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NEWS AND NOTES

SUPPLEMENT

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CORRESPONDENCE

Correspondents are urged to write briefly so that readers may be offered as wide a selection of letters as possible. So many are being received that the omission of some is inevitable. Letters should be signed personally by all their authors.

The bran-wagon

SIR,—Dr Robert C Hall (1 May, p 1076) criticises the paper of Mr A J M Brodribb and Dr Daphne M Humphreys (21 February, p 425) because it was an uncontrolled trial. He states that they "provide no proof that bran had anything to do with relief of 60% of symptoms in their patients. All parameters assessed were subjective and there were no control patients."

When I published an "uncontrolled" trial of bran in diverticular disease[1] I had been advised by a medical statistician of repute that, as I believed a low-residue diet was the cause of the condition,[2] it would have been both immoral and unethical for me to have given such a diet to my patients. I submit that Mr Brodribb and Dr Humphreys were in the same position. If they believe that fibre deficiency causes diverticulosis then they would have been wrong to prescribe a low-fibre diet. It is up to those who do not believe in the fibre hypothesis to carry out a double-blind trial.

Subjectively, bran relieves over 80% of the symptoms of uncomplicated diverticular disease[1] and patients notice that these symptoms return if they revert to a low-residue diet, even for a holiday of two weeks, but disappear once they return to a high-fibre diet. As it is their symptoms that cause patients distress, why discount the importance of subjective improvement?

Objectively, not only do these patients act to some extent as their own controls, but several investigators have measured the effects of bran in the disease. Bran relaxes the colonic musculature and reduces the intracolonic pressures and hence differs from simple bulk formers.[3,4] Bran alters the consistency and weight of the stools, lessens the need to strain during defecation, and alters both the transit time and the electrical activity of the colon towards the normal.[1,5,6] Following sigmoid myotomy or resection for diverticular disease the intracolonic pressures fall but rise again in three years if patients continue to eat the same diet.[7] In contrast, these lowered pressures show no sign of rising again for at least five years if patients change to a fibre-rich diet postoperatively.[8] This last observation suggests that while the surgeon can attack the complications of the disease, the replacement of fibre in the diet counters its underlying cause. Furthermore, one month's treatment with bran lowers the intracolonic pressures in diverticular disease more efficiently than does a simple bulk-former when compared with pressures recorded from untreated patients.[3]

All these objective observations are consistent with the contention that diverticular disease is caused, at least in part, by a deficiency of dietary fibre. It is difficult to prove anything in medicine, but the "British bran-waggon" submit that it is up to those who disagree with their views to put forward an alternative hypothesis that fits the observed facts.

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[2] Painter, N S, and Burkitt, D P, *British Medical Journal*, 1971, **2**, 450.
[3] Srivastava, G S, Smith, A N, and Painter, N S, *British Medical Journal*, 1976, **1**, 315.
[4] Findlay, J M, *et al*, *Lancet*, 1974, **1**, 146.
[5] Burkitt, D P, Walker, A R P, and Painter, N S, *Lancet*, 1972, **2**, 1408.
[6] Taylor, I, and Duthie, H L, *British Medical Journal*, 1976, **1**, 968.
[7] Smith, A N, Giannakos, V, and Clarke, S, *Journal of the Royal College of Surgeons of Edinburgh*, 1971, **16**, 276.
[8] Smith, A N, in *Fibre Deficiency and Colonic Disorders*, ed R W Reilly and J B Kirsner. New York, Plenum, 1975.

Pancreatic pain

SIR,—Your statement that "chronic upper abdominal pain cannot certainly be attributed to pancreatic disease short of a laparotomy" (leading article, 17 April, p 921) does less than justice to modern methods of pancreatic diagnosis. Indeed laparotomy itself is not always diagnostic.[1] We regularly receive patients for further investigation following inconclusive exploration or many months later when the patient's progress seems at variance with the confident operative diagnosis of cancer. There is often a reluctance to perform operative pancreatic biopsy.[2]

There are several new approaches to pancreatic diagnosis. Non-invasive imaging of the pancreas is now possible using grey-scale ultrasonography,[3] and computer tomography (EMI-scanning). Preliminary results are most encouraging. Fibreoptic duodenoscopy permits cannulation of the papilla of Vater under direct vision in conscious patients and can provide high-quality cholangiograms and pancreatograms.[4] An abnormal pancreatogram is certainly diagnostic of pancreatic disease, although the distinction between chronic pancreatitis and cancer is sometimes difficult from the radiographs alone. However, cancer can often be confirmed by cytological examination of the pancreatic secretions collected from within the duct during the cannulation procedure.[5]

Histological information may also be obtained without laparotomy. Duodenoscopic biopsy is effective when pancreatic cancer involves the duodenal wall or papilla. Attempts are now being made to take deeper transendoscopic drill biopsies through the duodenal wall. Laparoscopy has been used to provide visual and biopsy access to the body of the pancreas.[6] Most remarkable of all is percutaneous pancreatic biopsy, a routine practice in several centres. A needle is passed directly through the abdominal wall into the pancreas to a lesion located by ultrasonography[7] or concomitant radiology (pancreatography, arteriography,[8] or transhepatic cholangio-