Book Reviews

The Biology of Degenerative Joint Disease. By LEON SOKOLOFF. 1969. Pp. 162, 13 figs. University of Chicago Press, London. (59s.)

This is a delightful, discursive, and well-documented series of essays reviewing various aspects of the cytopathology and cyto-physiology of osteoarthritis. Since the author's own work lies primarily in the field of experimental and comparative pathology, these aspects of degenerative joint disease, which in the past have been rather neglected, are particularly well treated. The detailed analyses of other aspects of arthritis, such as disturbances in cartilage, collagen, and ground substance, are very well written. Again, the chapters on "biomechanics" and the role of "systematic factors" are likely, by reason of their practical implications, to be of particular interest to surgeons and physicians.

Sokoloff's monograph is in fact an expansion of an earlier essay published in 1963, and does not aim at being comprehensive. His analysis, for example, of diseases of the vertebrae is rather a light-weight effort, but it is difficult these days for most workers to match the experience of human disease acquired by careful and protracted participation in necropsies by Schmorl in Dresden or by Landells and Collins at the London Hospital and Leeds respectively. Collins' monograph on joint disease remains among the best in the field.

Sokoloff's approach is more detached, and to this extent has its own particular merits. He is primarily writing a review, presenting as he puts it "a framework for systematic research". The obscurity of the picture within this frame is apparent enough in one of his own conclusions (p. 23):

"The pathological findings in osteoarthritis are those of plastic remodelling and abrasion of the joint surfaces, and constitute clear evidence that mechanical factors influence the development of the lesion. They describe a disturbance of an internally interacting system, and, in this reviewer's judgement, no single point can be recognized from them as the initial or central focus of the pathological process."

This is a frank if rather confusing admission, but it does reflect Sokoloff's ability to view dispassionately his own experimental work and that of others. Yet I would hope that in the future he will devote time to developing and justifying his own working hypotheses: to writing as it were an apologia rather than a review. This would be of even greater value to research workers in the field of joint disease.

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Rheumatoid Factors and their Biological Significance. Edited by H. BARTFELD and W. V. EPSTEIN. 1969. Proceedings of a Conference sponsored by the Arthritis Foundation and the New York Academy of Sciences and held on May 26/27, 1969. Pp. 207. Published as a Supplement to the *Annals of the New York Academy of Sciences*, 1969, vol. 168, 1-207.

Early controversy about the nature of the rheumatoid factor (RhF) appears to have been resolved. It is now widely agreed that RhF reactivity is antibody activity directed against immunoglobulin G (IgG). The papers presented at this Conference represent an attempted reassessment of the nature of this reactivity, its mode of formation, and its role in the body.

The first section is concerned with methods of measurement of RhF and its distribution in health and disease. From the immunological standpoint, RhF can be considered as one of many kinds of anti-IgG reactivities which modern methods have revealed in the serum. Sensitive methods which detect reactivity directed against the intact IgG molecule reveal antiglobulin activity both in health and in a wide variety of diseases. In confirmation of previous observations, the latter have been noted to be preponderantly chronic infections, but it is of interest that antiglobulin activity has now been reported after the rejection of renal transplants. This suggests that an immune response to homologous tissue can provoke an antiglobulin reactivity similar to that aroused by "foreign" organisms.

It is widely held that RhF reactivity most probably arises because the combination of IgG as antibody with antigen alters the molecular conformation of the IgG with the exposure of determinants rendering the molecule autoantigenic. This is supported by the evidence, presented in the second section, that RhF shows higher affinity of interaction for aggregated or structurally distorted IgG than for the native molecule. In such interactions, RhF reactivity appears to be directed against a site (or sites) located on the C-terminal half of the heavy chain of IgG. This specificity distinguishes RhF and "RhF-like" reactivity from antiglobulins directed at sites on the light chains of IgG or at selected allotypic determinants. But the problem remains of defining the hypothetical "antigen" characteristic of rheumatoid arthritis which initiates the whole process.

Interest has been centred recently in IgG-antiglobulins and the capacity of RhF to interact with and reveal IgG-anti-IgG complexes in the joint fluids. In some circum-