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BOOK REVIEW

FIBRINOGEN 4, CURRENT BASIS AND CLINICAL ASPECTS, edited by M. Matsuda, S. Iwanaga, A. Takada and A. Henschen, International Congress Series No. 892, EXCERPTA MEDICA, Amsterdam - New York - Oxford, 1990 x + 226 pp., ISBN 0-444-81330-6, US$ 92.25/Dfl.180.00

The book can be ordered from ELSEVIER SCIENCE PUBLISHERS, P.O.Box 211, 1000 AE Amsterdam, The Netherlands, or in the USA/Canada from ELSEVIER SCIENCE PUBLISHING CO., INC., P.O.Box 882, Madison Square Station, New York, N.Y. 10159, U.S.A.

This volume contains the Proceedings of the International Fibrinogen Workshop held in Kyoto, Japan, 27-28 Aug. 1989, in conjunction with the ISTH Satellite Symposia. The book includes the following chapters: Structure-function relationships of fibrinogen, monoclonal antibodies, interactions of fibrin(ogen) with cells, fibrinolysis, molecular abnormalities, clinical aspects.

A main emphasis of the papers is put on the studies of fibrinogen-fibrin-conversion on molecular basis under special consideration of molecular abnormalities of fibrinogen. Important methodical advances were obtained by the development of monoclonal antibodies and of DNA-techniques.

For our readers some papers seem to be of major interest. Their topics are, for instance, the relations of fibrin, fibronectin and factor XIIIa in wound healing (E.L.R. Barry and D.F. Mosher), the uptake of plasma fibrinogen into platelet alpha-granules (E.M. Cramer, P. Harrison et al.), the binding of fibrinogen to surface GPIIb-IIIa receptors of platelets and macrophages as well (D.G. Moon, J.R. Shainoff and S.R. Gonda).

Fibrin enhances the rate of plasmin formation, due to the exposure of two stimulatory sites, Aα-[148-160] and two γ-chain fragments, known as FCB-5 (O. Yonekawa et al.). Human plasma fibrinogen contains three populations: the main fractions of high and of low molecular weight (340 KD, and 300 KD respectively) and a minor LMW' fraction. The distribution of these three fractions in normals are: HMW 57.2 %, LMW 34.3 %, LMW' 6.3 % (n= 35). There was no difference in patients suffering from thrombotic disorders (n= 46) (M. Mirshahi, J. Soria et al.). A. Takada et al. investigated the fibrinolytic potential of the blood during daytime and found that the low potential in the early morning and its enhancing in the afternoon is due to fluctuations in plasma PAI-1 levels, whereas there are no significant circadian rhythms of AT III, α_2AP, CIINH, α_2M, fibrinogen, plasminogen and urokinase.

The present status of structure-elucidation of molecular abnormalities of fibrinogen has been reviewed by M. Matsuda et al. From more than 200 abnormal fibrinogens, reported over the last 25 years, 21 types are listed. Their functional defects have been found mostly as altered fibrin polymerization. Some of these abnormalities had no clinical signs, others showed a life-long thrombotic tendency or posttraumatic and postpartum bleeding.

E. Wenzel et al. described the only rheological findings in such cases: a remarkable low viscosity and a reduced red cell aggregation. The heterogeneity of symptoms associated with the same biochemical findings needs until now a conclusive explanation.

J. Koopman and F. Haverkate applied DNA-techniques to elucidate the structural defects in abnormal fibrinogens on a more solid basis. J. Soria et al. studied patients with prethrombotic disorders and found in 24 out of 29 cases a concentration of soluble fibrin >500 ng/ml. D-dimer was elevated correspondingly. High D-dimer levels are strongly associated with head and spinal cord injuries (J.P. Chen et al.), with the progression of breast cancer (M. Neises et al.), and are a highly sensitive parameter of lung embolism (J. Lichey et al.).

The book documents the high research standard of fibrinogen, however, it also confirms the great gap in our knowledge between the biochemical and the hemorheological significance of fibrinogen.

Siegfried Witte

Medical Department
Diakonissen-Hospital
7500 Karlsruhe 51, Germany