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CLINICAL GLAUCOMA

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FOREWORD

Glaucoma is in many ways a mysterious disease process. It is more than merely the reflection of an intraocular pressure, or a field defect, or a change in the optic nervehead. It is a congery of conditions and a variety of pathologic features which, when somehow fitted together, fall under the broad heading of glaucoma.

The ophthalmologist faced with a patient who has the constellation of an elevated ocular pressure, a typical field defect, and cupping of the optic nervehead has no problem in making a diagnosis of the condition. Very often medical management will suffice and the patient's treatment will be effective. Not infrequently, particularly in an advanced society such as ours, it is only a borderline intraocular pressure that is found by the clinician and the field may show only the smallest of defects, or the disc the faintest of cupping. What to do in this instance? What about the patient with anatomically narrow angles, whose ocular pressure is only elevated to a modest degree by provocative testing, and how does one manage such a patient? What about the new entities in glaucoma that have been described within the past decade; conditions such as plateau iris, ciliary block, and a host of pigmentary glaucomas? How is one to diagnose and manage this wide variety of conditions all falling under the broad heading of glaucoma?

Fortunately, George Gorin, who has for many years provided a continuing and ever-expanding series of lectures and clinical demonstrations on the various aspects of glaucoma to the ophthalmology staffs of the Albert Einstein College of Medicine and the Manhattan Eye and Ear Hospital, has provided us with this volume. It is the distillate of more than three decades of clinical experience in the area of glaucoma. Here we have a wise and expert clinician who blends his practical experience with modern scientific knowledge concerning this complex subject.

Readers of this volume, be they novice or expert, cannot fail to gain insight into what is one of the most important conditions in all of ophthalmology. Furthermore, they will appreciate the very detailed and up-to-date bibliography which adds such a valuable perspective to our understanding.

Most of all, this is a practical book which will help practitioners to properly treat their patients who have glaucoma.

Paul Henkind, M.D., Ph.D.

PREFACE

The purpose of this book is to describe the pathophysiologic basis of the major entities of glaucoma in adults.

My teaching experience in the Albert Einstein College of Medicine, Manhattan Eye, Ear and Throat Hospital, and at the Academy of Ophthalmology and Otolaryngology made me aware of the difficulties encountered by many ophthalmologists in mastering the intricacies of glaucoma. In addition, my experience with glaucoma consultations gave me an opportunity to deal with many difficult clinical problems.

No attempt is made in this book to offer a comprehensive classification of the glaucomas, as this is not possible at the present state of our knowledge. The pathophysiology of the angle-closure glaucomas and of the secondary glaucomas is described in detail. The pathophysiology of the most commonly encountered form of glaucoma, that of chronic simple glaucoma, remains, however, unknown in spite of major research efforts.

A large volume of work was done during the past twenty years in electron microscopy of the outflow channels in an effort to elicit the cause of open-angle glaucoma. I am aware that many of the findings described are controversial, such as the numerous theories of outflow mechanisms of aqueous, but they are included in the book in order to give the clinically oriented ophthalmologist a general picture of the problems encountered by researchers.

Much of our knowledge of the pathophysiology of glaucoma is based on research in rabbits and monkeys, the findings of which are presumed to apply to the human eye. However, as is seen from a detailed description of the anatomic, circulatory, and neurohumoral differences between these species and man, this may be a source of gross error.

A detailed description is given of aqueous formation and of the blood-aqueous barrier because it is deemed important in the treatment of one of the most severe forms of glaucoma, that of glaucoma secondary to uveitis.

This book also includes discussions on recent topics of interest, including the role of alpha and beta receptors, chemical sympathectomy, role of prostaglandins in uveitis, radial peripapillary capillaries, axoplasmic transport, laser iridectomy, panretinal photocoagulation in neovascular glaucoma, and the armamentarium for handling vitreous in malignant glaucoma.

Techniques of tonometry, tonography, gonioscopy, and perimetry are described only in connection with their clinical application.

I wish to thank Doctor Paul Henkind, Professor and Chairman of the Department of Ophthalmology at the Montefiore-Albert Einstein Medical School, for his encouragement to undertake the preparation of this volume and for going over and critically evaluating the manuscript. I am also grateful to the authors who graciously permitted me to use their illustrations. I am thankful to Mr. Henrick Malpica for the photographic work.

And finally, my deep appreciation to my wife for her unfailing support and patience during the writing of this book.

