

shows a low mitotic rate, it has regenerative potential when subjected to mechanical stresses.² We consider it possible that re-epithelialisation had occurred from a reservoir of intact epithelial cells lying in crypts, in a manner analogous to the re-epithelialisation of a split thickness skin graft from epithelial cells preserved in adnexal structures.

Our observation of relative preservation of crypt lining epithelium during lithotripsy may suggest that contrary to the authors' hypothesis of damage by acoustic cavitation, surface epithelial damage may result from direct stone fragment abrasion. Crypt lining cells would be protected from such abrasion, although still subject to acoustic cavitation.

In vitro lithotripsy to stone containing gall bladders may not faithfully reproduce the full range of *in vivo* changes. In particular vascular damage and haemorrhage, known to be direct effects of shock waves,³ together with the release of oedema fluid, may force apart tissue planes (such as the interface between epithelial cells and stroma) in the perfused organ.

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Reply

SIR, - We completely agree with Stephenson *et al* that *in vitro* shockwave application to tissue may not faithfully reproduce the full range of *in vivo* changes. Thus, in our experimental design we concentrated on acute and chronic *in vivo* studies. For scientific and ethical reasons we used animals to analyse the tissue reactions under piezoelectric shockwave application.¹ In addition to the extensively described acute *in vivo* shockwave effects in the wall of the gall bladder (bleeds, oedema), the liver (subcapsular and interstitial bleeds, venous thrombosis) and other organs, it is of particular interest, that in our *in vitro* experiments with surgically removed human gall bladders no noteworthy lesion could be detected in the gall bladder wall under piezoelectric shockwave application. The finding, that shockwave induced

damage only occurs in perfused tissue, supports the hypothesis that cavitation is the main cause for tissue damage under shockwave application.²

Stephenson *et al* found focal epithelial denudation in stone containing human gall bladders excised within 48 hours after shockwave lithotripsy. He states that focal epithelial denudation is a mechanical effect induced by rubbing between the stone(s) and the epithelium during shockwave application. This idea is supported by our findings in non-stone containing gall bladders of dogs. Here, the epithelium itself was intact, whereas relevant lesions in the gall bladder wall, liver, etc, could be detected.

In conclusion, two types of tissue damage may occur in shockwave lithotripsy of gall bladder stones due to different mechanisms: first, slight mechanical damage as seen in the focal denudation of the gall bladder epithelium, which regenerates, as described by Stephenson *et al* within five days. Second, more important and still not yet completely explicable tissue reaction under piezoelectric shockwaves consists of lesions between the interfaces of the organs in the shockwave path. Even if we did not observe persisting noteworthy morphological changes in the animals of the chronic study autopsied three weeks after shockwave application, an unreserved application of shockwaves is not justified.

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Chronic colitis after *Aeromonas* infection

SIR, - We were interested by the cases described by Willoughby *et al*. (*Gut* 1989; **30**: 686-90). We wish to report a similar case where chronic colitis developed after an infection with *Aeromonas hydrophila*.

A 59 year old man with a past history of pulmonary tuberculosis and partial gastrectomy for duodenal ulcer developed diarrhoea while on holiday in France. He became pyrexial and dehydrated and was admitted to hospital on his return. On examination he was unwell and sigmoidoscopy to 15 cm revealed diffuse erythema and friability. Investigations revealed *Aeromonas hydrophila* in three consecutive

stool samples and rectal biopsy showed a moderately active non-specific proctitis. Two weeks after his admission discrete ulceration and a thickened polypoid mucosa were evident in the descending and distal transverse colon at a limited colonoscopy. The appearances were thought to favour Crohn's disease and the histology was equivocal. He was treated with corticosteroids and was discharged feeling well three weeks after admission on maintenance sulphasalazine. Six months after presentation repeat colonoscopy showed a pancolitis which histologically looked more like Crohn's disease than ulcerative colitis. He remained asymptomatic until four years after his initial admission to hospital when diarrhoea recurred. A sigmoidoscopy showed a granular mucosa and barium enema showed a pancolitis. Rectal biopsy favoured the diagnosis of ulcerative colitis.

This patient had no bowel symptoms before his infective diarrhoea and it seems likely, as in the cases reported by Willoughby *et al*, that the chronic colitis was triggered by the enteric infection. There is much evidence to link the onset of chronic colitis to enteric infection with a variety of pathogens which more usually cause a self-limiting inflammatory colitis like *Aeromonas hydrophila*. Examples other than those referred to by Willoughby *et al* include cases where chronic disease was linked to infections with *Staphylococcus aureus*,¹ *Entamoeba histolytica*,² and *salmonella sp.*³ Furthermore in a prospective study of acute colitis *E coli* with characteristics associated with pathogenicity were found in patients in their first attack of ulcerative colitis.⁴

We agree that wide ranging microbiological studies are necessary early in the course of chronic colitis to elucidate this relationship more fully.

We thank Dr G Neale for allowing us to report a patient in his care.

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Omeprazole v ranitidine

SIR,—I was interested to read the recent paper by Delchier *et al*¹ in which omeprazole 20 mg once daily was compared with ranitidine 150 mg bd in the treatment of 'resistant' duodenal ulcers. The findings that the two treatments are equivalent is at variance with other studies in which omeprazole has been compared with ranitidine in the treatment of duodenal ulcers^{2,3} including 'resistant' ulcers.⁴

The authors comment on some of the reasons for this unexpected finding. It is difficult to accept that duodenal ulcers can be regarded as truly 'resistant' after only six weeks' treatment with an H₂ blocker,⁵ and it would seem important to have established that the patients were compliant with the prescribed H₂ blocker before entering such a study. Moreover, 60% of patients were asymptomatic at entry to the study. In practice, outside the constraints of a clinical trial, such patients would not be likely to be detected, and certainly not identified as a separate group, for endoscopy to confirm the healing of straightforward duodenal ulcers is uncommon.

The unusual findings in this study seem likely to be a reflection of patient selection, and do not bring into question the consistent findings from comparative studies of omeprazole and H₂ blockers which show a greater proportion of patients treated with omeprazole healed and experiencing symptom relief⁶ within two to four weeks, irrespective of the presence or absence of complicating factors.

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