Correspondence

References


Reply

Sir,—We are grateful for the interest which Drs Savarino and Mela have expressed in our work and for their detailed comments.

The aim of our study was to determine whether there was significant regional variations in intragastric pH. In view of the constant changes in gastric size and shape, we believe that anchoring the electrode to the mucosa is essential in order to monitor local intragastric pH over a period of time and while the subject undertakes normal activities. The endoscopic procedure for clipping the electrode to the mucosa is technically difficult and we do not advocate its use for routine studies of intragastric pH. It is, however, a valuable new technique for scientific studies in which precise localisation of electrodes in the upper gastrointestinal tract is required. The loss of satisfactory 24h recordings in two of our subjects was not because of damage to the electrodes by the fixation procedure but to malfunction of the digitrapper recording boxes.

We analysed nocturnal pH from 2300 to 0500 h as the patients all remained recumbent and fasted over this period. On looking at the individual pH traces, there is no evidence of differences between antral and body pH later in the morning.

We agree that taking all the patients together, there was no statistically significant difference in night time antral and body pH. We felt, however, it only honest and correct to draw attention to the episodic rise in nocturnal pH which occurred in two of our subjects and which was different in the antrum and body. The cause of this episodic rise of nocturnal pH is unclear and duodenogastric reflux is only one possible explanation. We have recently performed more detailed studies in a further subject, showing intermittent rise of night time pH.1 Aspiration of gastric contents during some of the episodes produced abundant amounts of non-bile stained fluid with a pH the same as that registered by the in situ electrode. On other occasions, however, when the pH was raised no aspirate could be obtained from the stomach raising the possibility that the electrode was recording mucosal pH caused by contraction of the stomach. We certainly agree with Drs Savarino and Mela and also with Dr Hostein and colleagues that studying intragastric pH (even with fixed dual electrodes) is not a specific tool for diagnosing duodenogastric reflux.

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References


Piezoelectric shockwave fragmentation of biliary calculi

Sir,—We were interested to read that EI and colleagues (Gut 1989; 30: 680–5) observed no macroscopic or light microscopic changes in the walls of their 10 stone containing human gall bladders subjected in vitro to 1000–4000 discharges from the Wolf Piezolith 2200 lithotripter.

We compared the macroscopic, light microscopic and scanning electron microscopic changes in stone containing gall bladders from 16 patients treated by 2000–2500 discharges from a Wolf Piezolith 2300 lithotripter at power level 3–4 for between four hours and five days before planned cholecystectomy. In comparison with stone containing gall bladders from age and sex matched control cases, the gall bladders excised within 48 hours of lithotripsy showed focal epithelial denudation, mural oedema, and serosal and mucosal vasodilatation with petechial haemorrhages.1 Scanning electron microscopy of the mucosa showed denudation of groups of columnar epithelial cells, while intact cells were seen in the mouths of crypts. Our one gall bladder removed five days after lithotripsy was lined by intact epithelium.

Although human gall bladder epithelium normally
shows a low mitotic rate, it has regenerative potential when subjected to mechanical stresses. We consider it possible that re-epithelisation had occurred from a reservoir of intact epithelial cells lying in crypts, in a manner analogous to the re-epithelisation 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, 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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References


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. 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.

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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References


**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
Piezoelectric shockwave fragmentation of biliary calculi.

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Gut 1989 30: 1435-1436
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