The temporal arteritis/polymyalgia rheumatica syndrome is a relatively common disorder that is regularly stated to be of unknown aetiology. We respectfully submit that its likely actinic basis is supported by sound observations and deserves wider recognition.

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Drs Wawryk et al comment:

O'Brien *et al* make the interesting suggestion that giant cell arteritis represents an auto-immune response against actinically altered arterial elastic lamina. The authors correctly identify a close relation between macrophages and the internal elastic lamina, as illustrated in our paper. We consistently found that macrophages which express p150/95 and are found in close apposition to the internal elastic lamina strongly expressed ICAM-1 and HLA-DR. A granular pattern of staining for these markers was also seen along the elastic lamina in affected arteries, possibly representing the expression of these molecules on dendritic processes ramifying along the elastic lamina. The functional relation between macrophages showing this phenotype and the elastic lamina, however, and in particular actinically damaged elastic tissue, remains uncertain.

Blood and bone marrow cultures in enteric fever

Dr Farooqui and colleagues present data which support the conclusion that bone marrow culture gives a higher yield than

blood culture in patients with enteric fever.¹ Although we agree in general with their suggestion that "bone marrow culture could confirm a diagnosis of typhoid fever in patients whose blood cultures are negative," we wish to make some further observations.

There is a considerable body of published work which compares bone marrow culture with blood culture for the diagnosis of enteric fever to which Farooqui *et al* did not refer. Many of these studies were summarised at a workshop in 1984.² Most workers have concluded that bone marrow culture is superior to blood culture for the diagnosis of enteric fever, particularly in patients who have received antibiotics. None of these studies, however, used optimal blood culture techniques; most compared a single set, often containing a small volume of blood (2-3 ml), with bone marrow culture. In several studies, including that of Farooqui and colleagues, sodium polyanethol sulphate (liquoid) was not included in the culture broth, and cultures were only incubated for seven days. Liquoid has been shown to antagonise both the intrinsic bactericidal activity of blood and that of certain antibiotics,³ while subculture of blood cultures after the seventh day of incubation may occasionally yield *Salmonella typhi*.⁴ Farooqui *et al* mention the possible effect of antibiotics on blood cultures, but they present no data on the previous treatment of their patients.

Our own data, obtained during studies of the antibiotic treatment of typhoid in Kathmandu, Nepal, are shown in the table. On admission to the studies, three blood culture sets (5 ml blood in 50 ml brain heart infusion broth containing liquoid; Gibco UK) were collected at least 15 minutes apart. Bone marrow (0.5-1 ml) was collected into 20 ml of the same medium. Although the numbers are small, the results show that blood cultures may be positive when bone marrow is negative, and vice versa. Two of the three patients with positive bone marrow and negative blood cultures had received antibiotics (chloramphenicol and cotrimoxazole) within the preceding three days. In two blood culture positive cases at least one blood culture set was negative.

We believe that further studies of the many possible variables are necessary before it is known whether bone marrow culture is superior to blood culture for the diagnosis of enteric fever. At present, we regard the two techniques as complementary. We would therefore disagree with the approach suggested by Farooqui and colleagues—that is, that bone marrow should be cultured in suspected cases of enteric fever only when blood culture is negative after three to four days of incubation. To optimise the yield of bacteriological investigation, we suggested that, whenever possible, both blood and bone marrow should be cultured when patients with suspected enteric fever are admitted.

Results of paired blood and bone marrow culture from 30 patients with suspected enteric fever

Blood	Bone marrow	Number
Positive	Positive	12
Positive	Negative	2
Negative	Positive	3
Negative	Negative	13

Of positive cultures, 14 grew *S typhi* and three grew *S paratyphi A*.

