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 depilating agent though we have already bought shares in cocoa butter. The future for histology looks bright.

PS HASLETON

JM EDWARDS

WY NASSER

Departments of Pathology and Surgery,
Wythenshawe Hospital,
Manchester.

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 Langerhans' cells in the cervical epithelium are poorly understood, but there is evidence that *Chlamydia trachomatis* may be immunosuppressive³ and that herpes simplex virus infection can increase the Langerhans' 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 cervixes 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 aceto-whiteness in abnormal epithelium.⁶ Only controls from colposcopically normal cervixes after acetic acid application should be used to minimise such potential sources of error.

SE BARTON

P MADDOX

M JARMULOWICZ

D JENKINS

Department of Histopathology,
Whittington Hospital,
London N19 5NF.

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;8:249-51.
- 4 Braathen LR, Berle E, Moberg-Hansen 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 HR, 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 colposcopy index for differentiating benign papillomaviral infections from high grade cervical intraepithelial neoplasia. *Am J 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.

and did not have acetic acid applied to the cervix.³ We do not know of any published work regarding the effect of acetic acid application on the measured width of cervical epithelium, and find the preliminary results of Barton *et al* of interest, although applying acetic acid to the cervix after hysterectomy and hemisection may not have the same effect as applying it during colposcopy. In fact, the width of cervical epithelium in biopsy specimens from our two groups of controls, one exposed to acetic acid and the other not, did not differ significantly.

We were unable to detect any significant differences in Langerhans' cell counts or in major histocompatibility class II expression when our two groups of controls were compared. The controls differed significantly from the patients with koilocytosis or cervical intraepithelial neoplasia (CIN) when assayed for several independent criteria.⁴ We feel, therefore, that our controls, most of whom did undergo colposcopy, are, in fact, more appropriate than those selected by Tay *et al*,³ and that our work supports the hypothesis that both HPV infection and CIN are associated with a localised disturbance of immune function.

R HUGHES*

M NORVAL†

S HOWIE†

*The Simpson Memorial Maternity Pavilion,
Lauriston Place,
Edinburgh EH3 9YW

†Department of Bacteriology,
University of Edinburgh Medical School,
Teviot Place,
Edinburgh EH8 9AG.

References

- 1 Levitt D, Barol J. The immunobiology of Chlamydia. *Immunology Today* 1987;8: 246-51.
- 2 Braathen LR, Berle E, Mobeck-Hanssen U, Thorsby E. Studies on human epidermal Langerhans cells. II Activation of human T-lymphocytes to herpes simplex virus. *Acta Dermatol* 1980;60:381-8.
- 3 Tay SK, Jenkins D, Maddox P, Champion M, Singer A. Subpopulations of Langerhans cells in cervical neoplasia. *Br J Obstet Gynaecol* 1987;94:10-15.
- 4 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.

Book reviews

Sjögren's Syndrome. Clinical and Immunological Aspects. Ed N Talal, HM Moutsopoulos, SS Kassan. (Pp 299; 51 figs and 63 tables; DM 218.) Springer. 1987. ISBN 3-540-17477-X.

This book presents an up to date broadly based review of clinical and research findings of Sjögren's syndrome. The book consists of chapters by a number of different authors well known for research in this field.

The introduction describes the historical aspects, the recognition of the syndrome in the 1930s by Sjögren, and the difficulties faced in having his work accepted. It also includes the patients' perspective of the disease and its morbidity. The first main section is on the clinical spectrum of the disease, which includes glandular and extra glandular findings, lymphoid malignancy, associations with other autoimmune diseases, diagnosis, and differential diagnosis. This is followed by the second main section on laboratory abnormalities, largely immunological, and which contains a most useful review chapter on animal models in Sjögren's syndrome. Finally, there is a chapter which provides an update on the treatment and management of patients with Sjögren's syndrome.

This book records a great deal of well referenced information about this unusual condition. As Dr Talal points out in his preface, the final chapter remains to be written. In the meantime, and to this end, this book will provide many research workers and all those who are interested in Sjögren's syndrome with the basic knowledge from which further progress can be made. It can be highly recommended to all workers in the field.

DK MASON

Human Inflammatory Disease. Clinical Immunology. Vol 1. G Marone, LW Lichtenstein, M Condorelli, AS Fauci. (Pp 327; £60.) Blackwell Scientific Publications. 1988. ISBN 1-55664-034-X.

This volume consists of lectures delivered at the 1st Capri Conference on Clinical Immunology, June 1986, and combines basic research with clinical aspects of inflammation. There are four subject groups: inflammatory cells and mediators, cardiovascular inflammatory disease, adenosine and adenosine receptors, and biochemical aspects of inflammation. A major strength of the book is its extensive coverage of the immunoregulatory effects of adenosine and of inflam-

matory processes in ischaemic heart disease, neither of which subjects have been extensively reviewed elsewhere. Individual chapters are particularly concise and informative, notably those by Rossi (IgE receptors), Kagey-Sobotka (mast cell mediators) and Hirschhorn (ADA deficiency). Conversely, topics equally worthy of consideration, such as complement anaphylatoxin receptors, leucocyte cytoadhesive glycoproteins, and inflammatory effects of cytokines, are conspicuously absent. In general, the level of presentations would suit a postgraduate student or young researcher seeking an introduction to the relevant field.

EF RIMMER

Human Retroviruses, Cancer, and AIDS. Approaches to Prevention and Therapy. UCLA Symposia on Molecular and Cellular Biology. New Series, Vol 71. (Pp 524; \$96.) Alan R Liss Inc. 1987. ISBN 0-8451-2670-9.

This volume represents the proceedings of one of the UCLA symposia on Molecular and Cellular Biology, held at Keystone, Colorado in April 1987. Despite its broad title, the 33 research contributions, save for one paper on foamy viruses from Flügel's group and one on a neurotropic mouse virus from Ruprecht, are wholly devoted to HIV. All these papers are research reports from individual laboratories whose results have simultaneously been published as papers in primary journals. Other than inserting extra data that did not pass muster in the peer review system, I fail to see any purpose for the academic community in publishing such proceedings. I must be in a minority as publishers like Alan R Liss Inc clearly find them profitable.

This volume might be useful for readers who wish to obtain a spectrum of papers, mainly by competent research groups, on the state of HIV art over one year ago. It covers the molecular biology of HIV, some half baked speculations on its pathogenesis, and now outdated results on experimental therapeutic and vaccine reagents. The symposium was surely a useful gathering for the participants, and that is where it should have rested.

There are, however, two excellent and perceptive overviews which are well worth reading by the Nobel prize winning discoverers of reverse transcriptase. In an introductory article, Howard Temin reviews the evolution of retroviruses and transposons, and as an epilogue David Baltimore reviews "HIV, 1987". Again, their views are available in numerous other journals so I would not buy the volume for them alone.

RA WEISS