Assessment of proliferation of squamous, Barrett's, and gastric mucosa in patients with columnar lined Barrett's oesophagus

EDITOR,—I comment on the interesting paper by Itilkar et al on assessment of proliferation in oesophageal squamous, Barrett's, and gastric epithelium by flow cytometric evaluation of Ki67 immunolabelling (Gut 1992; 33: 733–7). The authors found that biopsy specimens from squamous lined oesophagus contained cell populations with a higher percentage of Ki67 positive cells than Barrett's, and gastric mucosal biopsy specimens. Barrett's and gastric mucosal biopsy specimens had similar percentages of Ki67 positive cells. Their results may be misleading as the proportion of stromal cells in Barrett's mucosa greatly exceeds that in gastric and squamous mucosal biopsy specimens, which have over 90% pure epithelial populations; stromal cells are not excluded from the total cell count by their technique and dilute the epithelial cell population. Thus the finding that Barrett's mucosal biopsy specimens contain a similar percentage of Ki67 cells to gastric biopsy specimens (by this technique) is more likely to imply a considerably higher epithelial Ki67 labelling index in Barrett's than in gastric epithelial cell (as opposed to the total mucosal cell population).

This agrees with the studies performed on stained sections of mucosal biopsy specimens including our own.1 In our study of epithelial proliferation in Barrett's oesophagus we used PCNA immunostaining of specialised Barrett's, junctional, and metaplasia to evaluate proliferation in the epithelial cells only (excluding stromal cells). In our study specialised type Barrett's had a higher proportion of cells in cycle and an expansion of the proliferative compartment out of the crypt and into the luminal and gland cell compartments; implying a higher level of proliferative activity in the specialised Barrett's than in the other types of metaplasia. Our findings are consistent with the proved association of specialised type Barrett's epithelium with malignant change (in smokers).2,4

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Overview of screening and management of familial adenomatous polyposis

EDITOR,—In their review article Rhodes and Ibrahim (Gut 1992; 33: 575–9) discuss the necessity, and potential benefits, of long term screening of family members and siblings of probands diagnosed as having familial adenomatous polyposis. These screening programmes have identified B vitamin deficiency as a risk factor, usually by identification of rectal polyps at sigmoidoscopy,1,2 and reduced the occurrence of invasive colorectal carcinoma to less.


