

英文影印版

BECKER-SHAFFER'S

贝克-谢弗  
青光眼诊断与治疗  
Diagnosis and  
Therapy of the  
Glaucomas

(第7版)

Seventh Edition

Robert L. Stamper  
Marc F. Lieberman  
Michael V. Drake

科学出版社  
Harcourt Asia  
M O S B Y

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# **BECKER-SHAFFER'S**

# **Diagnosis and**

# **Therapy of the**

# **Glaucomas**

**Seventh Edition**

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*with 525 illustrations*

**SCIENCE PRESS**  
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We dedicate this book to our families, whose love, devotion, support, and sacrifice made this multi-year project, as well as most of our other accomplishments, possible: to the memory of Maurice R. Stamper and Alfred and Netsy Lieberman, and to our parents Lionel and Viviane Paradise and Carl and Beatrice Drake; to our spouses Naomi Stamper and Brenda Drake; and to our children Juliet and Alison Stamper, Marjorie Stamper-Kurn, Michael Lieberman, and Christopher and Sean Drake.

And to our teachers, especially Drs. Becker and Shaffer, whose wisdom and encouragement have been a source of inspiration and strength; to our students, who help us continue learning; and to our patients, who constantly stimulate our search for better ways of managing their glaucoma.

# Foreword

Writing a foreword for the seventh edition of *Diagnosis and Therapy of the Glaucomas* provides an opportunity for us to reminisce about our working together on the first two editions. In retrospect we were able to discuss and summarize the consequences of emerging research and clinical developments that promised to contribute to our knowledge about the glaucomas, their pathogenesis, diagnosis, and treatment. We have followed with interest and excitement the NIH-stimulated research, the changing concepts, the technological developments, and the newer methods of diagnosis and therapy. These have appeared in succeeding editions by Allan E. Kolker and John Hetherington, Jr., followed by H. Dunbar Hoskins, Jr. and Michael A. Kass, and in the present volume by Robert L. Stamper, Marc F. Lieberman, and Michael V. Drake.

A number of unresolved problems we faced 40 years ago continue to challenge current-day researchers, including the precise site and nature of obstruction to outflow in primary open-angle glaucoma, vascular versus mechanical effects of intraocular pressure on the optic nerve, the nature of genetic defects and their relationship to corticosteroid responsiveness, and the susceptibility to damage, as well as the possibilities for neuroprotection of the optic nerve in spite of the disordered pathophysiology.

We know from first-hand experience the time and effort that goes into library research in writing and rewriting a book such as this. We want to take this opportunity to express our appreciation to our students for revising and updating our early efforts in each of the subsequent editions. They have not only corrected our errors but have added the newest and timeliest concepts of research and practice to both diagnosis and therapy of the glaucomas. The present volume continues this tradition and once again offers the key references and provocative ideas to stimulate laboratory and clinical research. In addition, it provides in a single volume a practical approach to clinical problems, which should prove useful to students and residents, as well as practicing clinicians.

**Bernard Becker, M.D.**

**Robert N. Shaffer, M.D.**

# *Preface*

This book has a proud history. It has served as a guide for treating patients with glaucoma to ophthalmologists throughout the world for almost four decades. The book was conceived and written by Bernard Becker and Robert N. Shaffer. It was later revised through multiple editions by Allan Kolker and John Hetherington, Jr. and, most recently, by H. Dunbar Hoskins, Jr. and Michael A. Kass. The current edition is the product of a third generation of glaucoma specialists who trained and/or practice in St. Louis and San Francisco.

We have followed the lead of our mentors in using the book to summarize our clinical experience with glaucoma and to interpret in a practical way the current literature and thinking about glaucoma and its management. Our understanding of glaucoma and its treatment has undergone significant change in the last decade. The book has been extensively rewritten, with new illustrations, many in color, and an extensive up-to-date bibliography. The classification of the glaucomas has been revised to reflect new findings in genetics and pathophysiology of both trabecular dysfunction and optic nerve damage. In addition, the classification of the infantile and pediatric glaucomas has been updated. In just the last few years, several new medications and operations have been added to our armamentarium. The sections on management not only review up-to-date information but also provide some guidance to the practitioner in individualizing the expanded and expanding choices for treatment. Glimpses into the possible treatments of the future are given. We hope the readers will be able to understand what we do know and what we still don't know about the glaucomas.

