- (ii) An outline of the methods used.
 - (iii) A summary of the results.
 - (iv) A statement of the main conclusions.
- (3) Full references to previous work quoted must be given.
 - (4) Simple tables may be included as long as they are contained within the 'box' on the abstract form. Thus authors wishing to submit abstracts with tables may utilize two 'boxes' if necessary, i.e. one for the abstract and one for the tables.
 - (5) Accepted abstracts will be published as such with the proceedings of the Society in the *Annals of the Rheumatic Diseases*, so must be received in a form suitable for publication. In particular, statements such as "The data will be discussed" are entirely unacceptable. Abstracts may be revised for publication after the meeting.
 - (6) Abstracts should be sent to the Senior Honorary Secretary, The Heberden Society, c/o ARC, 8/10 Charing Cross Road, London WC2 0HN, and must be received not less than two months before the meeting at which it is desired to read the paper.
 - (7) When submitting abstracts, authors must state whether the communication has been or is about to be read at another meeting; or has been or is about to be published.
 - (8) The normal length of time for presentation of papers is 10 minutes. However if authors would prefer a longer (15 minutes) or shorter (5 minutes) time, this should be stated when submitting abstracts. The final allocation of time will however rest with the Executive Committee.
 - (9) Authors will be notified when an abstract is received and when it is either accepted for the following meeting or rejected. Abstracts which are acceptable but which cannot be included in the following meeting will be returned to authors, who may resubmit them for a subsequent meeting if they wish.
 - (10) It is the author's responsibility to keep the Senior Hon. Secretary informed of the date of publication of any paper submitted to the Society.

Clinical meeting

The following papers were presented at the Annual General Meeting on November 28 and 29, 1975.

Clinical study of Behçet's disease in the County of Yorkshire. M. A. Chamberlain (*University of Leeds*)

Over the course of 2 years from 1972 to 1974 all patients with established Behçet's disease, and all new cases residing in Yorkshire were personally surveyed and a family study also undertaken. The clinical, radiological, and laboratory findings in the 32 (12 male, 20 female) patients considered by referring physicians to have the disease were presented.

32 patients had mouth ulcers. Of these, 29 also had genital ulcers (there were no patients with genital ulceration without mouth ulcers). It was usually possible

to differentiate the mouth ulceration of Behçet's disease from aphthous ulceration in the general population; ulcers were painful and extended characteristically into the fauces and even into the oesophagus. Swallowing was difficult, soft foods only were eaten. Ulcers occurred many times per annum and in severe cases were rarely absent. Steroids ameliorated genital ulceration in 4 patients and contraceptives helped in one. Genital ulceration itself was found to be responsible for marital discord and even suicide attempts.

Arthritis occurred in 19 patients and usually affected the knees and ankles. Evidence of permanent radiological damage was found in the hands of only 2 subjects and in the feet of one of these, and in both cases was due to osteoarthritis. Psoriasis and psoriatic arthritis were not found in patients in this series, nor was there any radiological evidence of sacroiliitis. Acne, maculopapular rashes, and styes were reported and erythema nodosum was found in 7 subjects. Iritis was seen in 4 male patients. Phlebitis was found in 7 patients and severe neurological changes in one patient.

Patients were typed for HLA antigens. HL-A 5 was present in 4 patients, HL-A 27 in 6, W 5 was absent in all, and W 18 was found in one. Up to 71 features were analysed by computer for each patient and correlated feature-to-feature. Probandes were similarly correlated according to the common characteristics they exhibited.

Adjuvant arthritis in the rat. Distribution of fluorescent material after footpad injection of rhodamine-labelled tubercle bacilli. B. Vernon-Roberts, S. P. Liyanage, and H. L. F. Currey (*Bone and Joint Research Unit, London Hospital*). Published in the *Annals*, 1976, 35, 389.

Prostaglandin production in inflammatory joint disease. M. A. Bray and D. Gordon (*Laboratory of Applied Physiology, Kennedy Institute of Rheumatology, London*)

Raised prostaglandin (PG) levels have been reported in synovial effusions from patients with rheumatoid arthritis (Levine, 1973; Patrono *et al.*, 1975) and from experimental animals in response to the injection of inflammatory stimuli (Blackham *et al.*, 1974; Glatt *et al.*, 1974). Human rheumatoid synovial fragments produce substantial amounts of PGs during *in vitro* culture (Robinson *et al.*, 1973). The observations that nonsteroidal anti-inflammatory drugs (NSAIDs) are potent inhibitors of PG biosynthesis (Vane, 1971) and that this activity correlates with their anti-inflammatory potency (Flower *et al.*, 1972) suggest a likely involvement of PGs in diseases such as gout and rheumatoid arthritis. The cellular origin of such PGs remains unestablished. It has been suggested that the neutrophil might provide a source of PGs in inflammation (Higgs *et al.*, 1975; McCall and Youten, 1975) and we have reported that the activated macrophage possesses a marked capacity for PGE production (Bray *et al.*, 1974). We compared the efficacy of guinea pig peritoneal exudate macrophages and neutrophils in producing PGs and assessed the activity of some anti-inflammatory drugs on production by macrophages.

