evidence of vasculitis, as shown, for instance, in the fingers, because if there was a correlation here, arterial obliteration might well explain a decrease in renal function.

DR. BURRY This is an important omission. We did not see any nail-fold lesions in the whole series of 97 cases, nor evidence of arteritis in the renal biopsies.

DR. K. WHALEY (*Glasgow*) Did you screen your patients for keratoconjunctivitis sicca? Sjögren's syndrome is occasionally complicated by glomerulonephritis, and it would be interesting to know whether the diminished creatinine clearance is related to keratoconjunctivitis sicca.

DR. BURRY There were no cases of Sjögren's syndrome.

Cellular Immune Mechanisms in Rheumatoid Arthritis and Sjögren's Syndrome. By K. WHALEY, A. C. A. GLEN, S. D. DEODHAR, W. C. DICK, G. NUKI, and W. W. BUCHANAN (*Glasgow*).

The immune responses to 2, 4-dinitrochlorobenzene (DNCB) and old tuberculin (OT) were studied in four groups of patients in addition to controls: sicca syndrome, Sjögren's syndrome complicated by rheumatoid arthritis, and seropositive and seronegative rheumatoid arthritis. Patients with seropositive rheumatoid arthritis and patients with rheumatoid arthritis complicated by Sjögren's syndrome demonstrated a diminished ability to become sensitized to DNCB, whereas normal responses were found in the other two groups. Testing with OT, on the other hand, revealed normal responses in patients in all groups except the sicca syndrome group, which showed diminished sensitivity. Analysis of the RNA/DNA content of the peripheral blood lymphocytes revealed elevated values in those groups of patients with diminished DNCB responsiveness, whereas normal ratios were found in the other clinical groups. The mean diameter of the peripheral lymphocytes was closely related to the RNA/DNA ratio, which suggests that patients having elevated RNA/DNA ratios have a higher proportion of circulating lymphocytes of the large type, presumably lymphocytes committed in response to some antigenic stimulus. It is concluded that cutaneous anergy to DNCB may be due to a functional depletion of thymic dependent lymphocytes.

Discussion

PROF. K. W. WALTON (*Birmingham*) It has been shown in one or two other conditions with widespread involvement of the reticuloendothelial system that there may be some relation between the severity of the disease process and the lack of response to cellular immunity. I think it was Waldorf, Sheagren, Trautman, and Block (1966) who showed a difference between lepromatous and tuberculoid leprosy in the response to DNCB. In Hodgkin's disease, which may fluctuate, it has been found that the degree of response depends very much on disease activity. Have you had the opportunity in your cases, some of whom presumably will have waxed and waned, to see whether there was any difference in their DNCB response in relation to the disease?

DR. WHALEY No, we have not. We thought it would be adequate to study the disease parameters in the entire

group to determine correlations in those that were DNCB-positive and DNCB-negative because there was a spectrum of disease activity at the time of sensitization. The mechanism of anergy to DNCB in lepromatous leprosy and Hodgkin's disease is probably different. Patients with lepromatous leprosy show depletion of the thymic-dependent areas of the lymph node caused by the disease and this is probably why they do not respond to DNCB. It is not known whether treatment with Dapsone will reveal sensitization to DNCB, but I think it has been shown that the thymic-dependent areas become repopulated with small lymphocytes once the disease has been treated.

DR. P. J. L. HOLT (*Hammersmith*) First of all, you have not necessarily shown the dependence on thymic lymphocytes. After all there are several steps between antigen challenge and skin response. The first step would be macrophage digestion of the antigen and it has been shown that chemotaxis and phagocytosis is reduced in the rheumatoid subject, so the skin concentration of DNCB may have to be higher or maintained longer to see whether sensitized lymphocytes have been produced; in other words there is transformation in the circulating blood. Perhaps the last, and in some ways the most interesting, question is whether there are lymphocytes capable of expressing the skin sensitivity as measured by Mantoux type reactions. These may all be different defects, and I think they must be shown independently. You said that there was a diminished incidence of A2 isohaemagglutinin. We have been studying another condition with diminished skin expression to migration inhibition factor, all patients are group O with one exception who is A₂. Finally, returning to lepromatous leprosy, there is a genetic basis for the lack of skin expression in the leprin response.

DR. WHALEY Do you think the genetic basis depends on depletion of the thymic dependent areas in lepromatous leprosy?

