published literature on the gastrointestinal effects of acupuncture, we were unable to find any controlled study showing the effect of acupuncture on ulcer healing. There have been three reports, however, of uncontrolled studies suggesting that acupuncture may be of therapeutic benefit in peptic ulcer disease and Lux et al cited one of these.

It is unfortunate that Lux et al failed to recognise our own work, but more so that they did not extend our initial studies into the mechanisms participating in the inhibition of acid secretion by acupuncture.

Clearly, we agree with them that further studies are needed to examine therapeutic efficacy.

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Cancer surveillance in ulcerative colitis

EDITOR.—In re-reading the editorial I wrote (Gut 1994; 35: 587-9) I have identified an error that I wish to correct.

Paragraphs six and seven state that the analysis of 11 prospective colonoscopic surveillance studies compared patients with and without low grade dysplasia. This is incorrect, the two groups compared were all patients submitted for surveillance on the one hand and those found initially to have low grade dysplasia on the other.

Sentence two in paragraph six should have read ‘In all, 73 cancers were found in 1656 patients (4-4%) whereas 26 cancers were found in the subgroup of 313 patients with low grade dysplasia (8 3%). If dysplasia associated lesions or masses are excluded this falls to 0-2%’. A similar mistake occurs in paragraph seven. The second sentence of which should read ‘In all, cancer was present in 93 of 2044 patients (4-5%) whereas 35 cancers were found in 101 patients with high grade dysplasia (35%)’.

I apologise for the inaccuracies detailed above.

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Colorectal tumorigenesis

EDITOR.—We noted with interest the paper by Mulder et al (Gut 1995; 36: 76-80) on expression of mutant p53 protein and CD44 variant proteins in colorectal tumorigenesis. The authors in their report have shown that CD44 v6 expression is restricted to moderately and severely dysplastic adenomatous polyps and colorectal cancers, but that it is not expressed in normal colon and mildly dysplastic adenomas. They also suggested that CD44 v6 expression is associated with tumour progression. We have studied CD44 v6 in frozen and paraffin wax embedded tissue sections from 11 normal colons, eight adenomatous polyps, and in 18 colorectal adenocarcinomas, with immunohistochemistry using anti-CD44 v6 antibody. In contrast with Mulder et al we found expression of this variant in normal colon crypt epithelium, and similar expression was also seen by Fox et al. We also detected CD44 v6 protein in all eight adenomatous polyps irrespective of the grade of dysplasia, and in 15 of 18 colorectal adenocarcinomas. The positive colorectal cancers CD44 v6 expression was strong and homogeneous in three, and heterogeneous and weak in 12. Survival at five years was: 0 of 3 in patients with homogenous, 9 of 12 with heterogeneous and weak expression, and 3 of 3 in negative cases. In colorectal adenocarcinomas, Mulder et al saw a correlation with Duke’s stage and tumour progression. Our study shows no apparent correlation of CD44 v6 expression with tumour progression, there being no linear trend with Duke’s staging or differentiation.

The decreased survival of patients with colorectal cancer who express CD44 v6 strongly and homogeneously, however, suggests that this expression may be an independent adverse prognostic marker rather than a determinant of tumour progression.

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