Preface

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Glaucoma remains one of the leading causes of blindness, at least in Europe and in the USA. Despite great efforts of the scientific community in glaucoma research during the last two decades, the pathogenesis of this group of diseases has not been sufficiently clarified. From September 16 to 18 of last year on the occasion of the 75th birthday of Prof. Johannes Rohen, a group of pioneering scientists met in Erlangen for an international symposium in order to discuss the present stage of glaucoma research.

The Symposium was extremely fruitful and brought up so many unexpected new aspects that the papers submitted by the authors for consideration to Ophthalmologica have been collected after appropriate editorial review and correction for publication as a special issue of Ophthalmologica to make it more widely available. The volume is entitled 'Perspectives in Glaucoma Research Part II' and follows a part I which appeared in September/October 1996 (vol. 210/5/96).

The first topic concerned trabecular meshwork cells and glaucoma markers. One of the interesting new developments presented was the so-called TIGR protein, a specific new protein, which was expressed by trabecular cells under both corticosteroid and other stress conditions. During our meeting we discussed its potential role in physiological and perhaps anatomical processes in the trabecular meshwork in different types of glaucoma, including adult and juvenile POAG in addition to steroid glaucoma. During the process of reviewing and editing the manuscripts dealing with this peculiar protein, new information about genetic defects in TIGR protein by Stone et al. [Science 1977;275:668] appeared. An update of the papers dealing with this topic was accepted just prior to publication of the volume. The new results about the structure of that protein and the identification of the gene that 'causes POAG' may open a new field in glaucoma research and could be of great importance for the elucidation of pathogenetic processes and for the development of directed strategies in glaucoma therapy. Of interest in this respect was the finding that the trabecular meshwork in eyes with juvenile glaucoma shows morphological changes similar to those seen in eyes with steroid-induced glaucoma. Other important topics presented concerned new experimental studies on contractility of the trabecular meshwork and the phagocytic capabilities of trabecular cells in normal and glaucomatous eyes, which could also be useful for developing approaches to glaucoma therapy.

The second main topic of the Erlangen Symposium was related to the question how sustained intraocular pressure elevation affects the vascular system of the choroid and retina. One clinical study showed that POAG patients failed to demonstrate stabilization in retinal leukocyte velocity at higher intraocular pressure, particularly within the perimacular region. This is consistent with findings recently obtained from so-called monkey glaucomatous eyes. In eyes with sustained, long-term elevation of intraocular pressure the retinal vasculature also revealed characteristic changes, particularly within the perimacular region. These changes might be related to the

simultaneously occurring decrease in thickness of the choroid and number of choroidal ganglion cells located in the recently discovered nitrenergic nerve cell plexus of the choroid. The results may shed new light on the functional interrelationship between choroid and retina, and may also have an impact on understanding the essential pathologic processes in glaucomatous diseases. Other examples of the innovative ideas presented in this volume could be given, but it is left to the reader to consider the evidence from the different outstanding papers presented. Overall, the findings provide insights into essential biological processes that could play a role in the pathogenesis of glaucomatous diseases and may be crucial for the development of new therapeutic principles.

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