preparing the smear does not allow the enzyme to work well.

DR. A. J. PALFREY (*London*) It seems to me that Dr. Williamson has shown very convincingly that two types of fibre are present in these fluids, and that one of them looks like collagen. A logical conclusion is that there is a turnover in the fibres of the cartilage. These fibres may therefore be present either as newly formed or as damaged collagen fibres: both these possibilities have to be considered. Their unsatisfactory enzymatic behaviour fits in very well with these possibilities. The fibrinoid fibres may be associated with the presence of damaged collagen fibres, but I think this is more likely to be a result of the ACTH treatment. Fibrinoid fibres in synovial tissues commonly stick to the surfaces of all types of cells.

DR. WILLIAMSON I would not argue that the pictures of a cell eating material are not very convincing. I merely thought that this was a possibility, but obviously it could be surface adherence.

PROF. E. G. L. BYWATERS (*Hammersmith*) Have you measured the protein content or the fibrinogen of these fluids, especially of the transudates described?

DR. WILLIAMSON I have not performed quantitative estimations. We have detected fibrinogen qualitatively by gel diffusion in all these fluids with the exception of two of those induced by ACTH in cases of ulcerative colitis. The two normal subjects were not tested since very little fluid was obtained, but it is said that fibrinogen is not present in normal joint fluid. It seems that fibrin is present in fluids from patients with ulcerative colitis receiving ACTH; whether this represents fibrils which have been broken down and are detected as free fibrinogen, or free fibrin, I am not sure. I treated some of these again with streptokinase to see if one could find breakdown products. There was so little present that the gel diffusion estimations were not convincing. We do not have any figures on the total protein content of the fluids.

DR. H. L. F. CURREY (*London*) Have you had an opportunity of examining these fibrils by polarized light microscopy? I examine fluids without anticoagulant and I feel that I can differentiate between collagen and fibrin fibres.

DR. WILLIAMSON All our fluids are examined by polarizing microscopy to exclude crystals, and I was not convinced that one could make a distinction. I considered that they did not show birefringence, but this may be a matter of degree.

DR. H. L. F. CURREY (*London*) I think collagen fibres are clearly birefringent and the fibrin as well if you have a source of light sufficient to show them.

DR. WILLIAMSON I think the problem is that these are very fine fibrils and are probably coated with other proteins. This is something we could investigate further.

DR. D. J. WARD (*Oswestry*) We had a patient with ankylosing spondylitis who was started on ACTH and within 2 days developed a simple knee effusion—the knee was certainly not hot. There were less than 1,000 white cells per cu. mm. and the protein content was 5 g./100 ml. Perhaps these effusions are inflammatory after all.

DR. WILLIAMSON We did estimate the hyaluronate in these fluids and, except that they gave the impression of a diluted fluid, they were normal.

The Cracking of Joints—A Bioengineering Study. By V. WRIGHT, A. UNSWORTH, and D. DOWSON (*Leeds*). This paper with the discussion thereon is to appear in the July, 1971, issue of the *Annals*, vol. 30.

Synovial Fluid Waaler-Rose and Latex Tests. By E. C. HUSKISSON, F. DUDLEY HART, and B. W. LACEY (*Westminster Hospital*). This paper was printed in full in the January issue of the *Annals* (1971, 30, 67).

Discussion

DR. A. G. S. HILL (*Stoke Mandeville*) I think your false positives are a difficult group, because many of these conditions could conceal rheumatoid arthritis. Any polyarticular osteoarthritis may conceal a polyarthritis. What is the present concept at the Westminster Hospital of palindromic rheumatism and its relationship to rheumatoid arthritis?

DR. HUSKISSON We believe that many patients will ultimately develop classical rheumatoid arthritis. We were therefore interested to find a positive Waaler-Rose test in the synovial fluid of a patient with palindromic rheumatism, but four patients subsequently tested gave negative results. A titre of $\geq 1:16$ was taken as positive throughout the study.

DR. J. A. BOYLE (*Glasgow*). Could you give us some statistical confidence in the index of discrimination? For example, if you repeated the study, do you think you would still find that a Waaler-Rose titre of 1:8 was a better discriminant between rheumatoid arthritis and non-rheumatoid arthritis than a conventional titre?

DR. HUSKISSON This is what we were trying to find out. This was a retrospective study designed to examine the usefulness of synovial fluid tests and to get some idea of the best titres to take. We chose our index of discrimination because it was simple, the value representing the percentage of patients in our series who were correctly classified by any criterion. Our patients were to some extent selected and the optimum levels might not be reproducible. The results will, however, form the basis for a prospective study.

DR. J. A. MATHEWS (*London*) Some years ago I tried to break up white cells in joint effusions by two techniques, either freeze-thawing or ultra-sound, to see whether rheumatoid factor was released, so increasing the number of positive tests, and I failed. I wonder whether you have tried this?

