Letters to the Editor

erythematosus among the rheumatic diseases. Progress in the synthesis of DNA analogues and the identification of DNA determinants with monoclonal antibodies⁷ may, however, lead to a better understanding of the factors determining these immune responses and to improvements in the diagnosis and treatment of diseases in which they are detected.

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Tuberculosis and “sterile” pyuria

In the past “sterile” pyuria suggested the existence of tuberculosis of the urinary tract. In common with all forms of the dis-

ease the incidence of genitourinary tuberculosis has declined but the Medical Research Council survey of the notification of tuberculosis reported 154 such cases in the six months spanning the end of 1978 and the beginning of 1979.¹ These contributed 14% of all cases of non-respiratory disease.

“Sterile” pyuria is still common. To investigate the local situation every specimen received from general practitioners in the Cardiff area over a three year period which showed “sterile” pyuria was cultured for *Mycobacterium tuberculosis*. Between July 1979 and September 1982 a total of 803 samples were tested, of which three (0.4%) yielded *Myco tuberculosis*.

Urinary tract tuberculosis was therefore clearly not a major cause of “sterile” pyuria. A possible alternative explanation was that many of these patients had recently taken, or were still taking, antimicrobial agents at the time of sampling. A study of the request forms showed that antimicrobial treatment was mentioned in 161 (20.0%). One of the positive isolations came from this group.

Information on request forms is notoriously unreliable. All urines showing “sterile” pyuria were therefore tested for antimicrobial activity using a modification of the method described by Pelling,² and the actual findings were compared with the expected findings. The results are shown in the Table. They were discrepant in 47 (19.7%) of the 239 cases.

The local incidence of urinary tract tuberculosis in patients whose urine showed “sterile” pyuria was low, and so the cost effectiveness of surveying all such samples must be equally low. It is not possible to reduce the number of specimens requiring screening by excluding all those reported as being on antimicrobial therapy because one of our three cases came from this group and the accuracy of the reported data on the request forms is low.

Of the 11 bacteriologically proved cases of urinary tract tuberculosis which occurred in South Glamorgan between July 1979 and September 1982, three (27.3%) were detected as a result of this survey.

Such patients are unlikely to pose a major infection hazard to other members of the community, but all three cases detected by the survey were unsuspected clinically and one suffered complications which may have been related to his infection.

It is the practice of this laboratory therefore not to examine every specimen showing “sterile” pyuria for *Myco tuberculosis* but to advise local practitioners to consider the diagnosis of urinary tract tuberculosis when persistent unexplained “sterile” pyuria is observed.

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Batch screening method for detection of bacteriuria

While agreeing with Mr Kerfoot and his colleagues¹ that the increasing workload in urine microbiology places a considerable strain on laboratories, I believe that it is important that protocols for examining urine specimens should not give wrong, or unduly delayed, answers. I therefore have some reservations about the batch screening method which they describe. Firstly, and perhaps most important, I do not consider that it is possible for patients, especially women, to pass a mid-stream urine specimen into a small container of the size they use. If their procedure—and they do not say so—is that the urine is first passed into a container with a wide mouth, and then decanted, the cost of specimen collection must be greatly increased.

Secondly, since the boric acid preservation method was first described² further work³ has shown that the percentage of false negatives may be as high as 16%. Many would not consider it a satisfactory method of urine preservation, especially when definitive culture, as described by Kerfoot *et al*, may not be undertaken for up to 48 h after the specimen is collected.

This screening method inevitably delays definitive culture and sensitivity testing by at least 24 h. Kerfoot *et al* do not say

Comparison of expected and actual results of tests for antimicrobial activity in urine

	Antimicrobials mentioned on request form	
	Yes	No
Antimicrobials found in urine		
Yes	19	33
No	14	173

whether primary sensitivity testing is done on the stored urine specimens; if it is delayed until the definitive cultures are read the delay will be still longer.

Lastly, although the method they describe must obviously save money in terms of labour, the cost of their method of definitive culture may well equal that of plating all specimens on one third of a CLED plate.

