英文影印版

GOLDMAN'S
CECIL
MEDICINE

西氏内科学

第24版

眼、耳鼻喉及皮肤疾病分册

LEE GOLDMAN
ANDREW I. SCHAFER

ELSEVIER

北京大学医学出版社

Cecil

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24TH EDITION

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（第24版）

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北京大学医学出版社
Peking University Medical Press

图书在版编目（CIP）数据

西氏内科学：第24版. 眼、耳鼻喉及皮肤疾病分册：
英文/（美）戈德曼（Goldman，L.），（美）谢弗（Schafer，A. I.）
主编. —影印本. —北京：北京大学医学出版社，2012.1
ISBN 978-7-5659-0315-1

Ⅰ.①西… Ⅱ.①戈…②谢… Ⅲ.①内科学-英文
②眼病-诊疗-英文③耳鼻咽喉病-诊疗-英文
④皮肤病-诊疗-英文 Ⅳ.①R5

中国版本图书馆CIP数据核字（2011）第254546号

This edition of pages 2425 through 2556 of Goldman's Cecil Medicine, 24th Edition by Lee Goldman, Andrew I. Schafer is published by arrangement with Elsevier Inc.

ISBN-13：978-1-4377-1604-7
ISBN-10：1-4377-1604-0

Elsevier (Singapore) Pte Ltd.
3 Killiney Road #08-01 Winsland House I,
Singapore 239519
Tel：(65) 6349-0200
Fax：(65) 6733-1817
First Published 2012
2012年初版

北京市版权局著作权合同登记号：图字：01-2011-6967

西氏内科学（第24版）——眼、耳鼻喉及皮肤疾病分册

主　　编：Lee Goldman，Andrew I. Schafer
出版发行：北京大学医学出版社（电话：010-82802230）
地　　址：（100191）北京市海淀区学院路38号　北京大学医学部院内
网　　址：http://www.pumpress.com.cn
E-mail：booksale@bjmu.edu.cn
印　　刷：北京画中画印刷有限公司
经　　销：新华书店
责任编辑：冯智勇　　**责任印制**：张京生
开　　本：889mm×1194mm　1/16　　**印张**：8.5　　**字数**：423千字
版　　次：2012年1月第1版　2012年1月第1次印刷
书　　号：ISBN 978-7-5659-0315-1
定　　价：46.00元

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PREFACE

The 24TH Edition of *Goldman's Cecil Medicine* symbolizes a time of extraordinary advances in medicine and in technological innovations for the dissemination of information. This textbook and its associated electronic products incorporate the latest medical knowledge in formats that are designed to appeal to learners who prefer to access information in a variety of ways.

The contents of *Cecil* have remained true to the tradition of a comprehensive textbook of medicine that carefully explains the *why* (the underlying normal physiology and pathophysiology of disease, now at the cellular and molecular as well as the organ level) and the *how* (now frequently based on Grade A evidence from randomized controlled trials). Descriptions of physiology and pathophysiology include the latest genetic advances in a practical format that strives to be useful to the nonexpert. Medicine has entered an era when the acuity of illness and the limited time available to evaluate a patient have diminished the ability of physicians to satisfy their intellectual curiosity. As a result, the acquisition of information, quite easily achieved in this era, is often confused with knowledge. We have attempted to counteract this tendency with a textbook that not only informs but also stimulates new questions and gives a glimpse of the future path to new knowledge. Grade A evidence is specifically highlighted in the text and referenced at the end of each chapter. In addition to the information provided in the textbook, the Cecil website supplies expanded content and functionality. In many cases, the full articles referenced in each chapter can be accessed from the Cecil website. The website is also continuously updated to incorporate subsequent Grade A information, other evidence, and new discoveries.

The sections for each organ system begin with a chapter that summarizes an approach to patients with key symptoms, signs, or laboratory abnormalities associated with dysfunction of that organ system. As summarized in Table 1-1, the text specifically provides clear, concise information regarding how a physician should approach more than 100 common symptoms, signs, and laboratory abnormalities, usually with a flow diagram, a table, or both for easy reference. In this way, *Cecil* remains a comprehensive text to guide diagnosis and therapy, not only for patients with suspected or known diseases but also for patients who may have undiagnosed abnormalities that require an initial evaluation.

Just as each edition brings new authors, it also reminds us of our gratitude to past editors and authors. Previous editors of *Cecil Medicine* include a short but remarkably distinguished group of leaders of American medicine: Russell Cecil, Paul Beeson, Walsh McDermott, James Wyngaarden, Lloyd H. Smith, Jr., Fred Plum, J. Claude Bennett, and Dennis Ausiello. As we welcome new associate editors—Wendy Levinson, Donald W. Landry, Anil Rustgi, and W. Michael Scheld—we also express our appreciation to Nicholas LaRusso and other associate editors from the previous editions on whose foundation we have built. Our returning associate editors—William P. Arend, James O. Armitage, David Clemmons, Jeffrey M. Drazen, and Robert C. Griggs—continue to make critical contributions to the selection of authors and the review and approval of all manuscripts. The editors, however, are fully responsible for the book as well as the integration among chapters.

The tradition of *Cecil Medicine* is that all chapters are written by distinguished experts in each field. We are also most grateful for the editorial assistance in New York of Theresa Considine and Silva Sergenian. These individuals and others in our offices have shown extraordinary dedication and equanimity in working with authors and editors to manage the unending flow of manuscripts, figures, and permissions. We also thank Faten Aberra, Reza Akari, Robert C. Brunham, Ivan Ciric, Seema Daulat, Gregory F. Erikson, Kevin Ghassemi, Jason H. Huang, Caron Jacobson, Lisa Kachnic, Bryan T. Kelly, Karen Krok, Heather Lehman, Keiron Leslie, Luis Marcos, Michael Overman, Eric Padron, Bianca Maria Piraccini, Don W. Powell, Katy Ralston, James M. Swain, Tania Thomas, Kirsten Tillisch, Ali Turabi, Mark Whiteford, and Y. Joseph Woo, who contributed to various chapters. At Elsevier, we are most indebted to Dolores Meloni and Linda McKinley, and also thank Cathy Carroll, Taylor Ball, Virginia Wilson, Linda Van Pelt, Suzanne Fannin, and Steve Stave, who have been critical to the planning and production process under the direction of Mary Gatsch. Many of the clinical photographs were supplied by Charles D. Forbes and William F. Jackson, authors of *Color Atlas and Text of Clinical Medicine*, Third Edition, published in 2003 by Elsevier Science Ltd. We thank them for graciously permitting us to include their pictures in our book. We have been exposed to remarkable physicians in our lifetimes and would like to acknowledge the mentorship and support of several of those who exemplify this paradigm—Robert H. Gifford, Lloyd H. Smith, Jr., Frank Gardner, and William Castle. Finally, we would like to thank the Goldman family—Jill, Jeff, Abigail, Mira, Daniel, and Robyn Goldman—and the Schafer family—Pauline, Eric, Pam, John, Evan, and Kate—for their understanding of the time and focus required to edit a book that attempts to sustain the tradition of our predecessors and to meet the needs of today's physician.

LEE GOLDMAN, MD
ANDREW I. SCHAFER, MD

CONTENTS

XXVI

EYE, EAR, NOSE, AND THROAT DISEASES

431 DISEASES OF THE VISUAL SYSTEM

MYRON YANOFF AND DOUGLAS CAMERON

The eye is a compact, complicated structure (Fig. 431-1) that is remarkably stable throughout life. Once the growth of the eye is complete, at about the age of 3 years, the structure of the eye changes very little for the next 60 to 80 years.

The eyelids physically protect the eye. The visual axis contains blood vessel–free structures, including the tear film, cornea, intraocular aqueous, crystalline lens, vitreous, and retina that, except for the crystalline lens, remain essentially transparent throughout life. Delicate intraocular structures are protected by a tough collagenous "eye wall" composed of the cornea and sclera. The optic nerve is formed from retinal ganglion cell axons and the continuation of the brain dura, arachnoid, and pia maters. The optic nerve is long enough to allow free excursions of the eye through a 100-degree arc under the influence of six coordinated and critically placed rectus muscles. All these functional components are housed in a bony cavity, the orbit, which protects the eye from external injury.

The eyelid skin, only loosely connected to underlying structures, is among the thinnest of the body. The eyelid is unique because it contains the highest density of sebaceous glands in the body. These meibomian glands produce a sebaceous (lipid) material that is the principal evaporation retardant for the tear film. Malorientation of the eyelid margin or malorientation of the cilia (trichiasis) may cause extensive scarring to the anterior surface of the cornea, even to the point of blindness. The eyelid is opened under the influence of the levator muscle. The tendon of the levator tends to degenerate over time to produce mechanical ptosis. The soft tissue of the eyelid is separated from the soft tissue of the orbit by the orbital septum, a major collagenous barrier to protect orbital soft tissue from extension of eyelid inflammation. Extension of inflammation from preseptal cellulitis or ethmoiditis may cause septic optic neuropathy. Involution of elastic tissue supporting the skin of the anterior eyelid is reduced over time, causing dermatochalasis ("baggy eyelids"). The redundant tissue may be sufficient in quantity to restrict visual field, particularly superiorly.

