Correspondence

Interaction of *Campylobacter pylori* . . .

SIR,—We read with interest the paper by Bernatowska *et al* (*Gut* 1989; **30**: 906–11). Their observations that *Campylobacter pylori* will activate the classical pathway of complement directly and can be phagocytosed by neutrophils *in vitro* may well be of considerable importance. The role of complement *in vivo* at the gastric mucosal surface must, however, be questioned.

It has been known for many years that ammonia can inactivate complement. Host defence mechanisms are known to be compromised in the kidney because of the inactivation of complement.¹ These early studies of Beeson and Rowley suggested that ammonia production by renal glutaminase could inactivate the fourth component of complement. Ammonia production from urease positive bacteria colonising the urea rich environment of the kidney has also been proposed as a source of anti-complementary activity.² *C. pylori* is known to have potent urease activity³ and in the presence of urea it is highly likely that viable organisms will produce concentrated ammonium ions in close proximity to gastric epithelial cells.

The study of Bernatowska *et al* was carried out with heat killed *C. pylori* (60°C for one hour), a process which will virtually abolish the marked urease activity of the bacterium.³ *C. pylori* has been shown to generate considerable quantities of ammonia from urea *in vitro*⁴ and *in vivo* the concentration of ammonia in gastric juice correlates with the presence or absence of *C. pylori*.⁵ Local ammonia production by these bacteria at the gastric luminal surface might, therefore, comprise mucosal defences by inactivation of complement in a similar manner to that proposed in the kidney.

It would be of interest therefore to know whether the results of Bernatowska *et al* would have been different had the experiments been undertaken with live *C. pylori* in the presence of urea. The ammonia produced under such conditions might well be expected to modify the activation of complement by *C. pylori* and the subsequent neutrophil phagocytosis.

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References

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Reply

SIR,—Dr Crabtree and colleagues raise the interesting point that ammonia produced by live *Campylobacter pylori* might inactivate complement *in vivo*. Our studies on complement activation were performed with organisms scraped from blood agar plates and suspended in phosphate buffered saline, and then stored at –70°C until used. Only the organisms used for the ELISA antibody assays were heat killed. It is unclear, however, whether the organisms used in the complement studies were metabolically active and able to produce ammonia. This would need further investigation, and it might be difficult to design a system of *in vitro* complement activation with actively metabolising organisms. In order to assess the *in vivo* relevance of our findings, immunofluorescent studies to identify C5a in gastric biopsies from patients with gastritis and *C. pylori* infection may be rewarding.

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Acid perfusion in the assessment of non-cardiac chest pain

SIR,—We would like both to point out some anomalies in the paper on acid perfusion in the assessment of non-cardiac chest pain from Hewson *et al*¹ and to express doubts about their conclusion that the acid perfusion test (APT) may have been rendered obsolete by prolonged ambulatory intraoesophageal pH monitoring.

Firstly, they report that reproduction of chest pain (not heartburn) by APT occurred in 16/71 (22.5%) of their patients, a substantially different figure from that of 7% reported in a recent large series from the same laboratory.² The figure of 22.5% is similar to the results we³ and Janssens and colleagues⁴ have reported (35% and 27% respectively). It is difficult to understand why in Hewson *et al*'s study a symptom