The format for this seventh edition has been updated to make reading, studying, and finding relevant information easier. As in previous editions, the emphasis is on providing, in one volume, the information necessary to allow the individual involved in or wishing to become involved in the management of the glaucomas to do so effectively and with understanding. Ultimately, the goal is to reduce vision loss and improve the quality of life for our patients at risk for glaucomatous disease.

**Robert L. Stamper**

**Marc F. Lieberman**

**Michael V. Drake**



# Acknowledgments

First and foremost, we would like to thank our mentors, Drs. Bernard Becker and Robert N. Shaffer. They, of course, provided the first editions of this text. Perhaps more importantly, their clinical acumen, intellect, dedication to teaching, and integrity have inspired us in our careers. They have served as role models not only for us but for a half-century of ophthalmologists around the world.

We would also like to acknowledge with thanks the contributions of the following ophthalmologists:

- Michael Berlin, M.D., for his contributions to the chapters on laser surgery.
- M. Roy Wilson, M.D., for his contributions on the epidemiology of open-angle glaucoma.
- Jacob A. Dan, M.D., Ph.D., and James Heltzer, M.D., for contributions to the chapters on medical therapy.
- Maurice Mosseri, M.D., for his contributions to the chapter on angle-closure glaucoma.

We also thank William H. Spencer, M.D., for his inspiration, encouragement, and support for this as well as so many other academic endeavors.

Every book has authors and editors. It is the editors who are the unsung heroes of a book. They wheedle, cajole, nag, suggest, help create, and, in the end, edit. It is the wheedling and the nagging that actually move a book toward its publication. The authors would like to thank Wendy Buckwalter, Laurel Craven, Susie Coladonato, Karen Rehwinkel, Carol Sullivan Weis, Bill Drone, and the staff at Mosby for their good work, as well as their good nature, patience, persistence, professionalism, and skills. We also extend our appreciation to our talented illustrator for this edition, Terry Toyama, as well as our illustrator for past editions, Nadine Sokol.

Behind the authors were a string of supporters. They include our families, the Departments of Ophthalmology at the University of California San Francisco and at California Pacific Medical Center, Pacific Vision Foundation, That Man May See, and The Glaucoma Research Foundation, all of San Francisco, and the Tibet Vision Project of the Public Health Institute of Santa Cruz, California. We are most appreciative of their encouragement and support.

**Robert L. Stamper, M.D.**

**Marc F. Lieberman, M.D.**

**Michael V. Drake, M.D.**

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**PART**

**I**

***Introduction***

# Introduction and Classification of the Glaucomas

## DEFINITIONS

The concepts and definitions of glaucoma have evolved in the past 100 years,<sup>1</sup> but still they remain imprecise and subject to technical qualifications. The word *glaucoma* originally meant “clouded” in Greek; as such, it may have referred both to a mature cataract and to corneal edema that might result from chronic elevated pressure. Today the term does not refer to a single disease entity, but rather to a group of diseases that differ in their clinical presentation, pathophysiology, and treatment. These diseases are grouped together because they share certain features, including cupping and atrophy of the optic nerve head, which has attendant visual field loss and is frequently related to the level of intraocular pressure (IOP).

In this text, glaucoma is defined as a disturbance of the structural or functional integrity of the optic nerve that can usually be arrested or diminished by adequate lowering of IOP. An important distinction must be noted in the criteria currently used to define primary open-angle glaucoma (POAG), in contrast to all other forms of glaucoma. POAG is explicitly characterized as a “multifactorial optic neuropathy [with] a characteristic acquired loss of optic nerve fibers,”<sup>2</sup> developing in the presence of open anterior chamber angles and manifesting characteristic visual field abnormalities, in the absence of other known causes of the disease. In contrast, all other types of glaucoma—from primary angle-closure<sup>3</sup> to the secondary glaucomas—are defined first and foremost by the presence of elevated IOP, and not in reference to the optic neuropathy that follows sustained elevated IOPs.

Classically the primary glaucomas are not associated with known ocular or systemic disorders that account for the increased resistance to aqueous outflow; the primary diseases are usually bilateral and probably reflect genetic predispositions. Conversely, the secondary glaucomas are associated with ocular or systemic abnormalities responsible for the alteration in aqueous outflow; these diseases are often unilateral and acquired. Some have argued that the distinctions between “primary” and “secondary” simply reflect our imperfect understanding of pathophysiologic events that converge in the common final pathway of optic atrophy and visual field loss.<sup>4</sup> Although many risk factors have been associated with the development of POAG (Table 1-1), elevated IOP remains the most prominent factor—shared among the primary and secondary glaucomas—and the only factor contemporary ophthalmic intervention can reliably affect.