The conjunctiva is a mucous membrane covered by stratified, nonkeratinizing squamous epithelium containing goblet cells. The epithelium is supported by delicate fibrovascular tissue that contains lymphatic channels. Squamous cell carcinoma or malignant melanoma originating in the conjunctiva may extend through these channels to regional lymph nodes or beyond. The conjunctival epithelium contains melanocytes. Immune processing cells are present in the epithelium (Langhans cells) and in the stroma in the forms of collections of non-nodal B and T lymphocytes. Non-nodal primary lymphomas, which tend to have an indolent course in this location, may arise from this tissue. The aqueous portion of tears is formed constantly by accessory lacrimal glands in the conjunctiva and lid soft tissue and by reflex action from the lacrimal gland. Symptoms of itching and burning, as well as periodic disturbance of vision, may result from decreased tearing.

Tears drain through puncta at the nasal eyelid margin to the nasolacrimal duct that exits inferior to the inferior turbinate of the nasal cavity. The epithelium of the nasolacrimal duct also contains melanocytes and is supported by a resting lymphocyte population. Neoplasms including lymphoma, concretions (dacryoliths), and tissue injury from trauma may occlude the puncta in adults.

The cornea is avascular and lined both anteriorly and posteriorly by surface cells. Lack of an adequate tear film, called dry eye syndrome, may seriously alter the ability of the cornea to transmit light, thereby affecting visual acuity. The posterior cellular lining of the cornea is a single layer of highly modified corneal endothelial cells. Lack of effective pumping by the endothelial cells will allow excess hydration of the corneal stroma, seen clinically as corneal edema. The corneal stroma is particularly sensitive to proteolysis from collagenases found with certain inflammatory diseases such as herpes simplex keratitis. The result is progressive corneal thinning and possible perforation of the cornea with multiple recurrent episodes of herpes simplex keratitis.

The intraocular pressure is measured by indentation tomography. The amount of pressure necessary to flatten the central cornea is proportional to the intraocular pressure.

The anterior chamber is bounded by the posterior surface of the cornea, the anterior surface of the iris, and the anterior crystalline lens within the pupillary space. Aqueous material normally flows from the posterior chamber into the anterior chamber through the pupil and exits into the general circulation through the trabecular meshwork. Most causes of pathologically increased intraocular pressure and optic nerve damage, that is, glaucoma, are due to abnormalities of filtration through the trabecular meshwork. The posterior chamber is bounded by the posterior surface of the iris, the ciliary body circumferentially, and the anterior surface of the vitreous. The crystalline lens is located entirely in the posterior chamber.

The anterior segment is composed of the cornea and the anterior and posterior chambers. Most of the anterior segment is derived from the skin. The posterior segment is the remainder of the eye. Most of the posterior segment structures are derived from the central nervous system.

When first formed, the crystalline lens is a totally cellular structure bounded by a true basement membrane. Throughout life, the new cells that are continuously added from the outer layer epithelial cells compress the central cells, thereby resulting in cell degeneration in the central core (nucleus). The lens doubles in volume from birth to age 70 years at the cost of both pliability (presbyopia) and clarity (cataract). The lens is suspended in the posterior chamber by fibers (zonules) attached to the ciliary body.

The ciliary body is the posterior extent of the iris. Its surface cells produce aqueous, and its muscles function in accommodation.

The vitreous is composed mostly of water and type II collagen. The vitreous makes up the majority of the volume and weight of the eye. It functions as a biochemical sink as well as to maintain neural retinal attachment. With time, the vitreous shrinks and separates from the retina (posterior vitreous detachment). Condensed and displaced vitreous casts a shadow on the retina, which is perceived by the patient as "floaters."

The retina is the site of photochemical conversion of light to electrical energy. Ganglion cells and their axons in the internal retina aggregate at the optic disc to form the optic nerve. Only the inner half of the retina is supplied by the retinal circulation. The outer half of the retina is supplied by a large-caliber capillary lake in the choroid (the choriocapillaris). Only a 500-μm area of the posterior retina, the central macula (about 3 to 5% of the total retina), has the ability to resolve images to 20/20. The remainder of the retina has much less sensitive image resolution. Extensive biochemical support and control of stray light are performed by the retinal pigment epithelium located between the choriocapillaris and the photoreceptor outer segments. The blood-retinal barrier, which protects the biochemical integrity of the retina, is composed of anatomic attachments between neighboring retinal pigment epithelial cells, as well as attachments between vascular endothelial cells of the retinal circulation. The retina is held in place by physiologic forces that may be compromised by holes forming in the retina (rhegmatogenous retinal detachment) or by fluid accumulating in the subretinal space (serous retinal detachment).

The optic nerve is composed of approximately 1 million axons from retinal ganglion cells. Axons are separated into bundles by pial septa, which are in turn enclosed in an arachnoid layer. The dura is contiguous with the posterior sclera and the periosteum of the optic canal. The optic nerve is supplied by delicate vessels extending from the dura across the arachnoid to the pial septa. The central retinal artery is present in the axial layer of the optic nerve near the eye but does not supply blood to the optic nerve itself. The optic nerve travels through a collagenous sieve in the plane of the posterior sclera, the lamina cribrosa. The choroid is that portion of the uveal tract posterior to the retina. This layer is composed of various calibers of blood vessels that ultimately supply blood for the choriocapillaris. There are no lymphatic channels in the choroid.

The sclera is composed of dense, relatively disorganized collagen. It is opaque because of the nonhomogeneous structure of the collagen and the degree of hydration relative to the cornea. There are multiple scleral ostia for arteries, veins, and nerves, both posteriorly and anteriorly.

The orbit is composed of bones of the facial skeleton. Sutures between major bones exist in the superior nasal and superior temporal quadrants. Multiple vessels and nerves extend through the thin ethmoid bone from nasal sinus tissue medially. The orbital floor is poorly supported over the maxillary sinus and may rupture with increased intraorbital pressure. The nasolacrimal duct travels through a portion of the lacrimal bone. The optic nerve is protected by portions of the sphenoid bone. Major cranial nerves travel through the adjacent superior orbital fissure, also a portion of the sphenoid bone. There is no normal lymphoid tissue in the orbit outside of the lacrimal gland. The rectus muscle may be enlarged by inflammation in thyroid eye

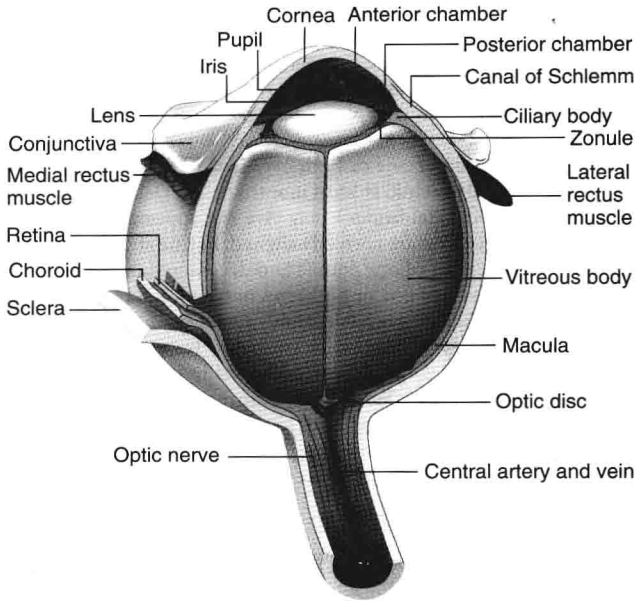

FIGURE 431-1. Anatomy of the eye.

TABLE 431-1 VISUAL ACUITIES REQUIRED FOR SELECTED DAILY TASKS

20/20	Physiologic vision
20/30-20/100	Driver's license, varies by state
20/50	Newspaper print
20/70	Large-print *Reader's Digest*
20/100	Write a check
20/200	Legally blind
20/400	Paper currency

disease, but the tendinous insertion into the sclera is not inflamed early in the course of the disease.

CLINICAL MANIFESTATIONS AND DIAGNOSIS

Patients may present with complaints of diminished vision, eye pain, red eyes, or pain around the eye. The causes may be primarily ophthalmic (e.g., cataract) or systemic (e.g., diabetic retinopathy). A comprehensive ophthalmologic examination also should evaluate for possible asymptomatic local (e.g., choroidal melanoma) or systemic (e.g., hypertensive retinopathy) abnormalities in patients with normal acuity and no subjective complaints.