DR. P. J. L. HOLT (*Hammersmith*) That I am not capable of answering. You were judging a chain of events by one end-factor without saying at which level this chain went wrong.

DR. WHALEY We used the DNCB and Mantoux tests because we believe the latter tests the ability of the memory cell to mount a delayed hypersensitivity reaction in response to OT and the efferent part of the arc. I agree that study of macrophage function in these patients should be tested.

DR. P. J. L. HOLT (*Hammersmith*) The thymic cell does not necessarily express skin sensitivity. There may be another cell that is conditioned by the thymic cell.

DR. WHALEY Agreed.

DR. P. A. BACON (*St. Bartholomew's Hospital*) We have been looking at peripheral lymphocytes in rheumatoid arthritis. In about 60 per cent. of rheumatoid subjects we have found large circulating lymphocytes which are similar to those seen in Hodgkin's disease, are metabolically active, and look like lymphoblasts. The presence of

these does not correlate closely with ability to sensitize to DNCB nor with depletion of small lymphocytes.

References

WALDORF, D. S., SHEAGREN, J. N., TRAUTMAN, J. R., and BLOCK, J. B. (1966) *Lancet*, 2, 773

The Choroid Plexus in Systemic Lupus Erythematosus. By C. J. ATKINS, J. KONDON, F. QUISMORIO, and G. FRIOU (*University of Southern California, Los Angeles*).

Autopsy studies were carried out in six patients with systemic lupus erythematosus in whom there were overt clinical features of cerebral disease. When the brains were examined by conventional as well as immunofluorescent techniques, no distinctive changes were noted.

It was then realized that the choroid plexus was a filter and that as in lupus nephritis circulating immune complexes might also be deposited here.

In the two patients studied, gammaglobulin deposits were demonstrated in the choroid plexus using the direct immunofluorescent technique. These deposits were decreased by elution at pH 3.2 and by treatment with DNAase. Electron microscopy in one case revealed dense deposits in the connective tissue of the choroidal villi similar to those described in the glomeruli of lupus nephritis.

The absence of significant perivascular inflammation and gammaglobulin deposition about the nutrient vessels is contrary to the prevalent view that the central nervous system symptomatology in systemic lupus erythematosus

is the result of cerebral vasculitis. These observations focus attention on the possible role of the choroid plexus in the pathogenesis of the nervous and mental manifestations of systemic lupus erythematosus.

Discussion

DR. K. WHALEY (*Glasgow*) Have you noted any correlation between the findings in the choroid plexus and in the kidney, and whether you found free DNA or anti-DNA antibody in the serum of patients with choroid plexus immune complexes?

DR. ATKINS We found anti-DNA in high titre in both patients with choroid plexus complexes. Both kidneys were conventionally stained but in the one examined by immunofluorescence we found both IgG and IgM deposits.

DR. K. WHALEY (*Glasgow*) Have you examined the subarachnoid granulations which remove the cerebrospinal fluid?

DR. ATKINS No. This obviously must be done.

DR. K. WHALEY (*Glasgow*) Have you measured the cerebrospinal fluid pressure?

DR. ATKINS No. This finding reveals many further problems.

Book review

First and Last Experiments in Muscle Mechanics. Edited by A. V. HILL. 1970. Pp. 141, 62 figs. Cambridge University Press, London (£2.75)

In the study of muscle physiology A. V. Hill stands out as one of the giants, and his account of his work in this field is therefore of immense interest, both for its scientific content and as a personal testament to a great scientist.

As is only to be expected, the standard of production and presentation in this slim volume is high.

It will probably be of no more than academic interest to the average rheumatologist, but it provides a valuable study in depth for those practitioners whose work involves a therapeutic application of muscle exercise.

J. G. P. WILLIAMS

Notes

Report of the British Branch of the Ligue Européenne contre le Rhumatisme

From the Delegate, E. G. L. Bywaters, November 21, 1970

The Heberden Society has represented British rheumatology on the world front since 1927 when the International Ligue was born. Because, however, of the Heberden Society's limited and selective membership, with then only 100 full members entitled to vote, the British Branch of the Ligue was started in 1947 and included all members, full and associate, of the Heberden Society as well as others

who joined directly. All that was needed for membership of the British Branch was an application and a fee of one guinea, later 5s. and recently £1. There have been no proposers or seconders, no selection, no criteria, and no exclusions. Up to a week or so ago, out of 238 members, only eight were not full or associate members of the Heberden Society. In the last week a further 33 members have been enrolled.