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 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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References


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 re-examine 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...
after two months. In the more severe patients, even though the acid levels were still low after two months, they showed a significant increase over the convalescent levels. These observations clearly suggest that hypochlorhydria in typhoid patients was secondary to infection and not a primary defect. Secondly, regarding the role of fever in causing hypochlorhydria, a prolonged suppressive effect of high body temperature on acid secretion has never been demonstrated. Studies in experimental animals\(^1\) and in humans\(^2\) have shown a transient hypochlorhydria which recovers completely within a short time of 24 hours or so. As we measured the acid output one week after the fever had subsided in both typhoid as well as non-typhoid patients, it is very unlikely that fever could have contributed significantly to hypochlorhydria.

Superficial gastritis was present in only seven out of 27 patients which is the same as that found in the normal healthy Indian population. Besides, there was no correlation between the severity of typhoid fever and the presence of gastritis. Therefore, it seems highly unlikely that superficial gastritis could be an important factor in causing hypochlorhydria. The aetiology of gastritis, however, is unclear, whether it is because of typhoid infection or any other secondary bacterial invasion is purely speculative.

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References

3 Tyagi KP, Mukhopadhyay AK, Aggarwal HK et al. Gastric mucosal morphology in tropics and influence of spices, tea and smoking. Nutr Metab 1974; 17: 129–35.

Books


Current opinion in gastroenterology. Volume 1, Number 1: Large intestine and gastrointestinal infections Bimonthly review, (Pp. 120 per issue, not illustrated; £10.00, or £25.00 for six issues.) London: Gower Academic Journals, 1985.

In an era when institutional libraries have less to spend on journal subscriptions, it is getting harder to keep up with the ever increasing volume of published papers in our subspecialty. Lists of papers by title, as in Current contents or in the computer data-bases, are of limited value; they are uncritical, and only useful if there is access to the journals themselves. Two recent publications seek to overcome these problems by offering a critical evaluation of the published literature of the preceding year.

Two distinguished Americans, Frank Moody and Norton Greenberger reviewed 10 000 papers from 250 journals, and from these made a selection of about 200 papers from 50 journals. These are summarised in the Year book of digestive diseases, arranged under appropriate subject headings. The summaries are lengthy and often include diagrams and tables from the original paper; they contain the substance of the paper and only the detail is omitted. Each summary, moreover, is followed by a commentary from one of the editors; these commentaries are stimulating, provocative, and thoughtful as might be expected from these two men. The book does not cover basic science related to gastroenterology and is presumably aimed at those whose interests are strictly clinical. It is perhaps surprising that all the papers that were finally selected come from North American or British journals (with one exception: the European Journal of Physiology is represented even though its British and American counterparts are not.) Does this imply that there were no memorable papers in journals from Europe, Japan, etc?

Current opinion in gastroenterology is a bimonthly review journal. Each issue covers two main topics, and a number of reviews by different authors are included under each topic. Each review carries a selection of references, annotated by the author, and often indicated as being either of 'specialist interest' or, rarely, of 'outstanding interest'. The annotations vary from critical mini-summaries to single sentences. Each of the two main sections concludes with a more extensive list of references which includes those selected by each reviewer. The contributors to this issue – who, despite an international editorial board, are all from the UK – tend to lack the authority of their American counterparts, and some of the reviews are somewhat leaden. Editorial policy on annotation of references needs to be more consistent, and we should be told whether we can regard the main list of references as complete, and if not, the criteria for selection. On the credit side, basic as well as clinical science is