other forms of endoscopy too and our colleagues are correct in drawing attention to this. Regulation of training in endoscopy in the United Kingdom are at present non-existent and although the British Society of Gastroenterology has made recommendations, the Royal Colleges and the Joint Committees for Higher Medical and Surgical training do not insist either on certification of endoscopists or accreditation of endoscopy units for training purposes. We understand that recent moves have been made to introduce a coordination of endoscopy units for training purposes and that BSG guidelines with some modification are probably the criteria that will be used in the accreditation process.

If this does happen then it is to be welcomed. It is unlikely that endoscopists in the UK will have to undergo a certification or recertification procedure as in the United States, but training in endoscopy is on the agenda of committees in the European Community and this may well in the future lead to legislation that will restrict endoscopy to those who have received suitable training.

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