The reason most often given for the failure to culture bone marrow is the invasive nature of the procedure.⁵ Bone marrow aspiration with a fine bore needle, however, has been used successfully for the diagnosis of typhoid and is well tolerated.⁴

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- 1 Farooqui BJ, Khurshid M, Ashfaq MK, Ata Khan M. Comparative yield of *Salmonella typhi* from blood and bone marrow cultures in patients with fever of unknown origin. *J Clin Pathol* 1991;44:258-9.
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Diagnosis of acute hepatitis B by qualitative assay of specific IgM antibody

Diment¹ disputes my conclusion that qualitative assay of high titre hepatitis B core IgM antibody (anti-HBc IgM) responses has a limited role in the diagnosis of acute hepatitis B surface antigen (HBsAg) positive hepatitis.² He believes that the disappearance of anti-HBc IgM reactivity in a relatively insensitive assay occurs two months after the onset of acute hepatitis B¹ and is therefore manifest as soon as "e" antigen to "e" antibody seroconversion takes place.²

My observation of anti-HBc IgM persistence beyond "e" antigen "e" antibody seroconversion was considered to reflect use of a particularly sensitive assay. Studies of serial anti-HBc IgM responses in acutely infected patients, however, have not detected loss of antibody until four months, even when the assay used was sufficiently insensitive to give, virtually always, negative results with serum specimens from patients with chronic disease.^{3,4} Though a relatively insensitive anti-HBc IgM assay may be preferable for diagnosis of acute hepatitis B, its use may be confounded by the low titres of antibody occasionally found early in acute hepatitis B.^{4,5} Thus detection of "e" antigen "e" antibody seroconversion one to two months after onset remains the most certain method of confirming the diagnosis of HBsAg positive acute hepatitis B.

I agree partly with Dr Howell and colleagues.⁶ A presumptive diagnosis of acute hepatitis B can be made by detection of a high titre of HBsAg in serum by reverse passive haemagglutination (provided false positive

reactions are excluded) when the patient has a relevant clinical history. In patients without a relevant clinical history, serial assay of "e" antigen and "e" antibody may be preferable to serial assay of HBsAg titre. The former method is qualitative; the latter relies on precise quantitation of HbsAg to detect a clinically important decline in titre.

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- 1 Diment JA. Sensitivity of anti-HBc IgM kits and the diagnosis of acute hepatitis B. *J Clin Pathol* 1991;44:438.
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Morris¹ and Diment² have discussed the relative merits of anti-HBc IgM assays and HBeAg to anti-HBe seroconversion in the diagnosis of acute hepatitis B infection. Although these assays certainly have a place in the diagnosis of difficult cases, they are relatively expensive and usually only available in specialist laboratories.

We recently identified an asymptomatic acute hepatitis B virus infection in a blood donor, using three, simple, low cost criteria: (i) HBsAg positivity, at high titre (RPHA 1/10 000); (ii) alanine transferase activity (ALT) noticeably raised (2 200 IU/l); (iii) relevant recent history.

If these tests and criteria had not provided a diagnosis then testing a follow up sample for HBsAg titre and ALT activity would, in our experience, usually provide final confirmation. To complement this, and where there is a pressing clinical need for rapid diagnosis, we would have the sera tested for the markers discussed by Morris¹ and Diment.²

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- 1 Morris DJ. Anti-HBc IgM assays and diagnosis of acute hepatitis. *J Clin Pathol* 1990;43:782-3.
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BOOK REVIEWS

All titles reviewed here are available from the BMJ Bookshop, PO Box 295, London WC1H 9TE. Prices include postage in the United Kingdom and for members of the British Forces Overseas, but overseas customers should add 15% to the value of the order for postage and packing. Payment can be made by cheque in sterling drawn on a United Kingdom bank, or by credit card (Mastercard, Visa or American Express) stating card number, expiry date, and full name.

Reaction Patterns of the Lymph Node. Part 1 Cell Types and Functions. Current Topics in Pathology. Eds E Grundmann and E Vollmer. (Pp 288; 102 figs; DM268.00.) Springer. 1990. ISBN 3-540-52287-5.

This is the first of a two volume set in the *Current Topics in Pathology* series, describing the morphological and immunohistochemical changes within the various functional compartments of the lymph node; the second volume describes changes occurring in lymph nodes in association with immunodeficiency and neoplasia.

