LETTERS TO THE EDITOR

Nervous dyspepsia

To the Editor: Marshall and colleagues are to be congratulated on their innovative ideas on the causation of acute and chronic gastritis and peptic ulceration.[1] The quotation and interpretation by these authors[2] of Osler's observations suggest that Osler knew in 1920 how to make a primary diagnosis of nervous dyspepsia, although he named it chronic gastritis.

In a preliminary report in 1979 on nervous dyspepsia and bile reflux,[3] I made the point that diagnosis can as a rule be made positively only after careful history-taking and physical examination. I still adhere to that view. Endoscopy or barium radiography are generally only indicated for patients who fail to respond to proven therapy over one month. In this regard I disagree with Marshall et al,[1] who appears to regard nervous dyspepsia as "at present a 'non-diagnosis' for patients without any proven cause of their symptoms".

In view of the common finding of pyloric campylobacter in antral biopsies from patients with non-ulcer dyspepsia,[1] it would seem possible, if not probable, that the duodeno-gastric reflux of bile reported by me in such patients[3] causes damage to the antral mucosa, which enables campylobacter organisms to invade opportunistically. The reported absence of these organisms in normal duodenal mucosa would make it impossible to suggest that the campylobacter infection induces the bile reflux.

Talley was certainly not the first to suggest that the name "nervous dyspepsia" might be appropriate for many patients, as patients with non-ulcer dyspepsia are freqently not overtly nervous; this was, in fact, the first of my nine observations on a series of 136 patients with nervous dyspepsia in 1979.[3]

Until the exact aetiology or aetiologies for nervous dyspepsia are worked out, there are no grounds to rename the syndrome. In addition, Marshall et al. appear to be providing further grounds for not regarding nervous dyspepsia as a diagnosis of exclusion, but in the present state of the art I doubt that it will be routinely necessary to search for campylobacter organisms in antral mucosa before making this diagnosis.

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1. Marshall BJ, McGechie DB, Rogers PA, Glancy RJ. Pyloric campylobacter infection and gastroduodenal disease. *Med J Aust* 1985; 142: 439-444.
2. Marshall BJ, Armstrong JA, McGechie DB, Glancy RJ. Attempt to fulfil Koch's postulates for pyloric campylobacter. *Med J Aust* 1985; 142: 436-439.
3. Graham JR. Nervous dyspepsia and biliary reflux. *Med J Aust* 1979; 2: 370-371.
4. Talley NJ. "Nervous dyspepsia" — a misnomer. *Aust NZ J Med* 1984; 14 (Suppl 14): 915.

In reply. — I am aware of Dr Graham's work, and prefer to discuss first the references upon which he bases his conclusions about bile reflux and gastritis.[1-4] At the time these papers were written, there was no good explanation for gastritis, so reflux of bile through the pylorus was invoked as a possible cause.

Goldner and Boyce quantitated the amount of bile actually present in the stomachs of their patients, and found that it was unrelated to histological gastritis.[5] It is proven, therefore, that not all patients with bile in the stomach have gastritis. On the other hand, allowing for very rare exceptions, all patients with *Campylobacter pyloridis* (CP) infection have gastritis.[6] Even in patients with Billroth II partial gastrectomies, who must all have some bile reflux, histological active chronic gastritis is only present in those who also have CP.[7] I conclude that it is no longer necessary to invoke bile reflux as a cause of gastritis, and would suggest that chronic inflammation of the pyloric valve is the primary cause of bile reflux.

I give ground on Dr Graham's point that "nervous dyspepsia" is not always a diagnosis of exclusion, as I implied when using the name "non-ulcer dyspepsia" in my recent paper.[7] This very common gastrointestinal disease may often be diagnosed on the history, as he suggests. The typical patient (usually a female) arrives carrying a negative barium meal (perhaps showing a little reflux, hiatus hernias, etc) and/or a negative oral cholecystogram. She complains of substernal and epigastric burning pain, reflux, burping, epigastric distension, a bloated feeling after meals, periodic nausea (especially in the morning), gnawing sensations, oppressive feelings in the lower chest region, shortness of breath, flatulence, and sometimes altered bowel habit. A common statement is that tight clothing around the abdomen cannot be tolerated. A careful history reveals that the symptoms have been present to a greater or lesser degree for some years, and that other family members (often the spouse, sometimes the children) suffer from duodenal ulceration or vague dyspeptic syndromes. The symptoms are worse during times of stress, but even during "remissions" the patient finds that she cannot eat certain foods, especially onions. Many patients cannot tolerate non-steroidal anti-inflammatory drugs. Epigastric, hepatic, or even diffuse abdominal tenderness may be present, but there are often no physical signs.

Although the "nervous dyspepsia syndrome" is not always associated with gastritis, I believe that gastroscopy is always necessary — not to exclude peptic ulceration, but to obtain an antral biopsy, just as a urine specimen is obtained when a patient has dysuria. The most severe cases of "nervous dyspepsia" have CP infection with gastritis, which responds to antibacterial therapy. Like Osler, I believe that bismuth is effective treatment: De-Nol (one tablet four times a day for one month) can be commenced if CP are seen on Gram staining of the antral biopsy. I review patients 14 days later, and give the appropriate antibiotic concurrently with the De-Nol for another two weeks. I do not tell patients they should change their lifestyle unless their habits are grossly excessive, I hardly ever use benzodiazepines, and I do not investigate CP-positive patients further unless they fail to respond clinically.

I hope that the medical profession will not become as polarized on this issue as Dr Graham and I obviously are, but will go out and look for themselves. It seems to me that all things are possible for a bacterium which infects 20% of the adult population and is continually being shed into the upper end of the gastrointestinal tract.

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1. Lawson HH. Effect of duodenal contents on the gastric mucosa under experimental conditions. *Lancet* 1964; 1: 469-472.
2. DuPlessis DJ. Pathogenesis of gastric ulceration. *Lancet* 1965; 1: 974-978.
3. Drew JH. Biliary reflux: postoperative and idiopathic. *Med J Aust* 1979; 2: 125-128.
4. Graham JR. Nervous dyspepsia and biliary reflux. *Med J Aust* 1979; 2: 370-371.
5. Goldner FH, Boyce HW. Relationship of bile in the stomach to gastritis. *Gastrointest Endosc* 1976; 22: 197-199.
6. Thomas JM. Campylobacter-like organisms in gastritis. *Lancet* 1984; 2: 1217.
7. Marshall BJ, McGechie DB, Rogers PA, Glancy RJ. Pyloric campylobacter infection and gastroduodenal disease. *Med J Aust* 1985; 142: 439-444.

Informed consent

To the Editor: In correspondence a year ago, Dr P. Gerber recommended that a patient undergoing a diagnostic procedure requiring the intravenous injection of contrast medium "is entitled to be fully informed and to decide for himself whether or not to take the risk of death or complications, however statistically insignificant".[1] He further states that "the duty to inform rests in the first instance with the referring clinician", or "alternatively (and preferably), radiologists may consider issuing a standard consent form . . . ".

Dr Gerber's opinion appears to conflict with his own expressed conclusions on informed consent.[2] The third of five "rules" advanced by Dr Gerber begins: "the extent of the duty to disclose must depend greatly upon the patient's expressed or apparent desire for information". Furthermore, Dr Gerber's opinion is not shared by Mr Allan Hunter, Solicitor of Adelaide, who states:

> 1. I do not believe that it is necessary for a radiologist to warn a patient of the statistically infinite risk of dying from a diagnostic procedure.
> 2. The duty to inform rests with the