results originate in two independent centres. Leary et al suggest that as HPV DNA is not always present in glandular neoplasia that HPV might be a cofactor rather than an initiating factor in glandular neoplasia. If this is so then HPV DNA need not necessarily be detected, possibly explaining the discrepant results from the United Kingdom and elsewhere.

To summarise, results from the United Kingdom1 suggest that infection with HPV types 6, 11, 16, 18 and 31 does not necessarily have a major role in cervical glandular neoplasia.

**Radiation colitis is another mimic of chronic inflammatory bowel disease**

We read with great interest the article written by Shepherd.1 This informative review will be of great use to practising histopathologists when they face an avalanche of colorectal biopsy specimens with relatively little clinical information. The article should persuade both pathologists and physicians that clinical information is of great importance in reaching a histological diagnosis. The colorectal mucosa has limited ways of expressing itself in response to injury—a single brick from the Berlin Wall may look identical to one from the longstanding Wall of China.

The authors describe the histological features of chronic inflammatory bowel disease, but this diagnosis must be based on a combination of several morphological features such as crypt distortion, metaplasia (Paneth cells or pseudopyloric), fibrosis of the lamina propria associated with loss of crypts and/or significant increase in chronic inflammatory cells. On this basis we believe that radiation colitis is a different entity to the diagnostic possibilities. Radiotherapy is a common form of treatment for many pelvic carcinomas and the clinical features of radiation enteropathy may appear for many years after the surgery may be unalike that the patient has been irradiated. Radiation colitis in the chronic phase demonstrates a very significant crypt distortion, vascular telangiectasia, and fibrosis of the lamina propria, which can easily be misinterpreted as healed or quiescent chronic inflammatory bowel disease, unless the relevant information is available.2

**Oestrogen receptors in conjunctival malignant melanoma**

Paridaens et al claim to have demonstrated oestrogen receptors in paraaffin wax sections of formalin fixed conjunctival malignant melanomas.3 It is not unreasonable to expect that these lesions may be susceptible to endocrine factors, but the authors’ results do not support their conclusions.

I have two reservations. First, the cytoplasmic staining they observed conflicts with the known lack of oestrogen receptors.4 Secondly, although the antibody to ER-D5 recognises an epitope on an oestrogen receptor related protein, several studies have shown that immunostaining with this reagent correlates poorly with the results of ligand binding assays for oestrogen receptors.5,6 Furthermore, the authors are mistaken to believe that ER-D5 is “... present only in oestrogen receptor positive tissues.”7

Finally, the statement that “... a nuclear binding assay, which identifies non-functional receptors, may be more appropriate” makes no sense. Surely it is more appropriate to identify functional receptors by, for example, seeking oestrogen regulated proteins, such as progesterone receptor and cathepsin D.

**Secretarial services to consultant microbiologists**

A questionnaire on the use of secretarial services sent to 21 consultant microbiologists in Yorkshire in July 1991 produced a