George Gorin, M.D., F.A.C.S.
New York

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SECTION ONE

PHYSIOLOGY, CHEMISTRY, AND ANATOMY OF AQUEOUS AND AQUEOUS PATHWAYS

CHAPTER 1

THE AQUEOUS AND AQUEOUS DYNAMICS

Glaucoma constitutes a group of disorders characterized, in general, by increased intraocular pressure, alterations in the optic nervehead, and abnormalities in the visual field (National Advisory Eye Council).*

Physiologic Chemistry of Aqueous

The aqueous is a clear fluid formed in the ciliary body at the rate of 3 μl/min, or 1% of the volume of the anterior chamber. It circulates from the posterior chamber through the pupil into the anterior chamber, trabecular meshwork, canal of Schlemm, and external collector channels into the intrascleral venous circulation. The flow of aqueous refers to bulk flow and does not include the turnover of water and ions by diffusional exchange through the iris capillaries, between the anterior chamber and the systemic circulation. According to Kinsey (1955), the volume of fluid turnover per minute in the rabbit eye is 50 μl, out of which only 4 μl are aqueous. Goldmann (1970) found the same in the human eye.

The parent fluid of aqueous is blood plasma, which supplies the constituents of aqueous through the blood vessels of the 70 to 80 ciliary processes. The region of the ciliary processes is one of the most richly vascularized parts of the body. The break-up of the small arterioles into wide, thin-walled capillaries is abrupt, causing high capillary pressure in the region. However, aqueous is not a simple dialysate of plasma, produced by the high hydrostatic pressure of the capillary bed of the ciliary processes. There are some differences between the composition of aqueous and plasma.

In the normal human eye the protein content in the aqueous is 5 mg/100 ml. In the rabbit, a commonly used experimental animal, the protein content of the aqueous is 10 times higher, 50 mg/100 ml. The concentration of glucose is lower in aqueous than in plasma. This could be accounted for partly by the utilization of glucose in the metab-

*Report of the National Advisory Eye Council, Vision Research Planning Committee, Vol. 1 and 2, Research Program Analysis, 1975.

olism of the lens and cornea, and partly by its slow diffusion into the aqueous. There is a high concentration of lactic acid as a result of glucose metabolism, and possibly through the activity of the ciliary epithelium

Normal human aqueous contains 25 times as much ascorbic acid as plasma, 25 mg/100 ml in the aqueous compared with 1 mg/100 ml in plasma. In the human fetus ascorbic acid rises during the last 3 months of gestation. This rise seems to coincide with the greater reliance of lens metabolism on aqueous, instead of oxygen, supplied by the perilental vascular circulation. It was shown, in electronmicrographic preparations, that in this period of fetal life the outflow channels undergo a transformation, tending to facilitate outflow of aqueous. This consists of a decrease in the thickness of the internal wall of the canal of Schlemm, which becomes reduced to a single layer of very thin endothelial cells, a feature absent in the 4-month-old fetus (Wulle 1968). The high content of ascorbic acid in the posterior chamber is due to come active process of secretion, the exact nature of which is still unknown. Ascorbic acid was considered, by Friedenwald, the substance that presumably supplied energy for the production of aqueous, but this theory was not proved.

The content of sodium in aqueous is slightly higher than that in plasma in all species, including man, as shown in both chemical and isotopic studies. According to Davson (1969), aqueous is slightly hypertonic to plasma in the order of 3 to 5 mOsm/liter. That an excess of sodium exists in aqueous was demonstrated by animal experiments in which aqueous was dialyzed against plasma, resulting in a sodium loss from aqueous and a gain by plasma. The excess of sodium is possibly responsible for the small osmotic excess of aqueous over plasma. However, Langham (1958) suggests that the bulk of aqueous is in osmotic equilibrium with blood, and that the difference found is to be attributed to methodologic error. Similarly, Levene (1958) established that the excess of sodium is balanced by a deficiency of other substances, so that there is no great difference between the osmolarity of aqueous and plasma.

There is a constant exchange of fluid and ions by diffusion between the anterior chamber and blood plasma through the iris capillaries. The only structure separating aqueous from the iris circulation is the endothelium of the capillaries. Lipid-soluble substances cross the iris barrier rapidly, water penetrates even more rapidly, as shown by experiments with heavy water. If the aqueous in the anterior chamber is in equilibrium with plasma, no diffusional exchanges between anterior chamber and plasma will occur. If the concentration of ions in the aqueous is higher than in plasma, as is the case with sodium in all species and bicarbonate in the rabbit, these ions will diffuse into the plasma. On the other hand, glucose, the concentration of which is higher in plasma, will diffuse into the anterior chamber through vessels in the iris but is constantly utilized in cornea and lens metabolism. As a result, the posterior chamber has a higher content of glucose than the anterior chamber. In the aqueous of the anterior chamber, there is a net gain of chloride through diffusion but a loss of bicarbonate which is replaced by chloride. Sodium diffuses back and forth constantly across the iris capillaries. Because substances are lost to the vitreous and lens, a knowledge of the concentration in the posterior chamber aqueous is very important. Without knowledge of the posterior chamber data, it is difficult to compare concentrations in aqueous with those in plasma. However, posterior chamber data cannot be obtained in human eyes and our knowledge is based on findings in animal eyes.