

the appreciable differences seen in the results.

The studies of Lauritsen *et al*, with faecal dialysates reflect only the intraluminal concentrations but provide no information about the intramucosal concentrations. The information that results from both types of studies, however, can be complementary.

Finally, we totally agree that the very low mucosal concentrations of 5-ASA after Salazopyrin supports the hypothesis that Salazopyrin has pharmacological properties that are different from those of 5-ASA and contribute to its therapeutic efficacy.

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Riedel's thyroiditis, retroperitoneal fibrosis, and sclerosing cholangitis: diseases with one pathogenesis?

EDITOR.—We have read the case report on multifocal fibrosis by Laitt *et al* (*Gut* 1992; 33: 1430–2) and agree that the combination of Riedel's thyroiditis with retroperitoneal fibrosis as well as sclerosing cholangitis is very rare. We have recently collected from worldwide studies 14 reports on the 14 patients with both Riedel's thyroiditis and retroperitoneal fibrosis and suggested a common pathogenetic mechanism.¹

In their article Laitt *et al* state that 'Bartholomew noted an association of Riedel's thyroiditis, sclerosing cholangitis and retroperitoneal fibrosis in the same patient.' This is not correct. In the article by Bartholomew *et al*² not one but two patients were described; one with sclerosing cholangitis and retroperitoneal fibrosis and the other with Riedel's thyroiditis and sclerosing cholangitis. This last patient, by the way, is the same patient as described by Woolner *et al*³ (reference 7 in the article by Laitt).

The patient described by Laitt *et al* is therefore not the third, but the second reported in published works with this triad of organ involvement. The only other patient was described twice, not only by Gleeson *et al*⁴ (reference 2 in the article by Laitt) but once again with a longer follow up in an article by Katsikas from the same hospital in 1976.⁵

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Reply

EDITOR.—We are grateful to Dr de Boer for setting the record straight on the number of

previous patients reported on with Riedel's thyroiditis, retroperitoneal fibrosis, and sclerosing cholangitis. We are not unhappy to learn that our patient is the second rather than the third. We are well used to coming second!

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Microvascular abnormalities in the mucosal prolapse syndrome

EDITOR.—The paper by Dr Lonsdale (*Gut* 1993; 34: 106–9) while presenting an intriguing and new theory of ulceration in mucosal prolapse syndrome, and highlighting a previously neglected aspect of its histopathology omits some diagnostic features. Diamond shaped crypts and intramucosal elastin¹ are features of all the conditions that fall within the bounds of the unifying concept of mucosal prolapse.^{2,3}

Also the relation between metaplastic polyps and mucosal prolapse is unclear. While we would agree that most metaplastic polyps are too small to induce mucosal prolapse changes in the immediately adjacent mucosa, histological features of mucosal prolapse are usually seen within the polyps themselves.⁴ Metaplastic change is seen overlying 30% of cases of mucosal prolapse in most series.^{3,5} The theories of Cripps⁶ and ourselves⁴ relating metaplastic polyps to mucosal prolapse, because of their similar features, may have to be reconsidered now that Dr Lonsdale's paper has uncovered a difference between the vasculature of metaplastic polyps and mucosal prolapse.

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Reply

EDITOR.—I am grateful to Dr Warren and Dr Davies for their comments on my paper. As this was mainly an histopathological examination of vascular changes in prolapsing mucosa, detailed description of all the microscopic features of this condition was not attempted. Diamond shaped crypts and intramucosal elastin, however, were present in some of the specimens. I would agree that metaplastic polyps and mucosal prolapse share some common histological features, and on occasion, especially in a small or distorted biopsy specimen, can be a problematical differential diag-

nosis. The lack of observable vascular changes and the rarity of ulceration in metaplastic polyps may be related to differing predisposing factors or initiating stimuli, and is not inconsistent with them being a manifestation of local mucosal prolapse.

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Inflammatory bowel disease in Asians

EDITOR.—The studies by Probert *et al*^{1–3} have provided important information on the incidence of inflammatory bowel disease in south Asians. Furthermore, the results have highlighted the fact that this is not a homogenous group of patients, with significant differences between Hindus, Moslems, and Sikhs. The heterogeneity is perhaps not surprising because of the vast size of the Indian subcontinent, from which these patients originate.

Diet is known to play a part in the cause and subsequent course of inflammatory bowel disease. In addition to the obvious differences between European and Indian diets, there are also important differences in the diet within India. Epidemiological studies have shown that a low fibre diet is a risk factor for inflammatory bowel disease.⁴ Areas in the south of India have a lower intake of unrefined fibre when compared with the northern regions.⁵

Other dietary factors, such as antioxidants, may also be of importance because of the recent evidence implicating oxygen derived free radicals in inflammatory bowel disease.⁶ Healthy subjects in Madras, in south India, have been shown to have lower plasma values of ascorbic acid and β carotene than healthy subjects in England.⁷

Thus, some of the differences seen in the incidence of inflammatory bowel disease in people from the Indian subcontinent may be related to dietary differences between Europeans and south Asians, as well as dietary differences between the various groups from within the Indian subcontinent. It would be interesting to know if the findings of Probert *et al* were mirrored in Hindus, Moslems, and Sikhs within India.

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