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 autoimmune 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 sulphonate (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 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 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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Diagnosis of acute hepatitis B by qualitative assay of specific IgM antibody

Diment[1] 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 B[1] 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