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Mucosal prolapse syndrome

Dr du Boulay and her colleagues¹ should be congratulated for their suggested rationalisation of what has never been completely satisfactory terminology for the histological picture associated with rectal mucosal prolapse. Their concept that many of the histological features are due to ischaemia, possibly from torsion of sub-mucosal arteries, is almost certainly correct in the case of surface erosion and loss of sulphated mucin from goblet cells. The distortion of crypt architecture may also be related to ischaemia.

I am unhappy, however, about their reference to the "disorganisation of the muscularis mucosae with extension of fibromuscular tissue into the lamina propria." What, in fact, happens in mucosal prolapse is that the chronic stretching and shearing of the lamina propria inherent in the process results in hypertrophy of the smooth muscle fibres which are normally inconspicuously present in the lamina propria. If one carefully examines a histologically normal rectal biopsy specimen moderate numbers of slender smooth muscle cells can always be seen in the lamina

propria, in continuity with, but at right angles to, the muscularis mucosae. The muscularis mucosae itself also undergoes hypertrophy (not "disorganisation") in the mucosal prolapse syndrome and, by contracting at the time of biopsy, often causes bunching and spurious thickening of the overlying mucosa.

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- ¹ du Boulay CEH, Fairbrother J, Isaacson PG. Mucosal prolapse syndrome—a unifying concept for solitary ulcer syndrome and related disorders. *J Clin Pathol* 1983; **36**:1264-8.

Book reviews

Techniques in Immunocytochemistry. Vol. 2. Ed GR Bullock and P Petrusz. (Pp 290; £25.) Academic Press. 1983.

These are the first two of several volumes in a proposed series dealing with the theoretical and practical aspects of immunocytochemistry. The books contain contributions from several authors, each of which represents some of the highest level of authority and expertise in the various topics discussed. At the outset, the objective was to compile a series of volumes in which new techniques could be critically "assessed" as well as established methods presented and progressively revised. Although each volume deals with diverse aspects of immunocytochemistry the editors should be congratulated on having maintained a uniform and easily comprehensible style of writing which makes these books a pleasure to consult.

Within the two volumes each chapter contains details of the methods the individual authors have found most effective, together with protocols for preparing buffers and reagents. Volume One deals with some of the fundamental processes of immunocytochemistry and includes chapters on tissue fixation, double immunoenzymatic labelling, and the application of proteolytic enzymes for the improved localisation of tissue antigens. Volume Two concentrates more specifically on several

different aspects of the use of colloidal gold and upon the avidin-biotin system for enhancing immunocytochemical localisation at the light and electron microscopic levels. The references cited throughout all chapters are very adequate and not only provide additional authority but also an enhanced depth of expertise for many of the statements made. These two volumes provide useful and detailed first-hand experience of tackling many of the problems which are frequently encountered in the employment of antibodies (both monoclonal and polyclonal) and lectins as immunocytochemical reagents at the light and electron microscopic level.

I recommend this series of books as a valuable introduction for the novice immunocytochemist and, more particularly, as useful and often enlightening reading for the experienced immunocytochemist.

CS FOSTER

Clinical Aspiration Cytology. Ed Joseph A Linsk and Sixten Franzen. (Pp 386; £50.) JB Lippincott Company. 1983.

With several well known names amongst the ten authors one would expect a very high standard and this is achieved. This book deals with fine needle aspiration cytology of all sites from the central nervous system to the testis, and includes deep lesions accessible only with the aid of imaging procedures.

For each anatomical site in the body the method is described, the appearances of the cells as stained both by Romanowski and Papanicolaou methods are illustrated, and the place of the cytology report in the clinical management of the patient is discussed. All these features are illustrated by more than 600 well positioned black-and-white photographs each with a very helpful legend, together with several diagrams and tables. As an additional bonus there are 17 high quality colour plates including 90 photomicrographs.

Throughout the authors readily acknowledge the histological basis of cytological diagnosis, and this is well illustrated.

This reasonably priced book may well become the standard work on needle aspiration cytology. Pathologists, clinicians, and radiologists will all find this a most useful description of the subject and an invaluable bench book.

JV LEVER