Functional Evaluation

The most objective and common measure of ocular function is line letter acuity, with normal vision (Table 431-1) defined as the ability to see at 20 feet what a normal person sees at 20 feet (Fig. 431-2). Less than 20/20 vision can be caused by an abnormality anywhere from the tear film to the visual cortex of the occipital lobe (see Fig. 431-1). However, normal vision also includes other functions, such as the perception of color, motion, contrast, brightness, field, and depth, for which there is greater variation among individuals and no universally adopted, standardized scales. Normal visual acuity is potentially achievable in essentially all individuals, either naturally or with visual correction.

Correction of vision is based on the refraction of light (Fig. 431-3). The diopter (D) is the unit of measurement of the ability of an optical system to refract (bend) light. The normal human eye has the refractive capacity of approximately 60 D. If the eye is too short, light will be focused behind the eye (hyperopia). If the eye is too long, light will be focused in the vitreous in front the retina (myopia). Normally, a person can voluntarily control the crystalline lens, alternating between near and distant tasks. At approximately age 45 years, the lens becomes stiff, and the eye becomes set for distance (presbyopia). Refraction is the method of determining the amount of optical correction (strength of glasses) needed to establish 20/20 (6/6) vision.

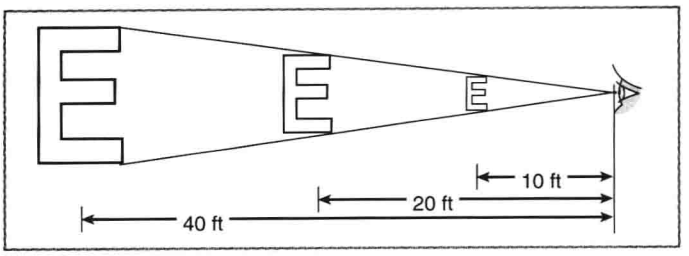

FIGURE 431-2. Snellen visual acuity. The most common test for visual acuity describes the eye's ability to resolve linear images at a test distance of 20 feet, approximating infinity (parallel rays of light). A 20/20 E subtends 5 minutes of arc at a distance of 20 feet, with each segment of the E subtending 1 minute of arc. The larger letters (e.g., 20/30, 20/40) are determined by the distance at which they subtend an angle of 5 minutes. Thus, an E that subtends 5 minutes at 40 feet, if viewed clearly at 20 feet, indicates 20/40 visual acuity.

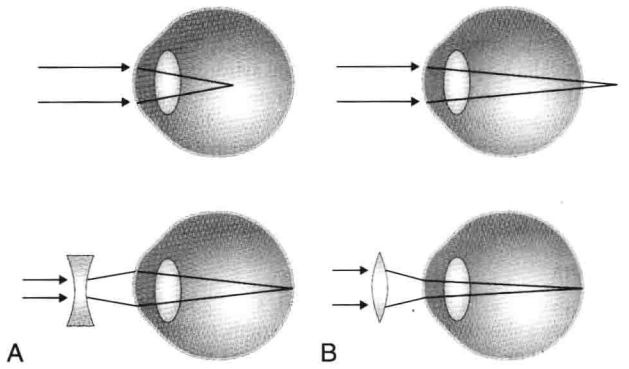

FIGURE 431-3. Myopia/hyperopia. A, In the myopic eye, parallel rays of light are focused anterior to the retina. A divergent lens can be used to compensate for the mismatch between refracting power and axial length. **B,** The hyperopic eye requires the additional power of a convergent lens to bring images into focus on the retina.

Examination of pupillary response assesses whether neural function is intact (see Figs. 432-2 and 432-4 in Chapter 432). Confrontational visual field testing (see Fig. 432-1 in Chapter 432) should be performed in each eye to detect gross quadrantic defects. Extraocular motility should be assessed to exclude nerve or muscle abnormalities (see Table 432-4 in Chapter 432). Color vision testing plates are a sensitive indicator of optic nerve function.

Diagnostic testing during a routine eye examination also includes external examination of the lids and adnexa, applanation tonometry to determine intraocular pressure, biomicroscopy (slit lamp examination) of the anterior segment, and ophthalmoscopic examination of the ocular fundus. Other office tests when indicated include exophthalmometry (measurement of proptosis), visual field and electrophysiologic testing, and vascular imaging (fluorescein angiography, mainly in diabetic patients; ocular computed tomography [OCT] to investigate retinal macular disease; and corneal topography).

The conjunctiva, cornea, lens, and anterior chamber are evaluated using a slit lamp. The slit lamp is composed of a binocular microscope with variable magnification (40× and 80×) in conjunction with adjustable light sources. An increased concentration of protein can be detected in the anterior chamber because of the Tyndall (flare) effect, indicating vascular incompetence associated with either inflammation or ischemia. Even individual inflammatory cells can be resolved with the slit lamp. A cobalt blue filter can be used to detect fluorescein dye that accumulates in regions of abnormal epithelium (dendrite of herpes simplex keratitis or a corneal abrasion). The slit beam is used to examine the crystalline lens to determine the degree of opacification from a cataract. By using a green filter with the 90 D lens, the retinal vessels and retinal vascular abnormalities such as microaneurysm can be seen at relatively high magnification.

COMMON CLINICAL CONDITIONS

Chronic Abnormal Vision

MYOPIA

Nearsightedness (myopia; see Fig. 431-3) is usually discovered during childhood when children cannot perform distant tasks during school (reading the

blackboard) or during school screening. Myopia usually progresses until the eye is fully developed, typically by age 20 to 25 years. Rapidly progressing myopia during childhood or at any time after age 25 years requires evaluation for juvenile glaucoma, diabetes mellitus (reversible metabolic changes in the crystalline lens), trauma (development of a cataract), or use of corticosteroids (development of a cataract). Myopia is usually fully correctable with glasses. Laser in situ keratomileusis (LASIK) is a surgical procedure that may be used in adults to correct myopia and other refractive errors, with 95% of patients achieving visual acuity of 20/40 or better. Complications of LASIK include glare symptoms, dry eye, and undercorrection or overcorrection. Rare but serious complications include epithelial ingrowth, diffuse keratitis, and flap dislocation.

Pathologic myopia is a heritable condition causing progressive weakening of the posterior sclera and resulting increases in the axial length of the eye. The posterior radius of curvature of the eye increases (posterior staphyloma). The refractive error is usually above −8.00 D and may be as high as −20.00 D in severe cases. Abnormal physical forces in pathologic myopia may lead to retinal hole formation, retinal detachment, or intraocular hemorrhage. Associated systemic conditions include trisomy 21, Cornelia de Lange's syndrome, Stickler's syndrome, and Marfan syndrome. Clinical surveillance must be maintained to watch for treatable complications such as retinal detachment. Pathologic myopia is usually treated with spectacles or contact lenses. Refractive procedures are less successful in pathologic myopia because of the severity of the refractive errors and the presence of posterior segment abnormalities. Surgical and laser procedures may be required to treat retinal and choroidal lesions (e.g., subretinal neovascularization).

HYPEROPIA

In hyperopia (farsightedness; see Fig. 431-3), in contrast to myopia, the eye tends to have a shorter than average axial length. Compensatory mechanisms of the crystalline lens may functionally correct small degrees of hyperopia until age 40 years, when the crystalline lens loses its pliability. The 40-year-old hyperopic changes from latent to manifest hyperopia simultaneously. The initial pair of glasses may have to correct for both distance and near tasks (bifocals). LASIK can correct up to 5 D of hyperopia.

ASTIGMATISM

Astigmatism usually is the result of irregularities in the radius of curvature of the cornea. Astigmatism is not a pathologic state, but rather a variation in anatomy; most people have some degree of astigmatism. Trauma to the cornea can cause alteration of structure, leading to irregular astigmatism. Symptoms are predominantly blurry vision and difficulty seeing fine detail. Regular astigmatism can be corrected with spectacle lenses or with rigid contact lenses, whereas irregular astigmatism requires rigid contact lenses. Some forms of astigmatism can also be corrected with laser ablation of the cornea.

KERATOCONUS

Keratoconus is an acquired irregularity of corneal curvature, possibly caused when inappropriate activation of matrix metalloproteinase weakens the structure of the cornea, particularly in the inferior nasal quadrant. Onset generally is during adolescence, and the process often evolves over 5 years or so. The prevalence in the United States appears to be about 55 cases per 100,000 people but is probably greater when subclinical cases diagnosed by computerized videokeratography are considered.

