LETTERS TO THE EDITOR

TREATMENT OF GOUT AFTER TRANSPLANTATION
Sir—Byrne et al. [1] have recently reported two cases of the successful control of gout after cardiac transplantation with allopurinol and a reduced dose of azathioprine. Gout may become a problem in cardiac transplant recipients [2] as they are usually treated with cyclosporin A and diuretics. Hyperuricaemia is a common side-effect of cyclosporin therapy [3] and, in patients on cyclosporin A, hyperuricaemia is more frequent in those who are also on diuretic therapy [4]. Both cyclosporin and diuretics induce hyperuricaemia by reducing urinary excretion of urate [4, 5].

In the two patients reported [1], azathioprine was reduced in order to avoid side-effects due to the concurrent administration of allopurinol, and the dose of frusemide was also reduced. One of them suffered a further attack secondary to an increase in the dose of frusemide. Data regarding renal function and renal handling of urate in these patients would be of interest, because they would have probably shown severe under-excretion of urate. Uricosuric therapy would then appear to be preferable to allopurinol, especially in patients in whom the dose of azathioprine or diuretics could not be reduced.

Recent reports have demonstrated the usefulness of benzofurans such as benziodarone and benzbromarone for the therapy of hyperuricaemia and gout in renal transplant recipients despite moderate renal insufficiency [4, 6] and in patients receiving cyclosporin [7]. The use of uricosuric drugs is probably a more pathogenic approach to therapy in patients showing underexcretion of urate and will avoid the risks of combining allopurinol and azathioprine (especially myelotoxicity) in transplant recipients. Recently, Cummings et al. [8] have suggested that uricosuric drugs should be considered for treating hyperuricaemia in such cases.

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ANTIMALARIAL DRUGS IN THE TREATMENT OF RHEUMATOLOGICAL DISEASES
Sir—The review by Rynes [1] of the use of antimalarials was timely and, in most respects, extremely helpful. However, the comments about ophthalmological supervision, particularly as far as hydroxychloroquine is concerned, should not be allowed to pass unchallenged. The author produces very little evidence to suggest that there is a true problem with retinal toxicity when using low-dose hydroxychloroquine. Bearing in mind the vast experience of using this drug over 30 yr or so, it is remarkable just how few case reports there are of toxicity, including in longitudinal studies.

The author works in a country where there are a large number of ophthalmologists and where screening is relatively easy. In many countries, this luxury is not available and if Rynes’ advice is followed then patients who would benefit from the drug will be denied it. I have not undertaken routine ophthalmological review of patients on hydroxychloroquine for over 20 yr and am a relatively frequent user of the drug. I do get an ophthalmological opinion on the over-seventies so as to establish a baseline in case of visual deterioration later so that any pre-morbid changes can be recorded. In these days of evidence-based medicine, I would urge that suggestions about unreasonable monitoring are critically appraised and that, as a speciality, we abandon what is, in medical terms, a fairly expensive and relatively useless test.

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KELLEY–SEEGLINNER SYNDROME: A CASE REPORT AND REVIEW OF THE LITERATURE
Sir—We would like to report the case of a 26-yr-old man referred to our department with poorly controlled gout in spite of treatment with allopurinol. He was investigated for loin pain and haematuria at the age