The contents include chapters correlating structure and function within various lymph node compartments, descriptions of the germinal centre response, of T lymphocytes in non-neoplastic lymph nodes and plasmacytoid T cells, and of macrophages and accessory cells. The volume concludes with an in-depth study of immunoelectronmicroscopy of lymph nodes.

The authorship of this book shows a strong European bias (20 of the 25 contributors) and in some of the chapters it is apparent that English is not the authors' first language, which makes for a rather turgid style. Nevertheless, this is an admirable book for the lymph node enthusiast containing information not often seen in other pathological books. The text is well illustrated with high quality photomicrographs, electron-micrographs, and line drawings. Most of the chapters are extensively referenced and contain references up to 1989.

Although this book will not be of particular interest to the general surgical pathologist, it can be strongly recommended to histologists and immunologists with a particular interest in structural and functional correlation within the immune system.

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Standard Haematology Practice. Ed Bryon Roberts on behalf of the British Committee for Standards in Haematology. (Pp 264; £39.50.) Blackwell Scientific Publications. 1991. ISBN 0-632-02623-5.

Of these collected reports from the British Committee for Standardisation in Haematology (BCSH), the best is on haemoglobinopathy screening, with good accounts of G6PD deficiency, some aspects of blood-banking, massive blood loss, management of anticoagulant treatment, thrombophilia and transfusion "menus".

The title is misleading, however, and should perhaps have been "Standards for Some Aspects of Haematological Practice".

Standard haematological practice in the United Kingdom today is surely broader than this book implies. Excluded are laboratory aspects of cytology and cytochemistry, general coagulation, cytogenetics, haematinic assays, haemolysis, immune cytopenias, cellular immunophenotyping and viscometry, not to mention clinical management of anaemia, many aspects of haemostatic failure, and haematological malignancies. Yet 8% of the book is an uncritical description of haemapheresis and 10% an account of blood-bank microplate techniques which most NEQAS contributors do not use. This last chapter is at least a positive if unbalanced statement unlike chapters 2 to 4 (another 10%) on automated cell counters and manual blood films which will probably neither reflect nor influence selection criteria for the latter in most United Kingdom laboratories.

Chapter 1 epitomises the heterogeneity of style, breadth, and depth of this book. It is intended as a description of "good laboratory practice". But with no emphasis on the importance of the medical consultation process, NEQAS, and the CPSM and College accreditation schemes it is unbalanced, incomplete, and poor PR for the specialty.

Those who have not collected BCSH reports as they appeared may welcome this convenient but costly binding. Nobody should think that it describes British haematology, standard or otherwise. Perhaps an early second edition will present a more balanced and comprehensive description of the specialty.

AG PRENTICE

Immunology: A Short Course. 2nd edn. E Benjamini, S Leskowitz. (Pp 559; \$29.95.) John Wiley & Sons Inc. 1991. ISBN 0-471-56751-5.

The authors have succeeded in their aim to present "the bare essentials of immunology in a palatable form..." which "will enable most students to grasp the essential principles". They have produced "lecture size" chapters which are readable and clear. Medical students will enjoy the snippets of clinical information which make the text interesting and relevant. There is good layout of the text and excellent diagrams. Review questions at the end of each chapter are quite detailed and will highlight any areas of inattention for the reader.

This book is very much a basic course and is not really a standard text, nor does it seem that it was intended to be. Any immunology text of basic principles needs to be selective in order to make it clear. Students will find it a great help, especially those who find immunology difficult or boring, and this text is neither.

Like all authors in this discipline, Benjamini and Leskowitz had to produce a second edition relatively quickly to cover the rapidly developing fields of genetic control, T cell differentiation, and therapeutic advances for immunological diseases as well as AIDS. The only criticism is that the index is awful and does not come up to the high standards of the book.

H CHAPEL

Pathology Reviews 1990. Ed E Rubin, I Damjanov. (Pp 255; \$74.50.) ISBN 0-896023-195-0.

This is an anthology of articles published in *Laboratory Investigation* under the "Biology