Patients typically have astigmatism, which may be severe. The condition is usually bilateral but not symmetrical. Mild degrees of keratoconus can be corrected with glasses or contact lenses. For severe cases, corneal transplantation (graft) often is very successful.

STRABISMUS

The control of simultaneous orientation of the two eyes to ensure that the visual axes of both eyes are aligned is not complete until several years following birth. Misalignments of the two eyes (strabismus) may be the result of abnormalities in the central nuclei of the brain, malfunction of one or several peripheral nerves, or intrinsic abnormalities of the rectus or oblique muscles (see Fig. 432-6 in Chapter 432). If the eyes are not simultaneously stimulated with the images of the same degree of clarity or complexity, only one eye will develop normally. The image processing ability of the blurred eye will not develop (amblyopia). Generally, amblyopia can be corrected until about age 7 to 8 years of age, after which an amblyopic eye is not likely to develop normal (20/20) vision. In most cases, only the central vision is affected. The peripheral vision in both eyes is likely to be equal and normal.

Amblyopia also may be caused by a marked difference in refractive error between the two eyes (anisometropic amblyopia) or eyelid ptosis (deprivational amblyopia). Ptosis may be neurogenic or mechanical (e.g., congenital eyelid hemangioma). To avoid amblyopia, it is extremely important to refer a child with strabismus to an ophthalmologist as soon as the strabismus is noted.

Esotropia is deviation of one or both eyes inward. Esotropia may not be clinically evident until the child is 3 to 4 months of age. Delay in facial maturation (underdeveloped nasal bridge) may give the appearance of esotropia, even though the visual axes are correctly aligned. In true strabismus, the light reflex will be in the center of one cornea and decentered in the other.

Exotropia is deviation of one or both eyes outward. Exotropia tends to be intermittent and less likely to result in amblyopia.

Both esotropia and exotropia may be treated by using appropriate corrections of refractive error with glasses (occasionally bifocals). Occasionally extraocular muscle surgery is necessary to correct alignment. To allow the second eye to develop to its full potential, amblyopia is corrected by occluding the stronger eye with a patch or by pharmacologically occluding (blurring) the better eye with atropine. Success is directly related to compliance.

DIPLOPIA (DOUBLE VISION)

Acute onset of diplopia is an ominous sign suggestive of a third nerve palsy (Chapter 432). Diplopia of any kind is an intolerable symptom, but vertical diplopia is less tolerated than horizontal diplopia.

COLOR VISION CHANGE

Most cases of congenital color blindness go undetected for many years. Acquired color deficiency at any age may be caused by a cataract or optic nerve disease.

CHANGE IN VISION

If only one eye has a change in vision, the problem, such as a cataract or retinal detachment, is most likely in that eye. If both eyes have a change in vision, the problem generally is outside of the eye, such as homonymous hemianopia (Chapter 432). Improvement of near vision in middle age may be a sign of cataract ("second sight") or hyperglycemia. Transient complete unilateral or bilateral loss of vision may be caused by vascular abnormalities inside or outside of the eye (Table 431-2).

Acute Eye Abnormalities

PAIN

The most severe eye pain (Table 431-3), typically associated with a red eye, is caused by acute angle-closure glaucoma. Sharp, intermittent pain is usually caused by ocular surface abnormalities (e.g., corneal foreign body). Burning pain that clears with blinking generally relates to tear-film abnormalities (dry eyes). Deep boring pain most often is associated with an ocular abnormality (e.g., uveitis).

RED EYE

A red or inflamed eye can be caused by conjunctivitis, iritis (anterior uveitis), acute glaucoma, corneal trauma, or infection (Table 431-4). Of these causes, all are typically painful, with the occasional exception of conjunctivitis.

TABLE 431-2

UNILATERAL	BILATERAL
Amaurosis fugax (carotid artery stenosis)	Eclampsia
Central retinal artery occlusion	Vertebrobasilar infarct
Occipital lobe infarct	Trauma
Temporal arteritis	
Nonarteritic anterior ischemic optic neuropathy	
Hemorrhage	
Preretinal (high altitude, Valsalva)	
Vitreous	
Aqueous (hyphema)	
Trauma	

DISTORTED VISION

Distorted vision (metamorphopsia), which is the perception that straight lines are distorted or bowed, results from macular dysfunction. Causes include fluid under the retina, exudative macular degeneration, which tends to elevate the retina, and an epiretinal membrane, which tends to contract the retina.

NIGHT BLINDNESS

Retinitis pigmentosa, vitamin A deficiency, and systemic medications such as phenothiazines can cause true night blindness, in which patients have difficulty seeing any stars in the sky on a clear night and may be unable to ambulate without assistance in a dark environment. Patients with cataracts may have difficulty driving at night because of excessive glare and visual distortion.

SENSATION OF FLASHING LIGHTS

The sudden onset of flashes in the peripheral visual field suggests a posterior vitreous detachment with resulting traction of the vitreous on the peripheral retina, sometimes with a resulting retinal tear. The flashes, which may be more pronounced in the dark and with rapid eye movement, may be associated with the sudden onset of floaters, which can indicate debris or blood in the vitreous cavity. Because a tear in the retina can lead to a retinal detachment, urgent consultation with an ophthalmologist is required.

Flashing light with a migraine (Chapter 405) is described as scintillations or zigzagging lights that march across the visual field for a few minutes or as long as 30 minutes, sometimes associated with transient visual field loss. Headache is not universal.

FLOATERS

Floaters, which are caused by small aggregates in the vitreous cavity, result from the normal aging of the vitreous. The acute onset of vitreous floaters may be associated with uveitis or with the sudden onset of bleeding in the vitreous cavity owing to diabetes or sickle cell anemia. Acute floaters, however, particularly if associated with flashing lights, may indicate a posterior vitreous detachment or a retinal tear with an impending retinal detachment. Urgent ophthalmic referral is essential.

PHOTOPHOBIA

Photophobia, particularly if associated with eye pain, redness, and decreased vision, is a symptom of uveitis or traumatic iritis. Photophobia is also typical of acute migraine and meningeal irritation. Prompt ophthalmologic referral is prudent.

HALOS AROUND LIGHTS

Patients with cataracts commonly see halos around lights, particularly when driving at night. Episodic decreased vision, redness, and halos around lights may be symptoms of impending angle-closure glaucoma. Halos also can occur as a complication of LASIK eye surgery.

FOREIGN BODY SENSATION

A foreign body sensation is commonly caused by dry eyes. An entropion (Fig. 431-4) or misdirected lashes (trichiasis) also can cause a foreign body sensation. Most corneal abrasions cause severe pain, but minor corneal abrasions may be associated with a foreign body sensation rather than the severe pain that usually accompanies a more severe abrasion. An arc welder burn causes a punctate corneal keratopathy, and foreign body sensation may be a prominent symptom. A true conjunctival or corneal foreign body also may be present.

EXCESSIVE TEARING

Impairment of tear drainage can occur with an ectropion or any obstructions of the nasolacrimal drainage system. An entropion or abnormal lashes rubbing on the cornea (trichiasis) stimulate tear production.

EYELID TWITCHING

Any irritation of the conjunctiva or cornea can cause the eyelids to twitch. Occasional twitching of the lids usually is associated with stress or adrenergic stimulation. Benign essential blepharospasm is severe spasm of the lids leading to functional impairment. Multiple sclerosis (Chapter 419) also can cause lid spasm.

CONJUNCTIVITIS

Any ocular inflammation, including corneal ulcers, angle-closure glaucoma, endophthalmitis, and uveitis can be associated with secondary conjunctivitis. Conjunctivitis usually involves the entire conjunctiva, is associated with a discharge, and usually is not associated with pain (see Table 431-4).

PTOSIS (DROOPY EYELID)

Ptosis (Fig. 431-5) can be caused by a third nerve palsy, which usually is associated with diplopia and a reduction in elevation, depression, and medial movement of the pupil. Horner's syndrome (see Fig. 432-5 in Chapter 432) is associated with a small pupil. With myasthenia gravis (Chapter 430), other typical features of muscle weakness are usually present or can be elicited. Some patients have mild senile ptosis, especially after eye surgery.