Starch-induced peritoneal exudate cells (4 hours for neutrophils, 2-3 days for macrophages) from Hartley guinea pigs were incubated at 1×10^6 viable cells/ml for 24 hours at 37°C. PG generation by these cells was

measured by radioimmunoassay using sheep anti-PGE₂-BSA. Drugs were added immediately before incubation using the following concentration ranges: indomethacin (0.1 ng—10.0 µg/ml); aspirin (1 ng—100 µg/ml); phenylbutazone (1 ng—100 µg/ml); feprazone (1 ng—100 µg/ml), and naproxen (1 ng—100 µg/ml); colchicine (1 ng—10 µg/ml); dexamethasone (1 ng—10 µg/ml); prednisolone (1 ng—10 µg/ml), and dehydrocortisone (1 ng—10 µg/ml).

Macrophages produce substantial amounts of PGE (mean 11.6, range 2.4—43.2 ng, PGE₂ equivalent/10⁶ cells/24 h) whereas neutrophils produce much less (mean 0.4, range 0.2—0.62 ng, PGE₂ equivalent/10⁶ cells/24 h) measured by radioimmunoassay. The production of E-type PG by macrophages is inhibited by NSAIDs with a rank order of potency, on a weight basis, of indomethacin > feprazone > aspirin > phenylbutazone > naproxen > sodium salicylate. Anti-inflammatory glucocorticosteroids also inhibited macrophage PGE synthesis with a relative potency of dexamethasone > prednisolone > hydrocortisone. Colchicine, however, stimulated PGE production by the macrophages.

Whereas human rheumatoid synovial fragments produce substantial amounts of PGs and show comparable susceptibility to anti-inflammatory drugs as exhibited by guinea pig peritoneal exudate macrophages, cells present in human synovial effusions (predominantly polymorphonuclear (PMN) leucocytes) are poor effective sources of PGs *in vitro*. It is noteworthy that there is no correlation between the levels of PG and the PMN leucocyte count in synovial effusions (Patrino *et al.*, 1975) and that the appearance of PGs in urate crystal induced synovitis precedes PMN leucocyte infiltration (Glatt *et al.*, 1974); also that human peripheral blood PMN leucocytes are poorly active in generating PGs (Zurier, 1975). Both the NSAIDs and the steroidal anti-inflammatory drugs showed an efficacy in blocking PG production which broadly paralleled their clinical effectiveness in the former case and closely followed the anti-inflammatory activity in the latter. The stimulation of PG production by colchicine has also been noted in human rheumatoid synovial cultures (Levine, 1973) and during urate crystal induced synovitis (Glatt *et al.*, 1974). These observations suggest that macrophages are the most likely source of PGs in inflammatory joint disease, and indicate that the guinea pig peritoneal exudate macrophage may be an appropriate model for the evaluation of anti-inflammatory drugs.

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Selective concentration and localization of gold in macrophages of synovial and other tissues during and after chrysotherapy in rheumatoid patients. B. Vernon-Roberts*, J. L. Doré*, J. D. Jessop†, and W. J. Henderson†. (*Bone and Joint Research Unit, The London Hospital**; *The University Hospital of Wales, Cardiff.†*) Published in full in the *Annals*, 1976, **35**, 477.

Changes in collagen of synovial membrane in rheumatoid disease. C. R. Lovell, M. I. V. Jayson, and A. J. Bailey (*Department of Medicine, University of Bristol, and Agricultural Research Council, Langford, Bristol*)

Gross thickening of the synovium is a principal characteristic of rheumatoid disease. However, knowledge of the structure of synovial collagen and the changes in disease is limited.

Rheumatoid synovia were obtained at surgery from 10 cases of age range 40–70 years. Control specimens, matched for age and site, were obtained from autopsies of patients who had no evidence of rheumatic disease. We analysed the synovial membrane for the genetic type of collagen and the nature of the intermolecular crosslinks by reduction with tritiated borohydride (Bailey *et al.*, 1970). The rates of collagen biosynthesis were determined from the formation of nondialysable ³H-hydroxyproline after incubation of the tissue in ³H-proline labelled culture media (Herbert *et al.*, 1974). Fractional precipitation of the pepsin-solubilized collagen as described by Chung and Miller (1974) showed the presence of polymorphic forms, and these were identified as type III (60%) and type I (40%) collagens in both normal and rheumatoid synovia.

Analysis for the presence of borohydride reducible crosslinks showed both dihydroxylysinoxidation and monohydroxylysinoxidation. A high proportion of the latter crosslink was present in young tissue, but failed to decrease during maturation. However, analysis of synovium from rheumatoid patients in the older age group showed the presence of a high proportion of dihydroxylysinoxidation, indicating increased collagen synthesis. In contrast to the crosslink found in scleroderma skin this crosslink is not cleaved by D-penicillamine and tissue culture studies indicate that D-penicillamine fails to inhibit the increased collagen synthesis of rheumatoid synovium.

In summary, the type of collagen proliferated in rheumatoid synovium appears to be similar to normal but possesses a more stable crosslink that is resistant to D-penicillamine. Furthermore D-penicillamine therapy does not inhibit the rate of formation of this new collagen.

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Friction and lubrication of artificial hip joints. A. Usworth (*Leeds*)

The lubrication of artificial hip joints has been recognized as being important for some time. Not only would any full fluid film lubrication help to reduce friction, but it would also reduce the wear rate of such prostheses. Experiments were done on different types of prostheses, namely metal on plastic and metal on metal, to determine