LETTERS TO THE EDITOR

Adult coeliac disease, dermatitis herpetiformis and smoking

EDITOR.—Snook and colleagues report that cigarette smoking seems to exert a protective effect against the development of adult coeliac disease (Gut 1996; 39: 60–2). Dermatitis herpetiformis, an uncommon blistering skin disease of unknown aetiology. Importantly, gluten sensitive enteropathy is present in almost 100% of patients after gluten loading, giving rise to the hypothesis that coeliac disease age at the time of the study was 50–1 year.

This finding was not reported before and gives further support to the hypothesis that dermatitis herpetiformis and coeliac disease may be points on a spectrum of disease. Our study was on a small series of patients and it would, therefore, be useful to more patients in a larger cohort. Why patients with dermatitis herpetiformis and adult coeliac disease smoke less is uncertain. Snook et al suggest that, in adult coeliac disease, smoking history, permitting a number of pack years smoked (one pack year=20 cigarettes a day for one year). Twenty nine cases were included in the study. Patients were interviewed to identify their cigarette smoking history, permitting determination of a spectrum of disease.

Risk factors for pancreatitis

EDITOR.—Dr De Beauch, Carter, and Palmer in their thought provoking editorial have examined a number of possible risk factors for pancreatitis occurring after ERCP (Gut 1996; 39: 799–800).

However, several studies have failed to show that factors commonly thought to be at fault do actually present a risk, for example, the presence of concomitant disease of the pancreas, bacteraemia, etc.1,2 Sphincter of Oddi manometry seems to be a risk factor3 as does sphincterotomy for stones, although this does not seem to be related to duct clearance. It is clear therefore that pancreatic injury, although not uncommon, does not necessarily cause pancreatitis, the cause of pancreatitis is unknown. We would like to propose that the glutaraldehyde residues remaining after endoscope cleaning, could be at fault.

This hypothesis is based on our own experience following an outbreak of pancreatitis related to changes in our endoscope cleaning methods. Our usual rate of diagnostic pancreatic abnormalities is approximately 1%, which increased to 15% with a change from manual cleaning and use of the Keymed automed disinfectant (Keymed, Stock Road, Southend on Sea), to an automated closed circuit washing machine ( Customs Ultrasonics Automatic System 83-2, Specialist Endoscopy Equipment, Ormskirk, Lancashire). After a worrying six week period and based on the knowledge that even small spaces of glutaraldehyde to mucosal membranes and colonic epithelium, we very carefully examined our change in cleaning practice. It became apparent that rinsing of the elevator wire channel (forceps carrier) was possibly less than adequate after automatic cleaning with glutaraldehyde. Our practice now is to rinse the elevator wire channel manually with at least six rinses of sterile water. Since then, examination of our pancreatic ducts for the past year has shown that of 309 ERCPs, there was one case of pancreatitis in 174 diagnostic ERCPs (including 123 pancreatograms). An incidence of 0.6%. The one case was in a 15 year old girl with a history of abdominal pain. Of 74 patients undergoing stent insertion, with or without access sphincterotomy, there was one case of pancreatitis (1.3%). There were four cases with high pancreatitis among 61 sphincterotomies for stones (an incidence of 6.5%). This appeared unrelated to success or otherwise of duct clearance. Four cases were mild with discharge of the patient from hospital within five days. One case where duct clearance was inadequate was however, severe.

We believe that there is circumstantial evidence to suggest that glutaraldehyde might be implicated in the aetiology of post-diagnostic ERCP pancreatitis. Proof of this hypothesis will require an animal model, but in the meantime we recommend that units carrying out ERCP, carefully evaluate their rinsing procedures and consider the possibility of changing them if they are found to be less than satisfactory.

Gastric ulcers

EDITOR.—Interest in the effects of corticosteroids on the healing of gastric ulcers goes back a long time. The paper by M Carpena deKaski and colleagues is a valuable contribution to this area, which demonstrates that corticosteroids do reduce the reparative repair of the epithelium in experimental cryoprobe-induced ulcerations in the rat stomach (Gut 1995; 37: 613–6). The earliest work they cite on the healing of experimental ulcer with prednisone is that of Kuwawaya and Eastwood, published in 1988.1 But their memories do not go back far enough. My colleagues, with Franklin Holland, and I presented what we believed was the only extant report at that time (1957) on the effects of cortisone and corticosteroids on the healing of gastric ulcers in an experimental study in the dog. Using explants of the entire gastric wall of this species transplanted to the anterior abdominal wall, and protected by a mechanical metal guard, we induced circular ulcers whose diameters ranged from 5 to 25 mm and included the muscularis mucosae. Doses of cortisone of 10 or 20 mg/kg, or ACTH of 5 or 10 mg/kg, significantly delayed the healing but did not completely inhibit it. So that from a purely historical point of view, no report on some of the mechanisms that may be operative in the healing of human gastric ulcers is indeed a welcome addition to our knowledge.

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5 Corrasto OJ, Osman J, Davies RJ. Asthma and response to smoking history, permitting a spectrum of disease.

6 The Mount Sinai School of Medicine, New York, NY 10029, USA
