Effect of Helicobacter pylori on the gastric mucous 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, 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 microorganism 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, 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 mucous 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 is due to the 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. Reference to a study7 to justify the use of viscosimetry 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 patients9 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.


Reply

EDITOR,—Guslandi takes issue with our finding that gastric mucous gel in *H pylori* patients has a higher viscosity than in the unaffected. We simply gathered and tested the clear mucous 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 mucous gel in patients with *H pylori* infection may be more viscous than those without. As the mucous cells seem to be under pressure to actively secrete, the gel may be younger than in other 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 better considered a waste product. The fact that the mucous gel coat might be thinner in *H pylori* infected patients tells 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 This means that hydrophobicity seems 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 mucous have had no difficulty interpreting our findings.4


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 one can utilise the principles of Bayes’ theorem2 to predict the post-test probability of 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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The level of prolongation of the prothrombin time at which a contemplated percutaneous biopsy should be abandoned in favour of a transjugular or percutaneous procedure has likewise been noted. It would be helpful if future audits or reviews aimed to clarify this point. A prospective study of the efficacy and duration of action of fresh frozen plasma is underway at the Queen Elizabeth Hospital.

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Increased CA 125 in tuberculous peritonitis

EDITOR—O’Riordan et al reported the case of a patient with tuberculous peritonitis, ascites, and pleural effusion who also had a remarkably increased concentration of serum CA 125 (Gut 1995; 36: 303–5). Because of the increase in this tumour marker an ovarian cancer was suspected and a laparotomy was performed with negative results for cancer. The authors consider the increased values of CA 125 the most interesting aspect of the case, and review other published reports on increased CA 125 values in patients with tuberculous ascites.

I believe, however, that the only interesting and noticeable aspect of the case would have been if the patient had had a normal value of CA 125. This tumour marker, commonly used in the diagnosis of ovarian cancer, increases in a variety of processes including pleura, pericardium, and peritoneum including endometriosis, peritonitis, pelvic inflammatory disease, and surgical trauma.1 2 3 Thus, the increase in CA 125 is not unexpected because this antigen has been detected on mesothelial cells in pleura, pericardium, and peritoneum, particularly in areas of inflammation.4 5

Benign processes, such as ascites, are associated with increased serum concentrations of CA 125 with values up to 100 times the upper normal limit in some cases.4 6 CA 125 has proved to be an excellent marker of malignancy in patients with benign liver diseases (sensitivity 98–4%, specificity 95–9%, efficiency 96–9%).6 This marker is also very sensitive to minimal amounts of ascites and correlates very well with the amount of ascitic fluid. Thus, the increase of CA 125 for ovarian cancer is very low in the presence of ascites of whatever origin.

In addition, the return of CA 125 to normal after anti-tuberculous treatment in the patient of O’Riordan et al is not unexpected. CA 125 decreases to normal values when the ascites is removed in cirrhotic patients and increases again when ascites recur.7

The authors conclude that tuberculous needs to be considered in the differential diagnosis of ascites with increased tumour markers’. It is well known, however, that CA 125 is an unspecific marker of ascites of whatever aetiology. Tuberculosis, a disease that only uncommonly produces peritoneal effusions, represents only one of the many aetiologies of ascites. Finally, I believe that a laparotomy should not have been performed in this case because both computed tomography and cytological study of ascitic fluid were negative for ovarian cancer and the very high concentration of CA 125 could have been easily explained by the existence of both ascites and pleural fluid.

Reply

EDITOR—Dr Collazos’ points concerning CA 125 are valid. He is, however, an expert in the area and has published widely on CA 125. We felt this case to be of interest to a general audience because they might not have been familiar with the wide range of increases seen in CA 125. The concentrations of CA 125 are high in many conditions as outlined in Dr Collazos’ letter. We mentioned all of these and more in our discussion. Dr Collazos does not mention how he would have made the diagnosis. He might have legitimately requested laparoscopy in place of laparotomy. He suggests neither. We believe he overstates the value of negative computed tomography and non-diagnostic ascitic fluid cytology in excluding intra-abdominal tumour, however. Such measures can only reduce the relative risk that a tumour is present.

One additional point of some interest is that despite our considerable experience with immunocytochemistry and CA 125 in cytological preparations, we were unable to identify positive staining in the mesothelial cell population in the ascitic fluid specimen in this report. Had the immunopanel been performed during the case this too would have urged us towards more invasive investigations.

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Adenomas and a family history of colorectal cancer

EDITOR—The data presented by Gagliola et al (Gut 1995; 36: 385–90) imply that adenomas in hereditary non-polyposis colorectal cancer (HNPPC) occur with increased frequency, show a predilection for the proximal bowel but especially with respect to the sigmoid to show villous change. It is interesting to note that these data are at complete variance with our own.1 We have suggested that the high risk of colorectal cancer in HNPPC is explained not by increased initiation of
Audit of percutaneous liver biopsy.

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