Correspondence

Gastric secretion in patients with typhoid

SIR,—We read with interest the recent paper by Dr Bhalla and colleagues on gastric secretion in patients with typhoid (*Gut* 1985; **26**: 491–4). They make the interesting observation that patients with typhoid have reduced levels of gastric acid output during convalescence. Patients recovering from non-enteric infections had similar acid outputs in controls.

The authors speculate that typhoid infection suppresses acid output, and that the mechanism may involve a bacterial toxin. They also state that fever alone is unlikely to cause acid suppression.

Because gastric acid output could not have been measured in the typhoid patients before infection, it is not possible to state that the hypochlorhydria is a direct result of the infection. Hypochlorhydria could have antedated the exposure to typhoid and would therefore have increased the risk of these individuals to severe infection. Reduced acid secretion is a well recognised predisposing factor to enteric infections,^{1–5} a fact which the authors clearly state.

It is also not possible to be certain that fever alone did not contribute to the reduced acid output, as the gastric secretion studies were all carried out about one week after the temperature had returned to normal. It has previously been described that physically induced pyrexia can temporarily suppress acid output in the dog and man.^{6,7} Therefore, the patients with non-enteric infection could have had depressed gastric acidity while they were still febrile. Indeed, this has been described in patients with a variety of pulmonary infections and non-infective causes of pyrexia.^{8,9}

The hypochlorhydria which was seen in the patients with typhoid could have been present before the infection and been exacerbated by the fever. Seven patients had histological gastritis during the recovery phase from typhoid which could help to explain the persistently subnormal acid output in this group. This may have been a manifestation of typhoid, but might equally have been due to superadded infection with another organism which was able to gain access because of the hypochlorhydria. It is difficult, however, to speculate on the true significance of the gastritis as histological specimens were not obtained from the control subjects or those recovering from non-enteric infections.

This excellent study by Bhalla and colleagues does not solve the temporal association of infection and hypochlorhydria.

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Reply

SIR,—We thank Drs Howden and Hunt for their interest shown in our article. They have raised two important questions regarding diminished acid secretion seen in patients with typhoid fever. Firstly, is hypochlorhydria in typhoid patients a pre-existing defect or is it the result of salmonella infection? Because it was not possible to determine the gastric acid secretory status of subjects before the onset of infection, the only way of ascertaining this relationship was to reexamine these subjects after the infection had subsided. We measured acid output (BAO and MAO) during convalescence and two months later and found a significant recovery in acid output in all the typhoid patients. In patients with uncomplicated typhoid fever, acid levels recovered completely after two months. In the more severe patients, even though the acid levels were still low