LETTERS TO THE EDITOR

Effect of *Helicobacter pylori* on the gastric mucus gel

EDITOR,—In their paper on the basis of viscosimetric studies performed on human gastric mucus Markesich *et al* challenge the concept that *Helicobacter pylori* exerts unfavourable effects on the mucous layer coating the gastric mucosa (*Gut* 1995; 36: 327–9).

Their results are clearly at variance with the bulk of the experiments carried out in this area¹ and Markesich and colleagues try to show the reasons for this discrepancy by pointing out the potential drawbacks of the previous studies.

Even if we accept Markesich's concept, however, that the results of some experiments on *H. pylori* have to be discarded because they are not performed on human mucus, other data are consistent with a mucus damaging effect of the micro-organism in humans. Assessing the quality of gastric soluble mucus in humans we found that, although on the whole mucus is normal both in chronic gastritis and in duodenal ulcer,² the lowest values are detectable in subjects with *H. pylori* infection.³

Sidebotham *et al* also observed a breakdown of gastric mucus in patients infected with *H. pylori*, although they ascribed this phenomenon to a carbonate-bicarbonate buffer at the mucosal surface due to the urease activity of the germ, rather than to a direct mucolytic activity.⁴ Measurements of the gastric mucus gel thickness in vivo showed that the mucous coating is significantly thinner in *H. pylori* infected patients,⁵ possibly resulting from increased erosion.

In keeping with the above results *H. pylori* infection seems to reduce mucosal hydrophobicity of human gastric mucus, which returns to normal after successful eradication.⁶

The reason for the discrepancy of Markesich's results is probably the method used to examine mucus. Reference is made to a study⁷ to justify the use of viscosity analysis for assessing the characteristics of mucus, but it is well recognised that viscosimetry is an unreliable technique for evaluating mucus properties.⁸ The finding that gastric mucus viscosity is increased in duodenal ulcer patients⁷ is only a further demonstration of how misleading the method can be.

As only luminal mucus was examined in Markesich's study, it must be also considered that proteolytic enzymes produced by *H. pylori* could promote a greater peptic erosion of the adherent mucus gel with consequent increase in the mucoprotein content of gastric juice influencing viscosity measurements. Eradication of *H. pylori* would clearly reduce mucus shedding and thus apparently decrease the viscosity of intraluminal mucus.

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Reply

EDITOR,—Guslandi takes issue with our finding that gastric mucus gel in *H. pylori* patients has a higher viscosity than in the unaffected. We simply gathered and tested the clear mucus gel that is normally present in the stomach. We used standard methodology for evaluating the viscosity of a gel. Our data were both consistent and reproducible.

There are many possible reasons why the gastric mucus gel in patients with *H. pylori* infection may be more viscous than those without. As the mucus cells seem to be under pressure to actively secrete, the gel may be younger than in uninfected subjects. It may also contain many more cells and cellular debris including DNA, which will in itself enhance viscosity. We make no apologies for the fact that Dr Guslandi studied soluble mucus and obtained different results.¹ This should not be a surprise as soluble mucus is 'used' mucus that has completed its function and is probably best considered a waste product. The fact that the mucus gel coat might be thinner in *H. pylori* infected patients, tells us nothing about the rate of synthesis, erosion, effectiveness or viscosity. We have previously shown that changes in mucosal hydrophobicity can be separated from the *H. pylori* infection.² The changes in hydrophobicity seem more likely to be due to the inflammatory infiltrate (as has been previously shown in the colon) than for any factor made by the bacteria.³ More than 40 years ago, Hollander described increased viscosity in response to damage and other workers in gastric physiology who previously published on breakdown of gastric mucus have had no difficulty incorporating our findings.^{4,5}

The history of peptic ulcer disease has been one of exploring what turned out to be blind alleys. Researchers have seen their cherished beliefs destroyed and discovered that their lifelong work has not been in the main stream, but has been in investigating only a minor tributary.⁶ The truth will be discovered over time and only by additional experimentation.

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Audit of percutaneous liver biopsy

EDITOR,—The 50% liver biopsy rate cited by Gilmore *et al* (*Gut* 1995; 36: 437–41) for patients aged >65 with suspected malignancy would only be justified if concurrent clinical, biochemical, and ultrasonographic stigmata were equivocal. Given the fact that the diagnostic specificity of ultrasonography can be as high as 96.2%, with corresponding likelihood ratio=18 (depending on subtype of ultrasonographic stigmata),¹ you can utilise the principles of Bayes' theorem² to predict the post-test probability of hepatic metastases in a subgroup of patients who already have clinical and biochemical stigmata of this diagnosis. Under such circumstances the minimal increment in post-test probability, generated by the adjunctive use of needle biopsy, might well be largely offset by the risk of procedure related morbidity and mortality.

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EDITOR,—One of the benefits of this audit is that it has supported the widely held belief that coagulopathy predisposes to haemorrhagic complications following liver biopsy. It is surprising that previous large reviews have, in contrast, failed to support this belief, with the exception of haemophiliac patients undergoing biopsy.^{1–4}

It would have been interesting to know in the audit what measures, if any, were taken to correct the coagulation abnormalities before biopsy; practice probably varied between different units. Therein may lie the reason for the different incidence of haemorrhagic complications between this and other audits.

There are few data on which to base decisions on when and how to correct coagulopathy before liver biopsy. Parenteral vitamin K should be given at least six hours beforehand, but if coagulopathy persists then fresh frozen plasma is the agent of choice for correction.⁵ The level of prolongation of the prothrombin time at which fresh frozen plasma should be given has not been defined but a prolongation of three seconds or more is generally accepted to require correction.⁶ If fresh frozen plasma is given then the dose should follow published guidelines—that is, 12–15 ml/kg body weight,⁷ given immediately before the biopsy.

1 Slomiany BL, Slomiany A. Mechanisms of *Helicobacter pylori* pathogenesis. Focus on mucus. *J Clin Gastroenterol* 1992; 14 (suppl 1): S114–21.

1 Guslandi M, Ballarin E. Assessment of mucus-bicarbonate barrier in the stomach of patients with chronic gastric disorders. *Clin Chim Acta* 1994; 114: 133–6.