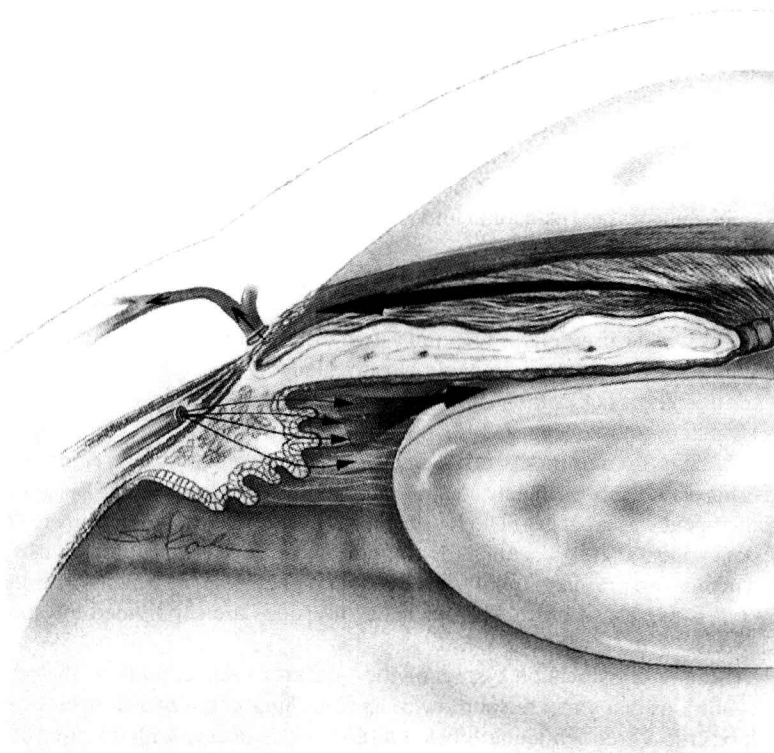
IOP is determined by the balance between the rate of aqueous humor production of the ciliary body, the resistance to aqueous outflow at the angle of the anterior chamber, and the level of episcleral venous pressure (Fig. 1-1). In most cases elevated IOP is caused by increased resistance to aqueous humor outflow. The optic nerve and visual field changes of glaucoma are determined by the resistance to damage of the optic nerve axons.

In most cases of glaucoma, progressive changes in the visual field and optic nerve are related to increased IOP; in some instances even “normal” levels of IOP are too high for proper functioning of the optic nerve axons. Although there may be absolutely no “safe” pressure that guarantees the absence of POAG,<sup>5</sup> lowering IOP to the low-normal range usually arrests or slows the progress of glaucoma.<sup>6,6a</sup> If this is not true, it can be postulated that (1) the IOP is not low enough to stabilize the disease; (2) the optic nerve is so damaged that even the normal age-related loss of axons produces further visual deterioration; or (3) there is a non-pressure-dependent element to the visual loss. If the third situation exists, it is not currently distinguishable from glaucoma.

**Table 1-1**  
**Risk Factors in Primary Open-Angle Glaucoma**

Factor	Quality of Evidence	Remarks
<b>Ocular Risk Factors</b>		
Intraocular pressure	Excellent	Most important
Myopia	Good	Related to IOP and to optic nerve?
Increased cup/disc ratio	Equivocal	May represent early POAG
Asymmetric cupping	Equivocal	May represent early POAG
Disc hemorrhage	Equivocal	Prognostically important
<b>Nonocular Risk Factors</b>		
Age	Excellent	Causal mechanisms unknown
Race (e.g., African ancestry)	Excellent	Causal mechanisms unknown
Family history	Good	Multifactorial genetic factors
Adult onset diabetes	Good	Vasculopathy related?
Systemic hypertension	Fair	Biologically plausible
Migraine and peripheral vasospasm	Equivocal	More relevant in "low tension" disease?
Gender	Inadequate	Contradictory reports
Alcohol consumption	Inadequate	Requires confirmation
Cigarette smoking	Inadequate	Requires confirmation

Data from References 18, 20, 32-35 and Wilson MR: *Glaucoma: epidemiology and risk factors*. In Higginbotham EJ, Lee DA: *Management of difficult glaucomas*, Boston, 1994, Blackwell.