TABLE 431-3

Blepharitis	Glaucoma
Blocked tear duct	Hordeolum (stye)
Chalazion	Iritis
Conjunctivitis	Keratoconus
Corneal abrasion	Optic neuritis
Dry eyes	Scleritis
Ectropion	Trauma
Entropion	Uveitis
Foreign object	

TABLE 431-4

FEATURE	ACUTE CONJUNCTIVITIS	ACUTE IRITIS†	ACUTE GLAUCOMA‡	CORNEAL TRAUMA OR INFECTION
Incidence	Extremely common	Common	Uncommon	Common
Discharge	Moderate to copious	None	None	Watery or purulent
Vision	No effect on vision	Slightly blurred	Markedly blurred	Usually blurred
Pain	None	Moderate	Severe	Moderate to severe
Conjunctival injection	Diffuse: more toward fornices	Mainly circumcorneal	Mainly circumcorneal	Mainly circumcorneal
Cornea	Clear	Usually clear	Steamy	Change in clarity related to cause
Pupil size	Normal	Small	Moderately dilated and fixed	Normal or small
Pupillary light response	Normal	Poor	None	Normal
Intraocular pressure	Normal	Normal	Elevated	Normal
Smear	Causative organisms	No organisms	No organisms	Organisms found only in corneal ulcers related to infection

*Other less common causes of red eyes include endophthalmitis, foreign body, episcleritis, and scleritis.
†Acute anterior uveitis.
‡Angle-closure glaucoma.

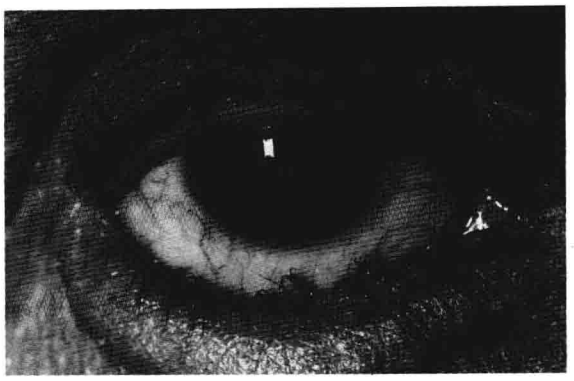

FIGURE 431-4. Involutional entropion. (From Palay DA, Krachmer JH. *Primary Care Ophthalmology,* 2nd ed. Philadelphia: Elsevier Mosby; 2005.)

FIGURE 431-5. Ptosis of the right upper lid. (From Palay DA, Krachmer JH. *Primary Care Ophthalmology,* 2nd ed. Philadelphia: Elsevier Mosby; 2005.)

PROPTOSIS (EXOPHTHALMOS)

Proptosis, or a prominent globe, can be a manifestation of thyroid abnormalities, especially Graves' disease (Chapter 233), in which proptosis is subacute and bilateral but sometimes asymmetrical (Fig. 431-6). An orbital pseudotumor can cause acute, usually unilateral proptosis, with severe pain, particularly with eye movement, and often with decreased vision. An optic nerve tumor causes chronic, unilateral proptosis associated with a slow onset of visual field loss. Acute cellulitis can be associated with unilateral proptosis, severe redness, and moderate to severe pain, commonly with sinusitis and an elevated white blood cell count.

SMALL PUPIL

A unilateral small pupil is best detected in dark conditions. Causes include Horner's syndrome, associated with ptosis on the same side; the bilaterally small, poorly reacting pupils of tertiary syphilis (Argyll Robertson pupils), which accommodate with normal constriction to a near object; miotic drops (e.g., pilocarpine); traumatic iritis; uveitis; and recent eye surgery.

LARGE PUPIL

Any α-adrenergic or anticholinergic agent placed into the eye can cause a large pupil. With eye trauma, the iris sphincter muscle can be damaged, and an abnormally large pupil can result. Tears in the iris sphincter can sometimes be appreciated on slit lamp examination. Third nerve palsy may cause a dilated pupil associated with ptosis and decreased elevation, depression, and medial eye movement. Adie's pupil (see Fig. 432-3 in Chapter 432) is an idiopathic, unilateral large pupil that is hypersensitive to weak cholinergic stimulation. Recent eye surgery, uveitis, closed-angle glaucoma, and traumatic iritis can cause a large pupil.

LEUKOKORIA

Leukokoria (white pupil) in a child is often a sign of retinoblastoma. However, any condition that changes transmission of ambient light to reflection of ambient light through the pupil may cause this sign. Some of the more common nonretinoblastoma conditions presenting with leukokoria (Table 431-5) include cataracts, retinal detachment, persistent hyperplastic primary vitreous (a developmental anomaly of the vitreous resulting in intraocular fibrosis and retinal detachment), Coats' disease (a developmental vascular malformation of the retina leading to retinal detachment), and ocular toxocariasis (a parasitic intraocular nematode infection, which leads to intraocular scarring and retinal detachment).

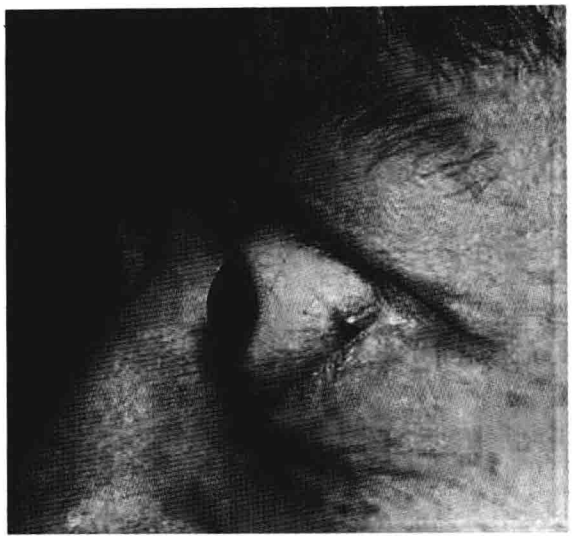

FIGURE 431-6. Graves' ophthalmopathy with characteristic exophthalmos and eyelid retraction.

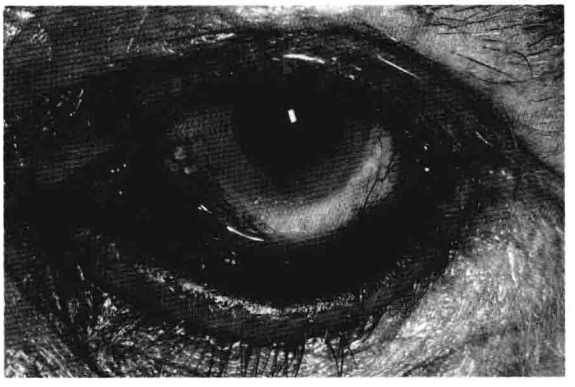

FIGURE 431-7. Involutional ectropion. (From Palay DA, Krachmer JH. *Primary Care Ophthalmology,* 2nd ed. Elsevier Mosby, Philadelphia, 2005.)

TABLE 431-5 DIFFERENTIAL DIAGNOSIS OF LEUKOKORIA

Retinoblastoma
Cataract
Persistent hyperplastic primary vitreous
Retinopathy of prematurity (retrolental fibroplasia)
Coats' disease (retinal telangiectasia)
Retinal detachment
Toxocariasis
Familial exudative vitreoretinopathy (FEVR)

Eyelid Abnormalities

ECTROPION AND ENTROPION

An ectropion is an out-turning of the lower lid (Fig. 431-7), typically with the inner part of the lower end visible between the eye globe and the lid. Causes include aging, scarring, a mass on the lower lid, and seventh nerve palsy. Common symptoms include burning, itching, tearing, and the sense of a foreign body. Treatment is symptomatic, unless the underlying cause can be surgically corrected.

FIGURE 431-8. Bilateral chalazion in the upper eyelids.

FIGURE 431-9. A lower lid stye *(1)*. (From Palay DA, Krachmer JH. *Primary Care Ophthalmology,* 2nd ed. Philadelphia: Elsevier Mosby; 2005.)

An entropion, which is an in-turning of the lower lid (see Fig. 431-4), is usually age-related and associated with irritation, burning, and a foreign body sensation. If it leads to trichiasis, in which the eyelashes rub or abrade the cornea, the lashes can be removed with forceps or surgery.

CHALAZION

A chalazion (Fig. 431-8) is a localized lipogranulomatous inflammation that results when the eyelid reacts to the contents of a ruptured sebaceous (meibomian) gland. The retained, lipid-rich sebaceous material acts as a foreign material that stimulates a lipogranulomatous foreign body inflammatory reaction. A painless or slightly tender, poorly demarcated, nonmobile nodule forms under the eyelid skin. Most lesions resolve over days to weeks with warm compresses or without specific treatment. Occasionally, an ophthalmologist may inject steroids to reduce inflammation or debulk the foreign material by incision and drainage through the tarsal conjunctiva. Some individuals may have recurrent chalazion.

HORDEOLUM (STYE)

A hordeolum (stye) (Fig. 431-9) is an extremely painful abscess in a hair or eyelash follicle or in a sebaceous gland. Styes are usually self-limited infections that respond to warm compresses and topical antibiotics (e.g., bacitracin or erythromycin ointment or moxifloxacin or gatifloxacin drops). Incision and drainage may be performed by an ophthalmologist if symptoms do not improve within 48 hours.