DR. B. M. ANSELL (*Taplow*) Dr. Huskisson, you dismissed the method very readily. I want to comment particularly on this because of some work that we have been doing at Taplow over some years. About 5 years ago I presented some work on the DAT and the tube latex tests in synovial fluid in Still's disease, in which we found relatively few positive tube latex results. Subsequently, David Champion has been working with a slide latex test and has found that this correlates much better with the synovial fluid DAT and the blood latex than the tube latex test. We have not yet worked out why, Dr. Holborow thinks that it may be something to do with immune complex development in the synovial fluid. I would therefore like to ask your method. Secondly, there is the question of pre-treatment of the synovial fluid. This is relevant because the technician working with us found, particularly in the viscous fluids, that all sorts of queer things happened if he did not pre-treat them with hyaluronidase. Our findings, of course, are at complete variance with yours because we have had only one positive latex in a synovial fluid using the slide method in a patient whom we did not consider to have rheumatoid arthritis.

DR. HUSKISSON I should be pleased to supply full details of our slide latex test. We found that the latex test gave false positive results less commonly than the Waaler-Rose test. I agree that it is necessary to use hyaluronidase for very viscous fluids but we avoided it because it has been shown to increase the incidence of positive tests.

DR. A. ST. J. DIXON (*Bath*) Results such as these are of considerable interest for the criteria for diagnosis of rheumatoid arthritis and their international standardization. Dr. Rotes-Quérol in Barcelona has suggested that joint fluid diagnostic for rheumatoid arthritis may show the combination of a positive Waaler-Rose test plus ragocytes.

DR. W. W. BUCHANAN (*Glasgow*) We have looked for ragocytes in synovial fluids in a large number of different joint diseases, and found them to lack diagnostic specificity. Inflammatory joint fluid contains many dead or dying polymorphonuclear leucocytes, and these will appear as ragocytes when examined by conventional staining methods. The polymorphonuclear leucocytes in chronic bronchitic sputum also appear as ragocytes.

Synovial Rupture, Experiments on Cadaveric Knees. By R. ASTLEY COWPER, M. I. V. JAYSON, and A. ST. J. DIXON (*Royal National Hospital, Bath, and University of Bristol*). This paper and the discussion thereon were published in the March issue of the *Annals* (1971, 30, 162).

Microradiographic Aspects of Articular Cartilage Ageing. By A. DHEM (*Department of Anatomy, University of Louvain, Belgium*).

This study was performed on the lower end of the human tibia at the ankle joint; 84 pieces were taken at autopsy from patients aged between 16 and 96 years, who had died from acute illness or after trauma. Undecalcified sagittal sections, embedded in methyl methacrylate, were submitted to microradiographic analysis. This

technique revealed clefts, or microfissures, in the calcified layer of cartilage only in subjects more than 50 years old. These clefts are limited by the tidemark (the interface between the hyaline and calcified cartilage), and extend to a variable depth into the subchondral bone plate; some are filled with hypermineralized material. The same features were found in decalcified paraffin embedded sections.

These observations suggest that ageing of joints is a specific phenomenon different from arthrosis.

Discussion

DR. D. L. GARDNER (*Kennedy Institute*) It would be important to know whether these fissures which you have demonstrated so clearly are real phenomena; are they present during life, or are they a reproducible result of a constant artefactual biophysical change in the matrix of the cartilage or bone? There is some evidence in other species (*e.g.* turkey) that, even in young creatures, a series of splits in cartilage can be observed in preparations like this. One thinks they are present because of a change in the biophysical structure during section preparation, the change taking place at a constant zone.

DR. DHEM No, we never find these changes before the age of 50 years, and in only half the specimens from subjects aged 50 to 60 years. We have not taken account of fissures which appear black under microradiography, but only of those in which the fissure is calcified. I have found the same changes in the hip joint but I have studied only five specimens.

Hearing in Rheumatoid Arthritis: Results of Audiometry in 76 Patients. By C. J. GOODWILL, I. J. LORD, and R. P. KNILL-JONES (*King's College Hospital*).

Copeman (1963) reported three patients with rheumatoid arthritis in whom increased activity of the arthritis was associated with deafness; in two the deafness was of conductive type. The incudo-stapedial and incudo-malleolar joints are synovial joints and so presumably could be involved in the rheumatoid process.

An unselected series of 76 patients with classical or definite rheumatoid arthritis have been examined by pure air-conduction and bone-conduction audiometry, salicylates being discontinued 3 days before. Patients with other possible causes for deafness, such as chronic otitis media, Menière's disease, and acoustic trauma, were excluded.

The activity of the arthritis was assessed using the Systemic and Articular Indices of Lansbury (1956). The duration of the arthritis, Waaler-Rose titre, and presence of nodules were noted, and the functional activity was graded 1 to 5.

No patient was found with conductive deafness sufficiently severe to merit exploratory tympanotomy, although minor degrees of deafness were found; no patient complained of deafness, and none had a negative Rinne test. Two patients had a single 'dead ear' preceding the rheumatoid arthritis by many years; these two patients were excluded, leaving 148 ears for analysis.

The mean hearing loss was not related to the duration of the arthritis or to the Systemic or Articular Indices.