our colleagues in Middlesbrough have reached similar conclusions from their data.

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Uric acid and intelligence

SIR, It was recently claimed that the superior intellectual powers of the higher primates may be to some extent a consequence of high uric acid levels. This is to let you know that we had the opportunity some time ago to investigate the correlations between serum uric acid level and 'intelligence.'[1] We studied 270 children aged 0 to 16 years (including subjects with epilepsy, with behaviour problems, with mental deficiency, and overgifted subjects). The results lend substantial support to the hypothesis that serum uric acid is related to intellectual level in the paediatric age group (mean serum uric acid level in mentally retarded children =3·98, in 'overgifted' children =4·77).

We may add that in our study we decided to investigate a number of children in order to exclude the many variables (so often stress in adults, eating habits, etc.) which could play an important role in the uric acid level in the adult population. In our search of the medical world literature we could not find any other investigator who had studied before us the same subject *in the paediatric age group.*

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Reference

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Streptococci and reactive arthritis

SIR, Reactive arthritis is a term reserved for a sterile polyarthritis following a variety of infections. In patients with the HLA B27 antigen the syndrome commonly includes sacroiliitis and a symmetrical, predominantly lower limb arthritis. It has been described as a consequence of shigella, chlamydia, and yersinia[1,2] infections. We have recently seen a case of reactive arthritis in an HLA B27 positive individual following a streptococcal throat infection.

A 22-year-old woman presented to her general practitioner in January 1980 with a short history of a sore throat. A clinical diagnosis of tonsillitis was made and the patient given penicillin. Although her sore throat rapidly improved, within 4 weeks she had developed a progressive, symmetrical polyarthritis with painful swelling of her knees and ankles. She complained of stiffness and pain in the low back. There was no history of urethritis, conjunctivitis, or gastrointestinal disturbance.

On examination she was afebrile, with no rashes or heart murmurs. She had evidence of a tender arthropathy, with synovitis and effusions in both knees and ankles and tenderness over both sacroiliac joints. Investigations revealed an erythrocyte sedimentation rate of 100 mm in the first hour. The initial ASO titre was 8330 units/ml. Rheumatoid factor and antinuclear factor were both negative. Radiology of joints showed no abnormality. The patient was HLA B27 positive.

Treatment consisted of anti-inflammatory agents, but improvement was slow. There were a number of exacerbations over a 12-month period, one requiring corticosteroid therapy. Throughout this period sacroiliac pain remained a prominent feature.

The association between the reactive arthritis, sacroiliitis, and streptococcal throat infection may be coincidental. However, the timing, the pattern of the disease, and the lack of other obvious triggering factors suggest that streptococci may need to be considered in the list of infections known to precipitate this condition.

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Allopurinol effect on renal function in gout

SIR, Many of your readers must regret that you no longer publish the discussion of papers which have been read to the Heberden Society. This was brought home to me by

reading the paper by Dr Gibson and his colleagues dealing with the influence of allopurinol treatment on renal function in gout.[1] In the discussion which followed the original presentation of this work at the society's meeting in November 1979 several speakers pointed out important criticisms of design and analysis which these authors have left unanswered. In my opinion these errors must continue to cast doubt on the validity of the conclusions they have drawn.

Central to these criticisms is the allocation of patients to treatment groups. Three patients who were prescribed colchicine and allopurinol but whose serum uric acid levels did not fall were considered to have been prescribed colchicine alone and transferred to that group 'for the purpose of the analysis.' A further patient was withdrawn from the trial because of large tophi, prescribed allopurinol, and included in the analysis. Thus 4 patients, making up 15–20% of their allopurinol group (depending on which set of measurements are being considered), were allocated outside the trial protocol. The result, as was made clear in the 1979 discussion, is to make all subsequent calculations, manipulations, and interpretation of the data irrelevant to clinical decisions regarding the prescription of allopurinol for patients.

This is further supported by inspection of the results tables in this paper, which show that many (if not all) of the significant changes reported are critically dependent on the inclusion or exclusion of one or 2 patients in one group rather than another. The real significance of these changes is thus entirely dependent on the validity of the initial allocation of patients to the groups.

Gibson and colleagues have the data and could have recalculated the between-group changes using appropriate patient allocations. With such an important topic it would still be worthwhile and relatively simple to do this.

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Reference

1 Gibson T, Rodgers V, Potter C, Simmonds H A. Allopurinol treatment and its effect on renal function in gout: a controlled study. *Ann Rheum Dis* 1982; **41:** 59–65.

SIR, It is nice of Dr J. R. Kirwan to pay so much attention to our paper. Like him I think that publication of the discussion of Heberden Society presentations was often rewarding and informative. Perhaps the society could consider this proposal. It would certainly have helped my reply to Dr Kirwan, since I recall disappointingly little discussion when this paper was presented. He implies that that there are several errors, and although I concede this is quite possible I do not know what he is referring to.

His sole criticism relates to the treatment allocation of patients and demonstrates a less than charitable interpretation. No patient was reallocated retrospectively. To reiterate, one man had exceptionally large tophi and was given allopurinol by choice rather than at random. Three patients defaulted from their 2—3-month appointments and stopped taking allopurinol within a short time of being introduced to the study. They were, however, willing to return for annual assessments and, not unreasonably in my view, were evaluated prospectively with the group not receiving hypouricaemic treatment. Exclusion of these 3 subjects from the analysis of GFR estimations does not alter the statistical significance of the downward trend of the mean. As to the clinical significance of our observations, I would refer Dr Kirwan to the rather circumspect discussion included in our paper.

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Another hazard of gold therapy?

SIR, I was interested to read the case report of Fulton and colleagues.[1] There are many similarities between their patient and the one I previously described.[2 3] Both patients developed a metal allergy around the time of onset of their rheumatoid disease, and both reacted adversely, in a similar way, to a test dose of sodium aurothiomalate (Myocrisin). The major difference is that the patient I described failed to show any response on formal patch-testing to nickel, dichromate, or cobalt. Neither, however, did she react to gold leaf or gold chloride. Young[4] has previously commented that patch-testing is often positive to gold salts rather than metallic gold and often positive with one salt and not another.

If, as the authors suggest, the reaction their patient demonstrated was the result of nickel contamination of the aurothiomalate, one would perhaps expect a higher incidence of this seemingly rare complication when one considers that some 10% of females have a history of nickel dermatitis.[5]

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