LETTERS TO THE EDITOR

Towards safer endoscopic retrograde cholangiopancreatography (ERCP)

EDITOR,—Diagnostic and therapeutic ERCPs are the most dangerous procedures regularly performed by medical gastroenterologists. The leading article from Thornton and Axon (*Gut* 1993; **34**: 721–4) discussing safety aspects is pertinent and helpful, but omits several key points and references.

The first problem is knowing what published figures mean. Bad outcomes cannot be discussed, studied, or minimised, without clear definitions. Suggested definitions for complications (and severity) were made recently after a consensus conference involving 25 experts in ERCP.[2] Reported data vary according to the completeness of their collection. Most retrospective (certainly multicenter) studies minimise the problems, and prospective analyses almost always show higher rates. Another issue 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.' It depends what you mean by 'clinically significant'. Much higher figures have been published.[3] 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 thirteen (71%) were 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 no major 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.[5,6]

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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1 Cotton PB, Lehman G, Vennes J, Geenen JE, Russell RCG, Moyers WG, *et al*. Endoscopic sphincterotomy complications and their management: an attempt at consensus. *Gastrointest Endosc* 1991; **37**: 383–93.
2 Cotton PB. Endoscopic management of bile duct stones (apples and oranges). *Gut* 1984; **25**: 587–97.
3 Sherman S, Lehman GA. ERCP- and endoscopic sphincterotomy-induced pancreatitis. *Pancreas* 1991; **6**: 350–67.
4 Bedford RA, Johnson GK, Geenen JE, Lehman G, Frakes J, Ryan M, *et al*. Does the use of non-ionic contrast prevent post-ERCP pancreatitis: results of a prospective multicenter study. *Gastrointest Endosc* 1993; **39**: 315.
5 Sternlieb JM, Aronchick CA, Retig JN, Dabezies M, Saunders F, Goosenberg E, *et al*. A multicenter, randomized, controlled trial to evaluate the effect of prophylactic octreotide on ERCP-induced pancreatitis. *Am J Gastroenterol* 1992; **87**: 1561–6.
6 Meier PB. Preventing ERCP-induced pancreatitis. *Am J Gastroenterol* 1992; **87**: 1536–9.

EDITOR,—Drs Thornton and Axon recently reviewed issues pertinent to the safety of ERCP. We feel, however, that the lack of any discussion about the role of training is a serious omission. Historically, scant attention has been paid to how endoscopists acquire their skills. Training in ERCP ranges from self instruction to the highly structured 'Third Tier' programmes for gastroenterology fellows currently popular in the United States. We have considered these enormous variations in training, as well as assessment of procedural competence, elsewhere.[1,2] These issues cannot be ignored, however, if the safety record of ERCP is to be maintained or – preferably – improved. As Cotton has pointed out,[3] a complication is an adverse and undesired outcome that does not necessarily reflect negligence or poor technique. We cannot eliminate complications of ERCP entirely, but appropriate supervised training, insistence on at least basic diagnostic and therapeutic skills, and ensuring that those who perform ERCP maintain and enhance their skills should minimise avoidable complications.

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1 Jowell P, Baillie J. Endoscopic training, present and future. *Gastrointest Endosc Clinics of North America* 1992; **2**: 299–311.
2 Baillie J, Ravich WJ. On endoscopic training and procedure competence. *Ann Intern Med* 1993; **118**: 73–4.
3 Cotton PB. Complications, comparisons and confusion: commentary. *Annual of GI Endoscopy*. London: Current Science, 1970: 7–9.

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 Cotton points out few advances have been made recently where pancreatitis occurs after ERCP.

The question of endoscopy 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. Regulations governing 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 support the concept of the accreditation 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 in the future lead to legislation that will restrict endoscopy to those who have received suitable training.

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Location of superoxide anion in the human colonic mucosa

EDITOR,—We read with interest the article by Oshitani *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 have apparently 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 anion[1,2]; however, the reduction of NBT is not so specific, and other molecules can favour its reduction in the cell environment. Accordingly, xanthine oxidase (which is localised in the endothelium[3]) 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 centre.[4,5] Furthermore, cell dehydrogenase enzyme systems reduce NBT physiologically, so that morphological techniques based on tetrazolium dyes 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.[6] 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,[2] 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[7]