target biopsy specimens can be obtained from an abnormal area.

It is true that a small proportion of patients develop carcinoma in colitis without dysplasia elsewhere in the colon and that dysplasia, when present, is usually patchy. The paper by Rasonhoff et al., however, is quoted as evidence that dysplasia at a distance from the cancerous lesion occurs in only 50% of colicky specimen. This paper was based on histological blocks from only 22 specimens with carcinoma in colitis, complete resections were performed in only 11 of the patients, and more than five blocks were available from only 13 of the specimens. Some dysplasia at a distance from the tumour was found in 73% of specimens and high grade dysplasia in 50%. The authors suggest that one reason why the incidence of dysplasia was less than in other reports may have been due to limited sampling. Careful examination of 62 colotomied specimens with cancer in colitis at St Mark's Hospital has shown dysplasia at a distance from the tumour in 87%. Dr Gyde selected three clinical studies for comment and we appreciate her remarks about the importance of high levels of follow up and of clinical care in regard to our surveillance programme. It is a pity that she made no reference to two excellent Swedish series, ‘one randomised controlled trial’ in which there has been cancer death and in which the three tumours treated surgically were at an early stage (Dukes' A).

Like Dr Gyde, we would welcome the development of a new marker of neoplastic potential in colitis. Apart possibly from analysis of aneuploidy, which is complex and expensive, no such marker has been identified. Despite its limitations, dysplasia is the only thoroughly tested marker we have and it should not be rejected.

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Reply

Stn.—Professor Blum et al report the superiority of sucralfate over placebo in the prevention of gastric ulcer recurrence. They speculate that continued treatment with sucralfate may have theoretical advantages over long-term maintenance therapy with antasycretory drugs, but fail to discuss the potential risk of aluminium accumulation. Administration of sucralfate (1 g twice a day) will result in a daily intake of 414 mg aluminium. The aluminium moiety of sucralfate can dissociate at a low pH, and short-term administration of 4 g daily may lead to appreciable increases in serum and urine aluminium concentrations. Long-term administration of sucralfate (1 g twice a day) did not result in statistically significant increases in plasma aluminium in either of two small maintenance studies but animal experiments indicate that bony accumulation of aluminium may occur in the absence of raised serum concentrations. Sucrel fate may prove to be effective and safe for the prevention of gastric ulcer recurrence. However, further studies of aluminium accumulation from long-term use are likely to be needed before this drug can be widely recommended as an alternative to antisecretory agents.

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BOOK REVIEWS


This monograph written by Starzl and Demetris is an up to date summary of liver