settled and the biochemical abnormalities came back to normal, including parathyroid hormone of 0.48 ng/l.

Our patient had dietary osteomalacia and secondary hyperparathyroidism which settled completely with calciferol treatment.

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Reference

- 1 Jawad A S M, Squire S B, Perry J D. Osteomalacia and coeliac disease presenting as isolated dactylitis. *Ann Rheum Dis* 1988; 47: 692-4.

SIR, The case report presented by Drs Sultan and Bruckner concluding that the dactylitis seen in their patient was a manifestation of secondary hyperparathyroidism is in keeping with conclusions we published previously.¹

We pointed out that the radiological bone changes in our patient's hand were confined to the middle phalanx, and although they may well have been caused directly by the osteomalacia, it seemed more likely that they were a manifestation of secondary hyperparathyroidism. Clearly the finding of a raised parathyroid hormone is not unexpected and does not prove that the radiological changes were due to hyperparathyroidism, particularly as in our case the histology was non-specific, though tetracycline labelling was not performed.

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Reference

- 1 Jawad A S M, Squire S B, Perry J D. Osteomalacia and coeliac disease presenting as isolated dactylitis. *Ann Rheum Dis* 1988; 47: 692-4.

Inhibition of neutrophil myeloperoxidase by rabbit anti-(human myeloperoxidase)

SIR, In the paper by Nurcombe and Edwards published in the *Annals*¹ the authors claim to have shown inhibition of fluid phase neutrophil myeloperoxidase by the IgG fraction of an anti-myeloperoxidase antibody in a chemiluminescence system. But incredibly for such a reputable scientific journal they have been allowed to present their data (Fig. 5) without any reference to the effect of normal

rabbit (control) IgG on fluid phase myeloperoxidase in the same system. Even if it had had no effect, this should have been mentioned. We have been using a similar system to evaluate the effect of antibodies to myeloperoxidase on the activity of the fluid phase enzyme, and there is undoubtedly considerable protein quenching by normal immunoglobulin (with apparent inhibition) of the chemiluminescence generated by fluid phase neutrophil myeloperoxidase, compared with activity in the absence of normal IgG. This must be taken into consideration in the interpretation of any 'inhibition' occurring in the presence of anti-myeloperoxidase IgG.

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Reference

- 1 Nurcombe H L, Edwards S W. Role of myeloperoxidase in intracellular and extracellular chemiluminescence of neutrophils. *Ann Rheum Dis* 1989; 48: 56-62.

SIR, Drs Thompson and Lee comment on the fact that in Fig. 5 (and 6) of our recent paper 'Role of myeloperoxidase in intracellular and extracellular chemiluminescence of neutrophils',¹ we did not state the effects of equivalent amounts of non-immune IgG on this system. In such experiments we routinely measure the effects of non-immune IgG^{2,3} as some types of chemiluminescence are susceptible to non-specific quenching by soluble proteins. The problem of non-specific protein quenching by antibodies is greatly reduced when IgG fractions are purified from high titre antisera. In our experiments such non-specific quenching of extracellular chemiluminescence by equivalent amounts of non-immune IgG was only 5-10% of that observed by our anti-(myeloperoxidase) IgG and hence the effects noted in Figs 5 and 6 were due to specific inhibition of myeloperoxidase.

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References

- 1 Nurcombe H L, Edwards S W. Role of myeloperoxidase in intracellular and extracellular chemiluminescence of neutrophils. *Ann Rheum Dis* 1989; 48: 56-62.
- 2 Edwards S W, Swan T F. Regulation of superoxide generation of myeloperoxidase during the respiratory burst of human neutrophils. *Biochem J* 1986; 237: 601-4.
- 3 Edwards S W. Luminol- and lucigenin-dependent chemiluminescence of neutrophils: role of degranulation. *J Clin Lab Immunol* 1987; 22: 35-9.