Towards safer endoscopic retrograde cholangiopancreatography (ERCP)

EDITOR,—Diagnostic and therapeutic ERCPs are the most dangerous procedures regularly performed.1,2 There is no 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 address safety problems. 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.1 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 2% of procedures.'1 It depends what you mean by 'clinically significant'. Much higher figures have been published. 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 five pancreatic complications, incidence of 3.7%; most cases (55%) were graded as mild (less than three days in hospital). Sphincter of Oddi manometry carry 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 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 fail to disperse.

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Location of superoxide anion in the human colonic mucosa

EDITOR,—We read with interest the article by Oshinath 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 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 anion; 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) 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. Furthermore, cell debris of endogenous or exogenous origin can 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 of cytochrome electron transport chain blockers, 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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