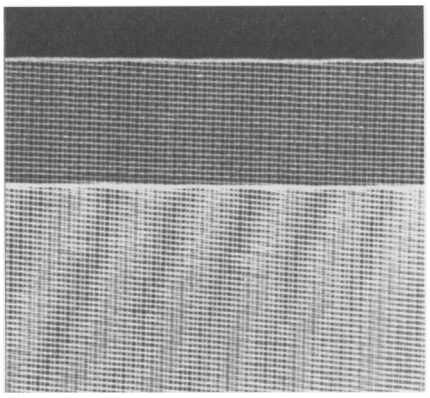

Figure 2 Shandon bag showing similar effect.

(fig 2) showed the pattern of the material to be similar in shape to the defect.

We have now returned to using perm paper and the artefact has disappeared. We have no experience of the use of teabags, but we recommend that perm paper is preferable to Shandon tissue bags (when processing renal biopsy specimens), as the latter may lead to considerable distortion of the tissue.

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1 Platt CC, Newman J. Tissue artefacts caused by sponges. *J Clin Pathol* 1993;**46**:780.
2 Farrell DJ, Thompson PJ, Morley AR. Tissue artefacts caused by sponges. *J Clin Pathol* 1992;**45**:923–4.

Dr Platt comments:
We read with interest the letter by Davison and Morley concerning elliptical defects which apparently develop whilst processing renal biopsy specimen in Shandon bags. Shandon bags are used routinely for the processing of small specimens in this department and we have not encountered tissue defects. However, renal biopsy specimens are routinely hand processed in this department and we have no experience of the difficulties that might arise in their processing in Shandon bags. The routine use of teabags remains a theoretical possibility which is cost attractive.

T cell lymphoid aggregates in idiopathic hypereosinophilic syndrome

Dr Metz and colleagues presented fascinating information which suggests that some cases of the hypereosinophilic syndrome (HES) may result from occult T cell proliferation and interleukin-5 (IL-5) secretion.[1] Unfortunately, in the absence of bone marrow genotypic studies, it must remain uncertain as to whether the proliferation in Dr Metz's case had a poly- or monoclonal origin. Interestingly, a relation between HES and cutaneous T cell lymphoma (CTCL) has also been highlighted in French publications, together with evidence that α interferon may be of therapeutic benefit.[2]

Dr Metz briefly discusses the relation between eosinophils and cutaneous T cell disease, but sadly makes no mention of helper T cell subdivision based on cytokine production. Recent evidence indicates that, as in the mouse, human T cells of the T_H1 subset synthesise and secrete interleukin-2 (IL-2) and γ-interferon (IFN), whereas those of the T_H2 subset produce interleukin-4 (IL-4) and IL-5 but not IL-2 and γ-IFN.[3] Furthermore, such cytokine profiles provide a new novel means by which to classify many cutaneous disorders. For example, diseases characterised by the presence of T_H2 cells (such as atopic dermatitis and CTCL) may display raised blood concentrations of IgE, IL-4, and IL-5.[4] Also, based on Dr Metz's findings, it seems reasonable to suggest that idiopathic HES may be a disease of T_H2 proliferation.

As well as HES, substantial cutaneous eosinophilic infiltrates are seen in the spectrum of eosinophilic fasciitis (Shulman's syndrome) and eosinophilic cellulitis (Well's syndrome). For several years, in common with Dr Metz's experience in HES, I have been impressed by the close association of eosinophils and T cell lymphoid aggregates in these conditions. Although there is no known association between eosinophilic cellulitis and CTCL, it is perhaps important that CTCL has recently been described as coexisting with eosinophilic fasciitis.[5] On this basis, investigations are already in progress to assess clonality and cytokine production in eosinophilic fasciitis and cellulitis, to ascertain whether these are additional T_H2 diseases.

The tinctorial brilliance of the eosinophil seems, to date, to have blinded histopathologists from appreciating that T cells may have substantial pathogenetic importance in this group of disorders.

