on causal but is sympathetically pathetic. There is no evidence that sympa-
thetic blockade, implying thermal imaging and triple phase bone scan. We must not forget, however, that both techniques only reflect
circulatory or metabolic alterations and not direct injury of the sympathetic nervous system, which could only be shown by electro-
physiological studies. To our knowledge these studies do not exist in reflex sympathetic dystrophy, but undoubtedly they would be
revealing.

It is obvious that the patient whose case we reported fulfills the reflex sympathetic dystrophy diagnostic criteria of Kozin, Dam-
geat, and other experienced authors. Even so, as we mentioned in the discussion, the patient presented characteristics ruling out a
specific clinical syndrome (absence of cyanosis and hyperhidrosis, shoulder not affected, prolonged duration, and rapid heal-
ing after suppression of the causal agent), and for this we prefer to use the term 'mimic'. In our view this case is an example of the
terminology, diagnostic and clinical confusion that exists around reflex sympathetic dystrophy that we have already men-
tioned on other occasions and that Dr Thomas has alluded to.

(As Dr Thomas notes, in fig 3 the bone scan pictures were incorrectly labelled. Additionally, fig 2 should show the left hand affected rather than the right.)

Rheumatoid factors and the disease state in rheumatoid arthritis

SIR: I read with interest the paper by Jones et
al. on the reactivity of rheumatoid factors with IgG of various species; and, in particular, their finding that in solid phase capture assay rheumatoid factors specifically bind human and baboon IgG but not the IgG of other subhuman primates.

In a study carried out in our laboratory some years ago1 we modified the standard Waaler-Rose haemagglutination method of
assaying rheumatoid factor by using sheep red cells sensitised with a subhuman primate IgG antibody. This allowed a greater degree of immunochemical cross reactivity with human IgG than does rabbit IgG antibody cus-
tomarily used for this purpose, and would therefore permit the use of the system in the inhibition-agglutination mode for the study of the
interaction of rheumatoid factors with human IgG isotypes and their enzymatic cleavage fragments. We chose to produce IgG antibodies against sheep red cells in baboons for two reasons—firstly, because we were using these subhuman primates at the time in other investigations in the allergy field and, secondly, radioimmune inhibition studies2 had shown a high degree of cross reactivity between the IgG of this species and human IgG heavy chains.

When sheep red cells sensitised with baboon instead of rabbit IgG were used it was possible to show inhibition-haemagglutination by human myeloma proteins of the IgG1, IgG2, and IgG4, but not the IgG3 isotype, nor rabbit IgG, while heat aggregated forms of all four human IgG isotypes were inhibitory. We also found that aggregated baboon IgG, unlike aggregated rabbit IgG, possessed inhibitory activity comparable to that of aggregated human IgG in this system.

In summary, our findings from that early study of the inhibition of haemagglutination by rheumatoid factors of sheep cells coated with baboon IgG are very much in line with the results now reported by other workers and the findings obtained by more modern solid phase
capture assay systems.
Rheumatoid factors and the disease state in rheumatoid arthritis.

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