**Figure 1-1** Anterior segment of the eye. Aqueous humor is formed by the ciliary epithelium, passes between the iris and lens to enter the anterior chamber, and leaves the eye through the trabecular meshwork and Schlemm's canal.

## EPIDEMIOLOGIC AND SOCIOECONOMIC ASPECTS OF THE GLAUCOMAS

Whether manifesting as POAG, primary angle-closure, or congenital disease, glaucoma is among the leading causes of blindness in the developing world<sup>7,8</sup> and a major health problem in the developed world. In the United States, glaucoma of all types is the second leading cause of legal blindness, affecting more than 80,000 Americans.<sup>2</sup> Among white and black populations POAG accounts for nearly two thirds of all reported glaucoma cases.<sup>9,10</sup> Among some Asian populations, however, primary angle-closure glaucoma is the more common disease.<sup>11,12</sup>

It is estimated that 2.25 million people in the United States over the age of 40 years have POAG,<sup>13</sup> half of whom are unaware of their disease despite demonstrable visual field loss.<sup>14</sup> Another 10 million Americans are estimated to have IOPs greater than 21; approximately 10% of these eyes will convert to POAG over the course of a decade.<sup>15</sup> The relationship between IOP and glaucomatous optic neuropathy is complex. On the one hand, the higher the IOP, the likelier the risk of POAG; conversely, one out of six eyes with POAG never demonstrates IOP higher than the age-appropriate normal range.<sup>16,17</sup>

An enormous amount of superb epidemiologic information has been forthcoming in the past decade, characterized by comprehensive population-based studies with rigorous criteria for pressure measurements, angle evaluation, and disc and visual field assessment.<sup>18-21</sup> These studies consistently report a prevalence rate for POAG in 1% to 2% of white adults. However, significant racial differences exist. Among blacks, the prevalence is nearly 4 times higher.<sup>14</sup> These patients are twice as likely to be blind as their white counterparts, and they have the disease nearly 27% longer.<sup>10,22</sup> These facts reflect neither the supply of ophthalmologists nor the patient's personal income.<sup>23</sup> Even higher rates have been reported among some Caribbean populations,<sup>24-26</sup> although there are lower and more variable prevalence rates among the genetically heterogeneous African populations from whom these New World populations descended.<sup>27</sup>

A major development in applying the epidemiologic evidence to clinical use is the concept of risk factors (see Chapter 2). Table 1-1 lists those factors that have demonstrated to a greater or lesser extent statistical correlation with either the development or the progression of POAG.

With the basic medical resources available in the developed world,<sup>17</sup> nearly all cases of blindness from glaucoma are preventable if the disease is detected early and proper treatment is implemented. Detection depends on education—educating the public about the importance of routine examinations, and training fellow health professionals to recognize the signs and symptoms of glaucoma. Screening strategies that rely only on IOP measures and that neglect disc and visual field assessment are inadequate;<sup>28</sup> and even when full testing is performed, it may not be cost effective.<sup>29</sup> Only through a massive public health effort of education and outreach can this scourge of preventable, unnecessary glaucoma blindness be addressed.