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1 Metz J, McGrath KM, Savoia HF, *et al.* T cell lymphoid aggregates in bone marrow in idiopathic hypereosinophilic syndrome. *J Clin Pathol* 1993;**46**:955–8.
2 Moraillon I, Bagot M, Bournerias I, *et al.* Syndrome hypereosinophilique avec pachydermie precedant un lymphome. Traitement par l'interferon alpha. *Ann Dermatol Venereol* 1991;**118**:883–5.
3 Romagnani S. Human T_H1 and T_H2 subsets: doubt no more. *Immunol Today* 1991;**12**: 256–7.
4 Rook AH, Vowels BR, Jaworsky C, Singh A. The immunopathogenesis of cutaneous T-cell lymphoma. Abnormal cytokine production by Sézary T cells. *Arch Dermatol* 1993; **129**:486–9.
5 Chan LS, Hanson CA, Cooper KD. Concurrent eosinophilic fasciitis and cutaneous T-cell lymphoma. Eosinophilic fasciitis as a paraneoplastic syndrome of T-cell malignant neoplasms? *Arch Dermatol* 1991;**127**:862–5.

Dr Metz comments:
We thank Dr Slater for his comments, and agree that the prominence of eosinophils in conditions like HES has distracted attention from the possible cell of origin: a T cell. We would agree also that the T cells in our patient were probably of the T_H2 phenotype, and we in fact demonstrated detectable concentrations of IL-5. However, although the T_H1/T_H2 cell classification is increasingly applied to murine cells, its clinical relevance in human disease remains to be fully documented.

With regard to the clonality of the lymphocyte proliferation, studies on bone marrow, as we reported, failed to demonstrate any cytogenetic abnormality and genotypic studies on blood showed a polyclonal pattern. We did not consider it warranted to repeat the bone marrow biopsy for the sole purpose of obtaining material for genomic Southern blot analysis. Although it remains possible that a clonal population of T cells was not detected, this would mean that the patient had had a clonal T cell proliferation for 15 years, which seems most unlikely.

We look forward to the results of Dr Slater's investigations to ascertain whether these conditions characterised by eosinophilia represent T_H2 diseases.

Book reviews

The Molecular Pathology of Cancer. Cancer Surveys. Vol 16. Guest Eds. NR LEMOINE, NA WRIGHT. (Pp 239; $69). Published for ICRF by Cold Spring Harbor Laboratory Press. 1993. ISBN 0-87969-389-4.

The title promises much, but in fact this volume in the *Cancer Surveys* series aims to illustrate a series of models for selected cancers, and comprises a mixture of short articles or reviews on experimental and human cell biology, and molecular pathology. Interpretation of "model" includes animal, cell culture, and theoretical systems, so that the collection is a little heterogeneous. Of the 12 chapters, four are concerned with breast cancer (the article on human breast cancer by Drs Walker and Varley is particularly well presented) and two with coloretal cancer. The other half of the book deals with some molecular aspects of thyroid and pancreatic cancers, lymphomas, tumour metastasis and, slightly curiously, prospects for cervical cancer vaccines. There is also a brief review of oncogenes, growth factors, and control of the cell cycle, and a short biography of each of the 26 authors, many of whom are from the Imperial Cancer Research Fund laboratories. There is a certain amount of repetitiveness and, confusingly, the nomenclature for p53 has been changed to TP53 throughout, except in the references. References are mostly up to 1991, with a few from 1992. The book is well produced but sparsely illustrated, mostly with line diagrams and a few photographs of cell cultures or gels. It is difficult to know who buys this sort of book, but the reference lists could be useful to workers in the relevant fields.

C FISHER

Clinical Microbiology. 7th edn. E Joan Stokes, GL Ridgway, MWD Wren. (Pp 398; £26.50.) Edward Arnold. 1993. ISBN 0-340-55423-1.

It is with mixed feelings that I review this book. Having had the privilege of being trained by Dr Stokes, I cut my microbiological teeth on the fourth edition, and have frequently referred to it and subsequent editions. The changes in microbiology in the 90s and the role of the routine clinical laboratory have been addressed in the seventh edition.

The details of a busy routine microbiology laboratory are helpful, particularly in the prevailing climate. A clearer understanding of "value for money" is now