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 is 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 and dermatitis herpetiformis may be part of a spectrum of disease.[1-3] Because the enteropathy is usually mild in dermatitis herpetiformis, overt malabsorption is rare.[2] We therefore wanted to assess whether the finding by Snook *et al* was also present in patients with dermatitis herpetiformis, as this would provide further evidence of a link between dermatitis herpetiformis and coeliac disease.

Twenty nine patients, 20 male and nine female, with dermatitis herpetiformis were identified. Only patients with histological and direct immunofluorescence findings compatible with a diagnosis of dermatitis herpetiformis were included in the study. Patients were interviewed to identify their cigarette smoking history, permitting determination of number of pack years smoked (one pack year=20 cigarettes a day for one year). Twenty nine age and sex matched controls with atopic eczema were also recruited and cigarette smoking history determined in the same way. A Wilcoxon sign ranked matched pair test was used to analyse pack year smoking data. The average age at the time of the study was 59·1 years (SD 14·12). We found that patients with dermatitis herpetiformis smoked significantly less than controls ($p<0·0009$, median difference 10 pack years).

This finding has not been 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 corroborate these findings 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, the immunomodulatory effects of smoking may be important and this would also be relevant to dermatitis herpetiformis. Most patients with dermatitis herpetiformis and coeliac disease will have been referred to a dietitian and so receiving counselling and general health advice may be a possible contributing factor, although this seems an unlikely explanation for the magnitude of effect seen. Further confirmation of this finding is needed, both in coeliac disease and dermatitis herpetiformis.

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Risk factors for pancreatitis

EDITOR,—Drs De Beaux, Carter, and Palmer in their thought provoking editorial have examined a number of possible risk factors for pancreatitis occurring after ERCP (*Gut* 1996; **38:** 799–800).

However, several studies have failed to show that factors commonly thought to be at fault do actually present a risk, for example, type of contrast media used,[1] acinarisation of the pancreas,[2] bacteraemia, etc.[3] Sphincter of Oddi manometry does seem to be a risk factor[4] as does sphincterotomy for stones, although this does not seem to be related to duct clearance. It is clear therefore that particularly for diagnostic ERCP, the cause of post-ERCP pancreatitis is unknown. We would like to propose the possibility that 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 pancreatitis of approximately 1% increased to 15% with a change from manual cleaning and use of the Keymed autodisinfector (Keymed, Stock Road, Southend on Sea), to an automatic 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 traces of glutaraldehyde are toxic to mucus membranes[5] and colonic epithelium,[6] we very carefully examined our change in cleaning practice. It became apparent that rinsing of the elevator wire channel (forceps raiser) was possibly less than adequate after automatic cleansing 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 pancreatitis rate 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 recurrent pancreatitis since infancy. Of 74 patients undergoing stent insertion, with or without access sphincterotomy, there was one case of pancreatitis (1·3%). There were four cases of 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 indicated 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.

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Gastric ulcers

EDITOR,—Interest in the effects of corticosteroids on the healing of gastric ulcers goes back a long time. The paper by M Carpani deKaski and colleagues is a valuable contribution to this area, which demonstrates that corticosteroids do reduce the regenerative 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 Kuwayama and Eastwood, published in 1988.[1]

But their memories do not go back far enough. My colleagues, who included the late Franklin Hollander, and I presented what we believed was the only extant report at that time (1957) on the effects of cortisone and corticotropin on the healing of gastric ulcers in an experimental study in the dog.[2] 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 this new, infinitely more sophisticated, 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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