routine oesophagoscopy, gastro-duodenoscopy, and colonoscopy to search for follicles in the upper and lower gastrointestinal tract. We have recently seen a hair within the wall of an intrahepatic abscess and this raises the advisability of biliary tree investigations for further hairs. If the result is positive it may be best, if the patient has any remaining money, to advise reference to a trichologist. As histopathologists and surgeons, we are loathe to actually advise a selective hair depiling agent though we have already bought shares in cocoa butter. The future for histology looks bright.

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References

1 Eggston AA, Wolff D. *Histopathology of the ear, nose and throat*. Baltimore: Williams & Williams, 1947.
2 Michaels L. *Ear, nose, and throat histopathology*. London: Springer-Verlag, 1987.

Other correspondence

Choice of control patients in studies of local cervical immunology

We welcome the paper by Hughes *et al*[1] because it adds further weight to the evidence for the role of a local immunodeficiency state in the aetiology of cervical neoplasia, previously described by our unit.[2] The method of selection of control patients used in their study, however, requires comment.

It was stated that one group of controls had been drawn from women referred because of "clinical concern about the appearance of the cervix" or postcoital bleeding. We are not told whether any of these women had a current cervical infection, although this seems likely from our experience. The effects of infections on the distribution of Langherhans' cells in the cervical epithelium are poorly understood, but there is evidence that *Chlamydia trachomatis* may be immunosuppressive[3] and that herpes simplex virus infection can increase the Langherhans' cell count in the skin.[4] Until these effects are further investigated it would seem prudent to screen for cervical infections, especially in such high risk groups.

A second source of controls was taken from women undergoing hysterectomy who had had normal cytological smears. In view of the known false negative rate of cytology alone,[5] it would have been appropriate if these women had undergone colposcopic examination to check further on the normality of their cervix.

Furthermore, the biopsy specimens of colposcopically visible abnormalities in the study group were exposed to acetic acid before collection; the hysterectomy specimens were not. We investigated the effects of acetic acid on three normal cervices from fresh hysterectomy specimens of three premenopausal women. After hemisection one half of each cervix was soaked in 5% acetic acid for one minute before freezing. Each half was then similarly processed for immunohistochemical staining using DAKO-T6, as previously described.[2] There was no detectable effect of the acetic acid on the immunocytochemical staining. Measurements of the width of the cervical epithelium were made at five points on each biopsy specimen using a microscope attached to a computerised digitising tablet. These were performed independently by two separate observers, using only measurements through the same numbers of cell layers for comparison.

The mean width of the cervical epithelium was greater in all the acetic acid treated specimens, the difference ranging from 10 to 45%. Such a difference could lead to an artefactual increase in the measured area of an epithelium with an apparent decrease in the calculated cell count. The cause of this apparent tissue swelling is unknown, but may be related to the osmotic changes which have been reported to be the cause of acetowhiteness in abnormal epithelium.[6] Only controls from colposcopically normal cervices after acetic acid application should be used to minimise such potential sources of error.

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References

1 Hughes RG, Norval M, Howie SEM. Expression of major histocompatibility class II antigens by Langerhans' cells in cervical intraepithelial neoplasia. *J Clin Pathol* 1988;**41**:253–9.
2 Tay SK, Jenkins D, Maddox P, Campion M, Singer A. Subpopulations of Langerhans cells in cervical neoplasia. *Br J Obstet Gynaecol* 1987;**94**:10–15.
3 Levitt D, Barol J. The immunobiology of chlamydia. *Immunology Today* 1987;**8**:246–51.
4 Braathen LR, Berle E, Mobech-Hansen H, Thorsby E. Studies on human epidermal Langerhans' cells; activation of human T lymphocytes to herpes simplex virus. *Acta Dermatovener* 1980;**60**:381–8.
5 Morell ND, Taylor JR, Snyder RN, Ziel HK, Saltz A, Willie S. False-negative cytology rates in patients in whom cervical cancer subsequently developed. *Obstet Gynecol* 1982;**60**:41–5.
6 Reid R, Scalzi P. An improved colposcopic index for differentiating benign papillomaviral infections from high grade cervical intraepithelial neoplasia. *Am Obstet Gynecol* 1985;**153**:611–8.

Drs Hughes, Norval, and Howie comment:

None of our first group of controls had clinical evidence of cervical infection, and no cytological or histological evidence of any specific infection. They were not screened for asymptomatic carriage of cervical pathogens, but to our knowledge there is no published work to suggest that infection with agents other than the human papilloma virus (HPV) affects Langerhans' cell counts in the cervical transformation zone. According to the paper of Levitt and Barol cited above, chlamydial infection may lead to cyclical systemic immunosuppression and stimulation but no evidence is presented to suggest that Chlamydia cause local immunosuppression. While Chlamydia have been shown to infect epithelial cells, fibroblasts, monocytes and granulocytes, we are not aware of any data on their interaction with Langerhans' cells or their effect on Langerhans' cell numbers in local epidermal sites. Braathen *et al* showed that HLA-DR positive epidermal cells, presumed to be Langerhans' cells, were able to present herpes simplex virus antigen to T cells but (contrary to the claim made above), did not even investigate whether herpes simplex virus infection had any effect on Langerhans' cell counts in skin.[2] Indeed, the association of herpes simplex virus with Langerhans' cells has not been established.

We are aware of the false negative rate of cervical cytology alone and it would, perhaps, have been appropriate for our second group of controls to undergo colposcopic examination. The published work from Dr Barton's own unit can be criticised on exactly the same grounds, as their six control patients did not undergo colposcopic examination at the time of cervical biopsy