in Copenhagen (Suejaard, Staub Nielsen, Ryder, Kissmeyer-Neilsen, Sandberg, Lindholm, and Thorsby, 1972) as may be associated with a new segregant series at the second locus closely associated with the second locus antigens, and this does raise the possibility that the HL-A picture may not be quite as straightforward as we thought. We have also looked at patients with Still's disease and we found that 24% of these had W 27 and 6 out of the 7 patients involved were male. All these patients had sacroiliitis, so it does seem to suggest that HL-A 27 may be a marker of sacroiliitis rather than associated with any particular disease syndrome.

DR. BREWERTON I agree that the serological findings are complex, and that it is not only a question of the presence of HL-A 27. It may be wrong to regard HL-A 27 as a marker of sacroiliitis. We have seen several adults with peripheral arthropathy alone, or acute anterior uveitis without evidence of any rheumatic disease. HL-A 27 may prove not to be relatively specific for the spine and sacroiliac joints.

DR. A. ST. J. DIXON (Bath) These clinical manifestations do not all appear at once. A follow-up may give you further information.

DR. A. M. DENMAN (Northwick Park) I wonder if you have tried incubating lymphocytes which lack this antigen with serum from patients with this group of diseases, perhaps after treating the cells with papain or other enzymes and then retyped them to exclude the rather remote possibility that this is a serum antigen which is picked up by circulating lymphocytes?

DR. BREWERTON We have not done this experiment, but I hope we will.

PROF. V. WRIGHT (Leeds) It is a very interesting and good study. I missed the spouse figures for your various groups. Secondly would you like to comment further about Reiter's disease because that is the one thing that does not fit too well into the story. Is your figure of over 50% positives in patients with long standing or acute Reiter's syndrome? The third question is to ask if you have any data on colitic arthritis without sacroiliitis?

DR. BREWERTON We have not studied the spouses specifically. This will be done when we conduct a more thorough family study. In our article on Reiter's disease in the *Lancet* and in subsequent figures, we divided those with peripheral arthropathy alone into those seen during their initial acute attacks, those that had had the disease for less than 5 years, and those who had had it for more than 5 years (with a mean of 13 years). The proportion in each group who had HL-A 27 was the same. This seemed to apply whether the duration of disease was 2 weeks or 25 years. We are studying colitic arthritis without sacroiliitis, but we have not yet seen enough to present the results.

DR. T. R. LITTLER (Liverpool) One point I would like to make is that I occasionally find some difficulty in making up my mind between an incomplete Reiter's disease clinically and the psoriatic with sacroiliitis and arthropathy. Am I correct in assuming that if your Reiter's and

ankylosing spondylitis are HL-A 27 positive and psoriatics are HL-A 18 positive would this new test help from the clinical point of view in differentiating between the two when these occasional problems of differential diagnosis arise?

DR. BREWERTON I cannot give a direct answer because we have excluded from our investigations patients with these overlap phenomena. At present this test is complicated, and I doubt whether it will prove to be a practical way of distinguishing Reiter's disease and psoriatic arthropathy. Anyway, I suspect that we may soon be thinking in terms of a series of underlying processes, overlapping in different proportions, rather than psoriasis with spondylitis and Reiter's disease with spondylitis as two completely separate entities.

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Lymphocyte Subpopulations in Rheumatoid Arthritis and Systemic Lupus Erythematosus. By M. POPOVIC, J. MUNRO, P. DAVIES, and P. J. L. HOLT (Royal Postgraduate Medical School, London)

Direct immunofluorescence and autoradiography for surface antigen receptors on lymphocytes and spontaneous rosette formation using untreated sheep red cells have been used to detect subpopulations of 'B' and 'T' lymphocytes, respectively, in peripheral blood. 12 patients with systemic lupus erythematosus, 22 rheumatoid arthritis, and 11 osteoarthrotic patients have been examined. Other tests of lymphocyte function such as transformation with phytohaemagglutinin and 'spontaneous' transformation have been performed and the results compared with clinical status and following treatment.

Increased proportions of 'B' cells were only found in rheumatoid arthritis 35% (range 21–53%), the controls' levels being 24% (16–34%). A decrease in 'T' cells was found in both rheumatoid arthritis, mean 19% (range 8–34%) and systemic lupus erythematosus, mean 10% (range 3–32%), our normal values being 32% (range 7–42%). All these results were highly significant ($P < 0.001$).

Rosette formation of normal lymphocytes was shown to be impaired by sera from patients with systemic lupus erythematosus, this impairment being reversed when the systemic lupus erythematosus sera was replaced with normal human sera. Both increased spontaneous transformation and impaired rosette formation were related to disease activity and, using DNA antibodies as a marker, returned to normal with successful treatment.