BLEPHARITIS

Blepharitis (Fig. 431-10), which is a nonspecific inflammation of the eyelid skin, is common, particularly in men. The condition is usually bilateral and symmetrical. Rosacea (Chapter 447) is the most common associated cutaneous condition, and *Staphylococcus aureus* is the most common infectious agent. If untreated, blepharitis becomes chronic and may lead to corneal and conjunctival inflammation (blepharoconjunctivitis). Ophthalmic antibiotic ointment (e.g., bacitracin or erythromycin) is more efficacious than eye drops, but systemic antibiotics (e.g., minocycline, 50 to 100 mg, or doxycycline, 100 mg, once daily; tetracycline, 250 mg twice daily; or erythromycin, 250 mg three times daily) are recommended if there is any evidence of inflammation of the cornea or conjunctiva.

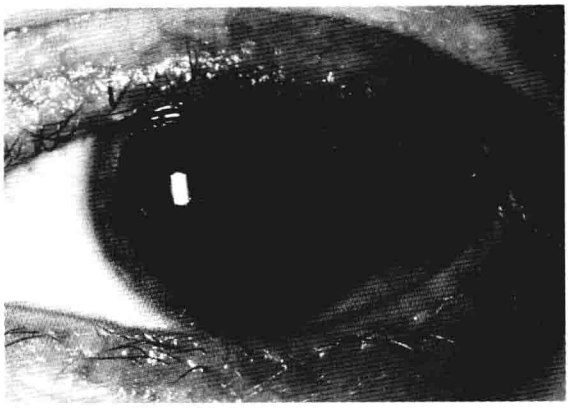

FIGURE 431-10. Staphylococcal blepharitis. The lid margins are very red and under high magnification demonstrate tiny ulcerations. (From Palay DA, Krachmer JH. *Primary Care Ophthalmology,* 2nd ed. Philadelphia: Elsevier Mosby; 2005.)

In seborrheic blepharitis, exfoliated keratinous debris accumulates along the eyelid margin, particularly at the follicles of the eyelashes, and irritates the conjunctiva. Treatment of this chronic condition is directed at mechanically removing the keratinous debris by scrubbing the eyelid and eyelashes daily with a mild detergent ("baby shampoo") in warm water applied with a soft cloth.

BENIGN EYELID NEOPLASMS

Skin tags, also known as squamous papillomas, are the most common benign skin lesions. Other skin lesions include seborrheic keratitis, actinic keratitis, inverted follicular keratitis, and benign lesions of the eccrine and apocrine systems. Most of these benign lesions are cured by simple excision.

SEBACEOUS GLAND CARCINOMA

Sebaceous gland carcinoma originates from sebaceous glands either in the tarsal plate (meibomian gland) or associated with eyelashes (glands of Zeis) and is capable of producing widespread metastasis resulting in death. Muir-Torre syndrome is sebaceous tumors associated with visceral malignancy. Except for chronic, unilateral blepharitis, owing to the peculiar manner of spread of this tumor in the plane of the skin epithelium (pagetoid spread) without causing the formation of nodules, few symptoms occur early in the course of the disease. The tumor may progress to involve the tarsal conjunctiva, the bulbar conjunctiva, and even the corneal epithelium. A characteristic sign is regional loss of eyelashes. When the mass thickens, it may have the appearance of a chalazion, and a history of multiple chalazia in the same region of the eyelid is suggestive of sebaceous gland carcinoma.

Treatment is surgical removal. Surgical margins are difficult to estimate because of the intraepithelial extension of the tumor. In advanced cases, removal of the eyelids, eye, and orbital contents (exenteration) may be necessary.

BASAL CELL CARCINOMA

Basal cell carcinoma (Fig. 431-11), which originates from the basal cell layer of the epithelium, is a common cutaneous malignancy (Chapter 210). The lesion, which usually is asymptomatic, is often a well-demarcated, elevated nodule that may have a central region of ulceration and fine cutaneous vascular channels (telangiectasias). A common benign cutaneous lesion, sometimes confused clinically with basal cell carcinoma, is seborrheic keratosis (Chapter 448), which tends to be soft and appear hyperpigmented; the most common site is the lower eyelid, especially in the nasal quadrant. Basal cell carcinoma, particularly near the medial canthus, may extend posteriorly into the soft tissues of the orbit. Imaging before surgical excision for medial canthal lesions may be necessary to determine the true extent of the tumor. Basal cell carcinoma is treated by surgical excision, using Mohs' technique with intraoperative histologic evaluation to determine adequate margins of excision, if possible.

Metastasis is extremely rare. Generally with early detection and adequate excision of the local lesion, the prognosis is excellent.

EYELID SQUAMOUS CELL CARCINOMA

Eyelid squamous cell carcinoma, which is much less common than basal cell carcinoma, arises from the surface squamous epithelium. Ultraviolet light exposure is the major risk factor. In contrast to basal cell carcinoma, squamous cell carcinoma can metastasize, most often to regional lymph nodes. Treatment is surgical excision. Except in rare circumstances, such as in immunosuppressed patients or patients with xeroderma pigmentosa, the prognosis is excellent.

Ocular Surface Abnormalities

DRY EYES

Even minor disturbances in the tear film can cause itching, burning, pulling, and transient changes in vision. Some patients are particularly disturbed by dry eyes that cause conjunctival hyperemia without purulent discharge. Paradoxically, decreased tearing can result in irritation and secondary increased (reflux) tearing.

Most daily tear production is not by the lacrimal gland but by small collections of lacrimal glands, mucous producing glands, and sebaceous glands located throughout the conjunctiva, eyelid, and anterior orbital soft tissue. Over time, particularly in women, production of tear film diminishes. Because tear film production is lower during sleep, patients often note symptoms on awakening followed by slow resolution over minutes or hours. Wind and low humidity environments, such as in commercial airliners, can exacerbate symptoms. The reduction in aqueous components of tears is often associated with a compensatory increase in mucous production, which tends to blur vision until the patient blinks or uses supplemental tears. These symptoms are particularly prominent in persons who have rheumatoid arthritis (Chapter 272), Sjögren's syndrome (Chapter 276), Stevens-Johnson syndrome (Chapter 448), and ocular cicatricial pemphigoid (Fig. 431-12).

Treatment is not definitive and is rarely satisfactory. No medication increases the production of tears. Low-viscosity artificial tears (e.g., polyethylene glycol 400 0.4%), which do not tend to blur vision but have a short duration of action, are best used during visually important tasks. High-viscosity tears (e.g., carboxymethylcellulose sodium) have a longer duration of action but tend to blur vision; they are best used at bedtime to maintain lubrication of the ocular surface during sleep. When artificial tears do not control symptoms, occlusion of the nasolacrimal duct with synthetic plugs or permanent surgical occlusion tends to retain the tears that are produced. Anti-inflammatory drugs (e.g., cyclosporine 0.05% drops, every 12 hours indefinitely) can preserve glandular tissue that may be affected by local inflammation. For patients with systemic disease associated with dry eyes, effective treatment of the systemic disease sometimes improves the eye abnormalities.

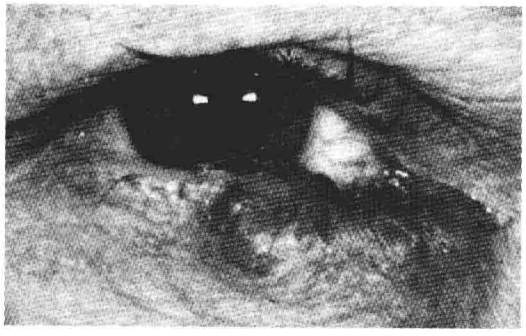

FIGURE 431-11. A typical nodular basal cell carcinoma. (From Palay DA, Krachmer JH. *Primary Care Ophthalmology,* 2nd ed. Philadelphia: Elsevier Mosby; 2005.)

PINGUECULA AND PTERYGIUM

A pinguecula (Fig. 431-13) consists of a limbal (at junction of cornea and sclera) and bulbar conjunctival degenerative process caused by ultraviolet light damage to the subepithelial tissue. It is very common and rarely causes symptoms. If the supportive tissue degeneration extends into the cornea, it becomes a pterygium (Fig. 431-14), which may cause visual changes and require surgical excision.

