Correspondence

Acute gout during hypouricaemic therapy: prophylaxis with colchicine

Sir, The efficacy of the uricosuric diuretic agent tienilic acid in the treatment of both gout and hypertension is confirmed in 2 papers.1 2 During our inpatient study of the 4 patients suffered attacks of gout, as is often the case when starting other kinds of hypouricaemic therapy. It is commonly held that the concomitant use of colchicine prevents such attacks, and accordingly we looked into this in a long-term outpatient study. Twenty patients were given tienilic acid alone and 10 patients were given tienilic acid together with colchicine 1 mg b.d. on a non-selective basis. The patients were followed for between 2 and 12 months, with encouraging results in lowering both the blood pressure and serum uric acid, but owing to reports from elsewhere of hepatotoxicity the study had to be abandoned. Of the patients who received tienilic acid alone 15 suffered 27 attacks of gout within 2 months of starting therapy, whereas none of the 10 patients who received colchicine in addition suffered an attack of gout over the same period.

This gives support to the unsubstantiated belief that colchicine prevents the attacks of gout which are seen after starting hypouricaemic therapy.

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References


Standardisation of tests for antinuclear antibody

Sir, The sensitivity of immunofluorescence (IF) tests for antinuclear antibody (ANA) is affected not only by the variables inherent in the IF procedure itself, but also by qualitative variations in the antigen substrate employed and the immunochemical features of antibody detected. Use of an agreed test system, however, even if practicable, would have inhibited many new observations in the field of antinuclear serology and recognition of their clinical associations. The clinical significance of reports issued by routine laboratories has thus usually been determined in the light of local experience. This incompatibility between laboratories leads to difficulty in the interpretation of results obtained on individual patients tested in more than one centre and in the assessment of reported research findings.

As long ago as 1970 the World Health Organisation made available an International Reference Preparation (66/233) for homogeneous 'antinuclear factor' on the basis of an international collaborative study which ascribed to this material a potency value of 100 international units. It was shown that its use as an external standard leads to satisfactory comparability between different laboratories in tests on the same sera. The preparation is available on application in this country to the Director, National Institute for Biological Standards and Control, Holly Hill, Hampstead, London NW3 6RB, and internationally from The Director, WHO International Laboratory for Biological Standards, Statens Seruminstitutt, 80 Amager Boulevard, DK 2300, Copenhagen S, Denmark. Precise instructions for using the material as a potency standard are issued with the preparation.

Thus we should no longer tolerate modes of reporting ANA levels exemplified at a recent Heberden Society meeting where in 3 papers ANA results were communicated as 'positive' or 'negative', or as titres, without reference to the sensitivity of the methodology used. We therefore respectfully suggest that speakers at Society meetings express their ANA findings in international units and further, Sir, that the Annals of the Rheumatic Diseases sets an example by requiring authors of manuscripts reporting ANA results to do the same.

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Gold binding to blood cells

Sir, Van de Stadt and Abbo-Tilstra2 reported the binding of gold to blood cells and serum proteins during auro-thioglucose therapy in patients with rheumatoid arthritis (RA). The authors state that the only other team of investigators to examine blood cell gold distribution in vivo was Ward et al.3 However, Freyberg et al.4 in 1941 found 'all the gold in blood was found to be present in the plasma', and Coke5 was unable to demonstrate gold uptake in circulating leucocytes. In 1961 Lawrence6 administered intramuscular 196Au gold sodium thiomalate to 10 RA patients, 7 of whom were currently receiving chrysotherapy. The red cell gold concentration was approximately one-quarter of the plasma level the day following injection and fell less rapidly than the plasma level. In 1973, using 196Au labelled gold sodium thiomalate, our group7 reported that 40% of 16 RA patients had measurable quantities of gold in the blood cell mass and in isolated red blood cells. This figure corresponds closely to the 45% occurrence of blood cell gold observed by Van de Stadt and Abbo-Tilstra. Other similarities between our data and Van de Stadt and Abbo-Tilstra's include: (1) the quantity of gold bound to the cells (up to 45% and 35%, respectively); (2) the lack of correlation between serum gold concentrations

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and the presence of gold in the cellular mass; and (3) the finding that ABO blood group and rhesus type has no influence on whether gold is bound to erythrocytes.

Taken together these 2 studies, using different highly sensitive and precise techniques to measure gold, confirm that a large proportion of RA patients have gold in blood cells, and that erythrocyte gold binding is similar with aurothioglucose and gold sodium thiomalate.

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References

Sir, We were interested to read the observation of Van de Stadt and Abbo-Tilstra that significant amounts of gold were incorporated into the blood cells of 45°, of all the patients they studied during treatment with gold salts for rheumatoid arthritis. As part of an investigation into the distribution of gold we have made observations on 9 patients who were being treated long term with Myocrismin (sodium aurothiomalate). Blood samples were taken immediately prior to a subsequent administration of the drug, and from these cellular samples were obtained by centrifuging. The gold content of 10 µl samples of whole blood were compared with that of cells from the same volume by neutron activation analysis. The values of the ratio of cell content to whole blood content of gold we found are, in ascending order, 0.02, 0.03, 0.03, 0.05, 0.06, 0.06, 0.17, 0.21, and 0.40. The uncertainty of each ratio is estimated to be about ±0.02.

These results seem to show 2 distinct groups, with one-third of the patients being in the group with the large quantity of gold associated with the cells. This is in good agreement with the observations of Van de Stadt and Abbo-Tilstra. Like them we also find no correlation between high gold content and any clinical feature.

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Conference proceedings wanted
Sir, May I appeal through your columns once again for the following conference proceedings which we still lack in the Heberden Library. I have endeavoured to make a full collection of all the rheumatological International, European, Seapal, and Pan-American congresses since they started, and we still lack the following:

9th International Rheumatological Conference 1957.

European Congress, Wiesbaden 1979 (not yet donated).

Pan-American Congresses—the 5th Congress 1970, Uruguay (Pta d’Este); the 6th Congress 1974, Toron’o; the 7th Congress 1978, Bogata.

Seapal Congresses, we still lack the 3rd Congress proceedings 1976 (Singapore) and those of the 4th (1980) in Manila.

I would be very grateful for gifts of any of these proceedings. The completion of this collection will make a unique reference resource for scholars in this field.

E. G. L. BYWATERS
Honorary Librarian, Heberden Society

Haemophilus influenzae tenosynovitis
Sir, We read with interest the report of Drs Bansal, Magnussen and Napodano describing infectious tenosynovitis caused by *Haemophilus influenzae*. We have recently reported a case of a healthy 51 year old black male with multivascular *Haemophilus influenzae* arthritis associated with tenosynovitis of the dorsum of the hand. We have subsequently observed another patient with *H. influenzae* tenosynovitis, a 39-year-old black male alcoholic with dorsal tenosynovitis of the hand and arthritis involving both knees, a wrist, and a first metatarsal phalangeal joint. Two additional cases of adults with *H. influenzae* arthritis and evidence of tenosynovitis have been reported. In our patients the tenosynovitis has resolved uneventfully over several days of antibiotic therapy, somewhat more rapidly than the associated joint infection. In the light of the published reports and our own experience we agree with Bansal et al. that *Haemophilus influenzae* infection should be considered among the diagnostic possibilities in acute tenosynovitis. Interestingly, although *Haemophilus influenzae* arthritis and *H. influenzae* infections in general are more common in young children, all of the aforementioned cases are adults. We know of no reports of *H. influenzae* tenosynovitis in children.

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Ann Rheum Dis 1980 39: 529-530
doi: 10.1136/ard.39.5.529-c

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