Discussion

DR. K. WHALEY (*Glasgow*) The low percentage of 'T' cells using your rosette test is extremely interesting; this is far lower than most people report. I wonder whether there are technical variations between your technique and that of other workers. Two things spring to mind; one, how do you define the rosette, perhaps you have more stringent criteria than most other people, and secondly at what temperature do you perform the assay?

DR. HOLT The rosette has to have 5 red cells on it to be a positive and the assay is performed at 4°C, but the preliminary incubation with sera is at 37°C.

DR. K. WHALEY (*Glasgow*) Quite. One of the problems at 37°C is that rosettes are very unstable and tend to break-down very quickly. Therefore, most people, including yourself, perform the test at 4°C. The other point of 5 red cells being necessary before one can definitely say a rosette is present is more than most people accept. This variation could account for your low results.

DR. HOLT We are aware of this point. Our interest was to find a level at which we could show the change. Because by using one method you can push the results for 'B' and 'T' cells up to 100%, it does not necessarily mean it is the ideal way of doing the test, nor that it represents normality.

DR. K. WHALEY (*Glasgow*) Yes, I agree; most people push it up to 60% but it may in fact improve your results. There may be bigger differences.

DR. HOLT If you push it like that, I think you would probably improve the ability of the rosettes to stick together and you do not get this variability coming out quite so easily.

DR. W. C. DICK (*Glasgow*) Have you completely excluded by studies both *in vitro* and *in vivo* that these as yet undefined factors which are albumin bound and of small molecular weight could not be drugs that the patients are receiving?

DR. HOLT I did not say they were albumin bound, I said they were of similar size. I think we can exclude the effect of drugs. We find similar effects in, say, pregnancy where there are no drugs being given, and we have dialysed the serum and after dialysis it is still the same. Further, it is rapidly reversible, which is a little against drug effect. I would say we have gone a reasonable way towards excluding this possibility.

DR. P. A. BACON (*Bath*) A couple of years ago I presented some evidence that in both RA and SLE there were large lymphocytes in the blood which in autoradiography were shown to be spontaneously transformed cells (Bacon, Crowther, and Sewell, 1971). Do these cells in fact rosette or do they contribute to your low number of peripheral 'T' cells?

DR. HOLT The labelled cells do not form rosettes.

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Comparison of ultrasound and positive contrast arthrography in the diagnosis of popliteal and calf swellings. By H. MEIRE, D. J. LINDSAY, D. R. SWINSON, and E. B. D. HAMILTON (*King's College Hospital, London*)
Published in full in the *Annals* 1974, 33, 221.

Discussion

DR. J. M. GUMPEL (*Northwick Park*) At Northwick Park I have had the privilege of working with radiologists who are very interested in soft tissue shadows and have been surprised how often on a plain lateral radiograph of the knee they have predicted that I will have found or should have found a Baker's cyst. Have you compared your findings with plain x-rays of the knees, to assess whether you could have seen a Baker's cyst?

DR. SWINSON We have not compared it with soft tissue x-rays. We have used it in patients with suspected rupture of the cyst and have found it useful, though these patients were not in the results.

DR. M. I. V. JAYSON (*Bristol and Bath*) I think that one should point out that when we (Genovese, Jayson, and Dixon, 1972) examined a series of rheumatoid knees routinely by arthrography we found that over 90% of them had popliteal cysts, so that I think that you are quite likely to find them in any series in which you are looking for them. I just wonder how much this technique depends upon the actual pressure within the cyst? With a low pressure cyst, which is difficult to detect clinically, might you find it difficult to detect it by ultrasonic scan because you do not have a nice tense surface under pressure to reflect the ultrasonic beam?

DR. SWINSON I think it depends principally on the size of the cyst. As long as it has fluid in it you have an area of different density to the structure around it and therefore it should show up if it is a reasonable size, say 2 cm.

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Atlanto-axial Subluxation—a 5-year Follow-up Study in Rheumatoid Arthritis. By J. A. MATHEWS (St. Thomas's Hospital, London) To be published in full in the *Annals*.

Rheumatoid Discitis in the Thoracic Region due to Spread from Costovertebral Joints. By E. G. L. BYWATERS (*M.R.C. Rheumatism Unit, Taplow and Royal Postgraduate Medical School, London*)

A detailed dissection of the rheumatoid spine shows lesions in thoracic discs due to spread from adjacent costovertebral (diarthrodial) joints. This has not hitherto been recorded clinically, radiologically, or pathologically. The process is essentially similar to that shown by Ball (1964) for the cervical discs where there is spread from the oncovertebral joints. Lesions in the lumbar spine previously described radiologically (Lawrence and Sharp, 1964) can be seen to be due to rheumatoid invasion of a degenerate disc.

Discussion

DR. J. BALL (*Manchester*) I would like to congratulate Prof. Bywaters on an excellent illustration of the thoracic lesions. I can certainly confirm that arthritis of costo-