RECURRENT EROSION

Recurrent erosion of the cornea usually is a delayed reaction to minor traumatic corneal abrasion. The abrasion heals abnormally, resulting in a weakness of the epithelial attachment to underlying tissue. Weeks to months to years later, the patient is awakened in the middle of the night with extreme

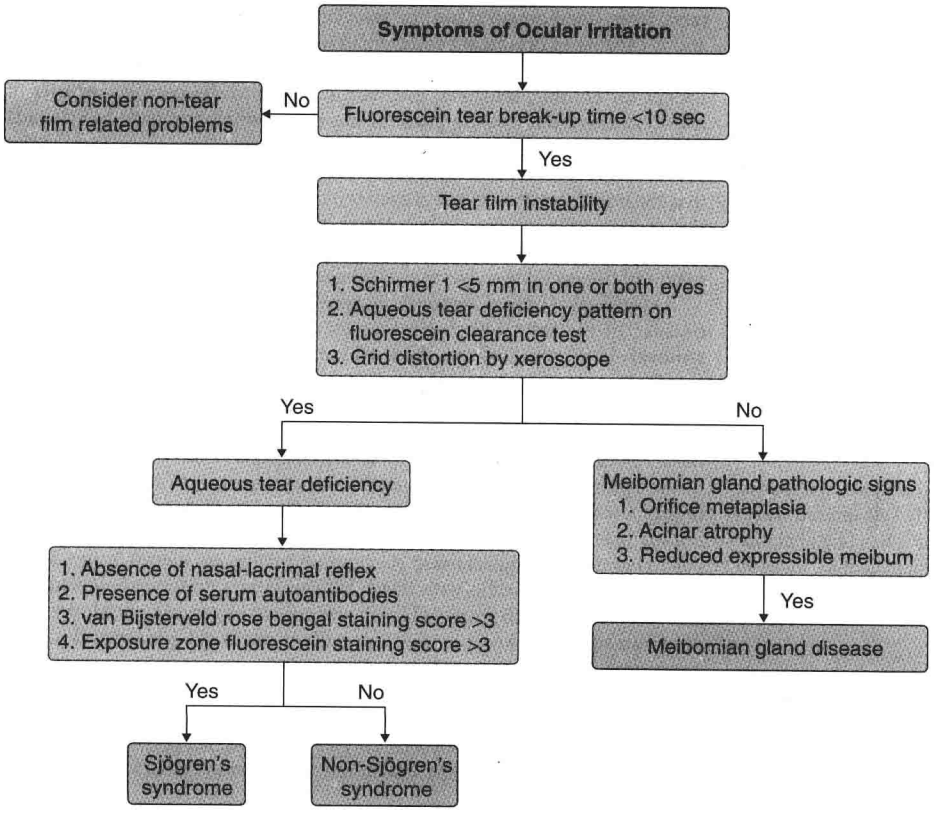

FIGURE 431-12. Diagnostic algorithm for ocular irritation. (Modified from Pflugfelder SC, Tseng SC, Sanabria O, et al. Evaluation of subjective assessments and objective diagnostic tests for diagnosing tear-film disorders known to cause ocular irritation. *Cornea.* 1998;17:38-56.)

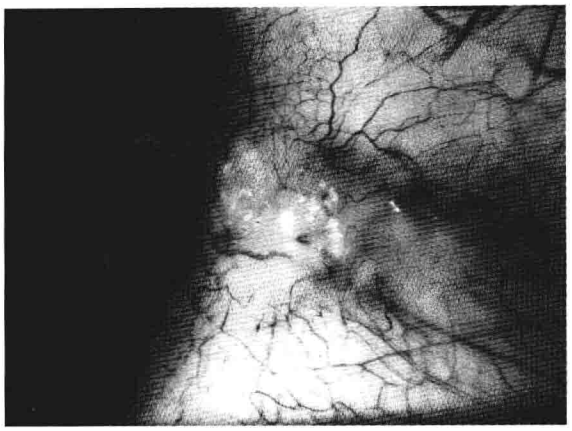

FIGURE 431-13. Pinguecula. These lesions are found at the 3-o'clock and 9-o'clock positions and are extremely common, especially in older patients. (From Palay DA, Krachmer JH. *Primary Care Ophthalmology,* 2nd ed. Philadelphia: Elsevier Mosby; 2005.)

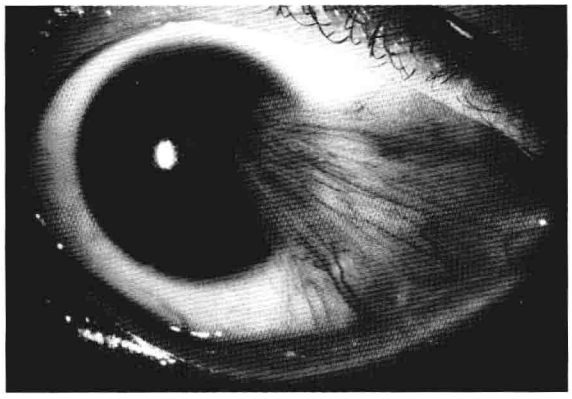

FIGURE 431-14. Pterygium. These lesions are found in the horizontal meridian, most common nasally. (From Palay DA, Krachmer JH. *Primary Care Ophthalmology,* 2nd ed. Philadelphia: Elsevier Mosby; 2005.)

ocular pain on opening the eyelids. The epithelium has become "stuck" to the overlying upper lid and is mechanically abraded. The condition is treated with hyperosmotic drops and ointment. There is a tendency to recurrence.

ACCIDENTAL TRAUMA

With ocular trauma, many tissues of the eye can be easily disrupted, and the effects of trauma may not be manifest for months or even years after the episode of trauma. If the traumatic episode disrupts the eye wall (cornea and sclera), surgical repair is necessary, usually urgently. If the eye wall is intact, surgical treatment is often not necessary, at least initially.

CORNEAL ABRASION

Corneal abrasion, one of the most common forms of ocular injury, may be caused by shearing trauma or hypoxia associated with overwearing of contact lenses. Symptoms are often intense and intolerable. Healing (i.e., re-epithelialization) of the cornea occurs within 24 to 48 hours. Rust from metallic fragments is toxic to the epithelium and should be removed. Fungal keratitis may complicate injuries from fingernails or vegetable matter, such as tree branches. Treatment usually consists of a topical antibiotic (e.g., erythromycin ointment, four times daily for 10 days) to prevent bacterial keratitis. Subsequent scarring usually does not occur unless deeper structures, such as Bowman's membrane, are affected. Topical anesthetics never should be prescribed to control pain because they increase the risk for microbial keratitis and scarring and may delay healing.

MAJOR OCULAR TRAUMA

Hyphema (Fig. 431-15) is hemorrhage into the anterior chamber caused by blunt trauma. If the patient is in an intensive care unit and supine, the blood will distribute uniformly over the iris to cause the appearance of increased pigmentation of the iris (heterochromia iridis). If the patient has been sitting,

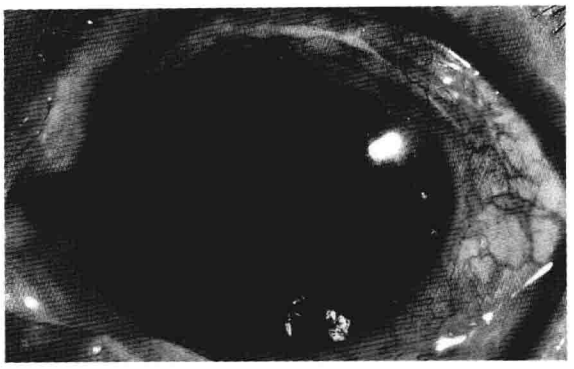

FIGURE 431-15. Hyphema following cataract surgery. (Courtesy of Dr. Myron Yanoff.)

the blood may settle by gravity to form an aqueous-blood interface with the blood in the dependent portion of the anterior chamber. Hyphema, which is a sign of serious ocular damage, may lead to secondary glaucoma and blood staining of the cornea. It requires prompt evaluation by an ophthalmologist.

The most common site of rupture of a globe is at the limbus (junction of cornea and sclera), where a pigmented mass may be noted. The mass may be either a blood clot or an anteriorly displaced uveal tract (usually iris). Any manipulation of the globe may force the remaining intraocular tissue through the wound and may make the injury irreparable. Surgical repair is usually indicated.

Cataract and retinal detachment are not common except in severe accidental trauma. A unilateral cataract or unilateral glaucoma may occur decades after the injury, even when an injury is too minor to be recalled. Traumatic cataract and traumatic glaucoma are treated in the same manner as other forms of these conditions.

INFLAMMATORY EYE DISORDERS

Uveitis

Inflammation of any part or parts of the uveal tract (iris, ciliary body, and choroid) may be called anterior or posterior uveitis, iritis, iridocyclitis, or choroiditis. Symptoms include a red eye (see Table 431-4), decreased vision, and photophobia. The inflammation is chronic, and a cause is rarely found. However, uveitis accompanies many autoimmune diseases, often without correlation with the activity of the systemic inflammation. Anterior uveitis or conjunctivitis is nearly universal in patients with reactive arthritis (Chapter 273). About 25% of patients with ankylosing spondylitis (Chapter 273) develop acute, recurrent anterior uveitis. Two to 12% of patients with inflammatory bowel disease (Chapter 143) develop anterior uveitis, which is also common with psoriatic arthritis but not with psoriasis alone (Chapters 273 and 446). Treatment with topical corticosteroids (e.g., prednisolone acetate 1%, one drop in the affected eye or eyes every 1 to 6 hours while awake) is usually sufficient to control the ocular disease.