## CLASSIFICATION OF THE GLAUCOMAS

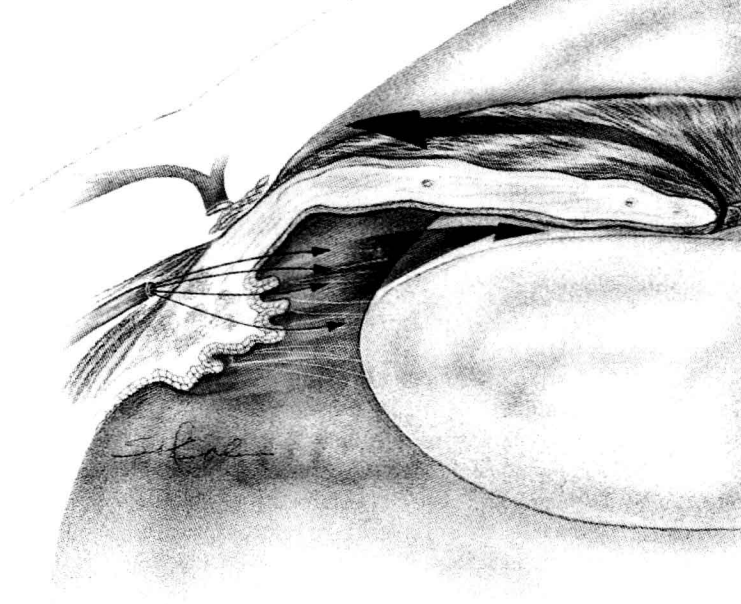
The most widely used classification system of the glaucomas separates angle-closure glaucoma from open-angle glaucoma. In angle-closure glaucoma, resistance to outflow is increased because the peripheral iris is blocking the trabecular meshwork—that is, the angle or periphery of the anterior chamber is closed (Fig. 1-2). In open-angle glaucoma, there is impaired flow of aqueous humor through the trabecular meshwork–Schlemm's canal–venous system (Fig. 1-3).

This classification scheme continues to be helpful because it clarifies pathogenetic mechanisms and therapeutic approaches. The four major divisions within this system are (1) angle-closure glaucoma; (2) open-angle glaucoma; (3) combined-mechanism glaucoma, in which two or more forms of glaucoma are present; and (4) developmental glaucoma, in which some anomaly of the anterior segment is present at birth. All four major divisions are subdivided into primary and secondary categories.

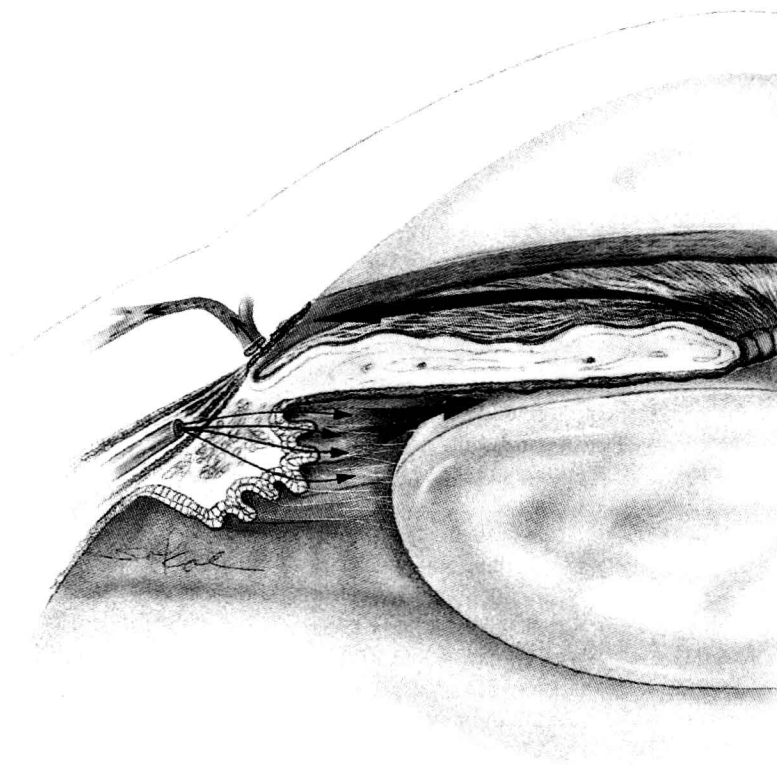
A similar classification system divides glaucoma into conditions that affect the internal flow, and conditions that affect the outflow of aqueous humor. Internal-flow block is caused by pupillary block or cilio-vitreo-lenticular block. Outflow block occurs with diseases of the trabecular meshwork, Schlemm's canal, collector channels, and the venous system.

Alternative classification systems<sup>4</sup> are based on other features of the diseases, including (1) the site of the outflow obstruction, which is divided into diseases that affect the pretrabecular passage of aqueous humor (e.g., posterior synechiae to the lens after ocular inflammation), the trabecular flow (e.g., glaucoma after administration of  $\alpha$ -chymotrypsin), and the posttrabecular move-





**Figure 1-2** In angle-closure glaucoma, the peripheral iris covers the trabecular meshwork, obstructing aqueous humor outflow.



**Figure 1-3** In open-angle glaucoma, there is impaired flow of aqueous humor through the trabecular meshwork–Schlemm's canal–venous system.