Fibrinolysis
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Although recognized for at least a century, fibrinolysis has received far less attention than coagulation, and workers in the United Kingdom in recent years cannot be said to have been especially productive in this neglected field. This seems the more surprising when fibrinolysis is appreciated as the antithesis of coagulation, since the process by which fibrin is removed would appear to be as important as that by which it is formed; and indeed coagulation and fibrinolysis are perhaps best viewed as two aspects of a system of temporary repair whose sealing material is fibrin.

The first ‘Fibrinolysis Workshop’ was held at St. George’s Hospital, London, in 1962, and the second took place at Queen Mary’s Hospital, Carshalton, in October 1963. By calling the meeting a ‘workshop’ we intend to convey its informal and practical nature. Consequently the papers were more in the nature of an introduction to each topic, and as such are more informally presented than many which are published in this Journal. The second Fibrinolysis Workshop was attended by about four times the number of research workers. The scope was constantly wider, but the venue no doubt accounts for the emphasis on the relationship of the fibrinolytic system to neurology, neurosurgery, and neuropathology in children. Nevertheless, general and other particular aspects of fibrinolysis received due attention, and one session devoted to methodology was an opportunity for the description and discussion of techniques not easily available to the average reader. These are described in full in the present report, and it is hoped that they will be of especial interest to workers in other countries. The workshop was organized in four parts. The first (Chairman: R. G. MacFarlane) was concerned with methods of measurement; the second (Professor W. H. Davidson) with thrombolytic therapy; the third (Professor F. J. Gillingham) with neurosurgical aspects of fibrinolysis; and the last (G. R. Fearnley) with miscellaneous aspects. For publication it has been necessary to shorten the discussion, but without, it is hoped, detracting from its value.

Finally, for those unfamiliar with the subject, a few definitions may be helpful. Fibrinolysis means the enzymatic breakdown of blood or fibrin clot, and the enzyme responsible is plasmin (fibrinolysin). Plasminogen (profibrinolysin), an inactive precursor, is present in the globulin fraction of plasma and can be converted to plasmin by a number of agents, some foreign to the body, e.g., streptokinase, and some native, e.g., blood activator and urokinase. Free plasmin is not present in circulating blood because a large reservoir of antiplasmin (antifibrinolysin) exists to neutralize it. Dissolution of fibrin, at least within blood vessels, is believed to be dependent on adsorption or diffusion of activator which converts the intrinsic plasminogen of the thrombus to plasmin, which then causes lysis of the thrombus. According to present concepts, there thus exists an elegant mechanism whereby fibrinogen is protected because of the antiplasmin defences in circulating blood, whereas deposited fibrin picks up activator which causes its lysis. It would be mistaken, however, to suggest that such a mechanism is the only function of fibrinolysis. Though dimly understood, it is becoming apparent that fibrinolysis plays an important role in inflammation and repair throughout the body. With several aspects of these functions the Workshop held at Carshalton was concerned.

G.R.F.

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SECOND FIBRINOLYSIS WORKSHOP

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