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 aetactic basis is supported by sound observations and deserves wider recognition.

JF O’BRIEN
Pathology Laboratory, Wales Medical Centre, Sydney
W REGAN
Department of Dermatology, Repatriation General Hospital, Sydney
E G CLEARY
Department of Pathology, University of Adelaide, Australia


Des Watryk et al comment: O’Brien et al make the interesting suggestion that giant cell arteritis represents an auto-immune response against a genetically 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 macropages which express p150/95 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 laminae altered 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 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 sulphonate (liquoid) was not included in the culture broth, and cultures were only incubated for seven days. Liquid 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 carrier 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 liquid; 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 co-trimoxazole) 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 bacillotroph-positive patients, we suggest 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

<table>
<thead>
<tr>
<th>Blood Culture Positive</th>
<th>Bone Marrow Culture Positive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive (12)</td>
<td>Positive (9)</td>
</tr>
<tr>
<td>Positive (3)</td>
<td>Positive (3)</td>
</tr>
<tr>
<td>Positive (5)</td>
<td>Positive (13)</td>
</tr>
</tbody>
</table>

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.2 Bone marrow aspiration with a fine bore needle, however, has been used successfully for the diagnosis of typhoid and is well tolerated.6

DAB DANCE
Department of Clinical Microbiology, London School of Hygiene and Tropical Medicine, Keppel Street, London WC2H 9SH, UK

MO HMO Microbiology and Infectious Diseases, University of Calgary, 100 Hospital Drive NW, Calgary, Alberta, Canada T2N 4N1

GACHARYA G
POKHREL P
TULADHAR R
Tripathi University Teaching Hospital, Kathmandu, Nepal


Diagnosis of acute hepatitis B by qualitative assay of specific IgM antibody

Diment 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.7 He believes that the disappearance of anti-HBc IgM reactivity in a relatively insensitive assay occurs two months after the onset of acute hepatitis B and is therefore manifest as soon as “e” antigen to “e” antibody seroconversion takes place.

My observation of anti-HBc IgM persistence beyond “e” antigen to “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.8 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.9 Thus detection of “e” antigen to “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. A presumptive diagnosis of acute hepatitis B can be made by detection of a high titre of HBsAg in serum by the Widal CHAREY haemagglutination (provided false positive

References
Morriss and Diment have discussed the relative merits of anti-HBc IgM assays and HBsAg to antibody conversion in the diagnosis of acute hepatitis B. 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 (RFHA 1/10 000); (ii) alanine transaminase (ALT) activity (ALT) noticeably raised (2 200 IU/L); (iii) recent relevant 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 Moriss and Diment.

DR HOWELL
JA BARBARA
PE HEWITT
National Blood Transfusion Service,
North London Blood Transfusion Centre,
Colindale Avenue,
London NW9 5BG


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, haematological 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-banking principles 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 4 epitomises the heterogeneity of style, breadth, and depth of this book. It is intended as a description of "good laboratory practice". They have included "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 enhanced with colour photographs. Yet 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


This is an anthology of articles published in Laboratory Investigation under the "Biologic
Diagnosis of acute hepatitis B by qualitative assay of specific IgM antibody.

D J Morris

doi: 10.1136/jcp.44.12.1038-b

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