Endophthalmitis

Endophthalmitis is extensive inflammation within the eye from any cause. Most cases of endophthalmitis involve a breach in the eye wall (cornea and sclera), associated with either accidental trauma (incidence of approximately 5%) or surgical procedures (incidence of approximately <0.1%). The initial symptom is usually decreased vision followed by dull ocular pain. The initial sign is often evidence of inflammatory cells either within the aqueous (anterior uveitis) or within the vitreous (vitreitis). The cells can be seen only by slit lamp biomicroscopy. Common microbial organisms include toxin-producing gram-positive species and gram-negative species that are often associated with rapidly destructive course. Other organism of relatively low virulence, *Propionibacterium acnes* and *Staphylococcus epidermidis,* follow a more indolent course with less potential destruction. Metastatic endophthalmitis infection from a primary source outside of the eye is an unusual cause.

Diagnosis is established by sampling anterior chamber fluid or preferably vitreous fluid (vitreous tap) and evaluation of that fluid by Gram stain and culture. Treatment often requires outpatient intravitreal injection of antibiotics. In severe cases, the microbial and inflammatory debris burden is reduced by performing a vitrectomy through a pars plana incision. Under certain

TABLE 431-6

DISORDER	ACUTE OR CHRONIC	UNILATERAL OR BILATERAL	KEY SYMPTOMS	DEGREE OF INJECTION	DISCHARGE TYPE	OTHER FEATURES
Viral conjunctivitis	Acute	Bilateral, possibly asymmetrical	Itching, burning, soreness	4+	Watery	Preauricular lymphadenopathy
Bacterial conjunctivitis	Acute	Unilateral or bilateral	Burning	3+	Heavy, mucopurulent	Lids possibly adherent
Chlamydial conjunctivitis	Subacute, chronic	Usually unilateral	Burning, irritation	2+	Scant, mucopurulent	Usual occurrence in young, sexually active adults
Herpes simplex conjunctivitis	Acute	Unilateral	Photophobia, irritation	1-2+	None	Dendritic ulcer on the cornea or vesicles on the lid possible
Allergic conjunctivitis	Chronic	Bilateral	Itching	2+	Stringy, mucoid	Usual occurrence in atopic persons, possible seasonal symptoms
Blepharitis	Chronic	Bilateral	Itching, burning, foreign body sensation	1-2+	Usually none	Inflammation and crusting of lid margins
Dry eye	Chronic	Bilateral	Foreign body sensation	1+	Mucoid in severe cases	Punctate fluorescein staining of the cornea

Adapted from Palay DA, Krachmer JH. *Primary Care Ophthalmology,* 2nd ed. Philadelphia: Elsevier Mosby; 2005.

FIGURE 431-16. Eyelid abscess. Preseptal cellulitis, commonly resulting from minor penetrating trauma, may evolve into an abscess. Treatment requires incision and drainage followed by systemic antibiotics.

circumstances, corticosteroids and antibiotics are injected simultaneously to minimize the irreversible destructive effects of inflammation to the retina.

Allergic Conjunctivitis

Allergic conjunctivitis (Table 431-6) is commonly associated with atopy, hay fever, and allergic rhinitis (Chapter 259). Itching, a foreign body sensation, and a watery discharge are common. Treatment includes cool compresses and topical vasoconstrictors or antihistamines (e.g., naphazoline drops, four times daily during the allergic season, or levocabastine drops, four times daily). Long-term treatment with mast cell stabilizers (e.g., pemirolast drops, four times daily during the allergic season) or the combination of an antihistamine plus a mast cell stabilizer (e.g., olopatadine drops, two times daily during the allergic season) can be extremely effective in treating chronic symptoms.

INFECTIOUS EYE DISORDERS

Cellulitis

Preseptal cellulitis (Fig. 431-16) is soft tissue inflammation of the eyelid anterior to the orbital septum. The orbital septum divides the soft tissues of the eyelid from the soft tissues of the orbit. Orbital tissue is more susceptible to damage by the inflammation than is the preseptal tissue.

The clinical signs of preseptal cellulitis include soft tissue swelling, hyperemia, and conjunctival chemosis (edema). Movement of the eye is not restricted. Extension of inflammation posterior to the orbital septum is indicated by proptosis of the globe and ophthalmoplegia (restricted motion). Treatment of preseptal cellulitis includes oral antibiotics (e.g., amoxicillin-clavulanate, 500 mg orally every 8 hours for 10 days). Treatment of orbital cellulitis, which can lead to septic optic neuritis, intracranial spread, and cavernous sinus thrombosis, may require intravenous antibiotics and surgical drainage of a paraorbital abscess.

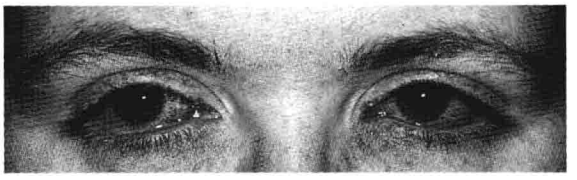

FIGURE 431-17. Diffuse injection of the conjunctiva with a watery discharge is evident in this case of viral conjunctivitis. (From Palay DA, Krachmer JH. *Primary Care Ophthalmology,* 2nd ed. Philadelphia: Elsevier Mosby; 2005.)

Adenoviral Conjunctivitis

Viral conjunctivitis (Fig. 431-17) is common (see Table 431-6), and adenoviral conjunctivitis (especially subtypes 7, 11, and 18) is the most common type. The condition is highly contagious through direct contact or inhalation of respiratory particles. After an incubation period of 5 to 15 days, the patient presents with very red eyes (see Table 431-4), itching, burning, a foreign body sensation, and often a discharge and ocular discomfort (see Table 431-3), which persist for 5 to 15 days. Preauricular lymphadenopathy may be present, and a history of upper respiratory tract infection is common. The disease is self-limited, and treatment is aimed at patients' comfort. Cool compresses are often soothing. Patients are advised to wash their hands frequently. Topical antibiotics are not required, and topical corticosteroids are contraindicated.

Bacterial Conjunctivitis

Fewer than 5% of cases of conjunctivitis are caused by bacteria, mostly *Staphylococcus, Haemophilus,* or *Streptococcus* species. Patients have a mucoid or purulent discharge (Fig. 431-18), often with crusting and edema of the conjunctiva (chemosis) and lids. Bacterial conjunctivitis responds to broad-spectrum antibiotic solutions or ointments (e.g., topical erythromycin ointment three times daily for 2 weeks) (Table 431-7).

Chlamydial Conjunctivitis

Adult inclusion conjunctivitis is a chronic conjunctivitis caused by sexual transmission of *Chlamydia trachomatis* (Chapter 326). Patients often have preauricular lymphadenopathy. Oral erythromycin (500 mg orally, four times daily for 7 days) or azithromycin (1 g orally twice daily for 7 days) is required. Trachoma, which is a chronic cicatricial conjunctivitis after repeated chlamydial infection (Chapter 326), is the world's leading cause of corneal blindness. It causes an entropion, inversion of the eyelashes (trichiasis), corneal vascularization, and opacification. Topical erythromycin or tetracycline, twice daily for 3 to 4 weeks, can be effective, but surgical epilation or eyelid reconstruction may be required.

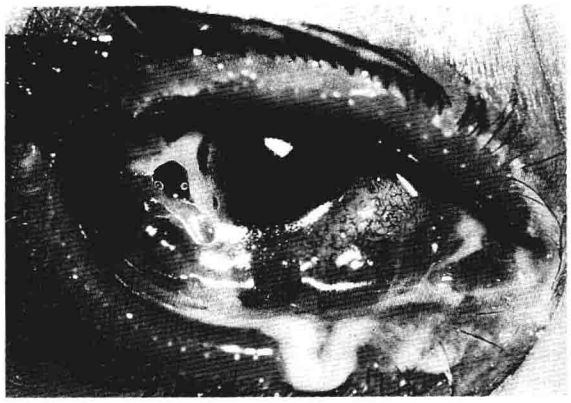

FIGURE 431-18. Bacterial conjunctivitis. Purulent discharge and conjunctival hyperemia suggest bacterial conjunctivitis. Viral conjunctivitis produces watery discharge, foreign body sensation, preauricular lymphadenopathy, and conjunctival follicles seen on slit lamp examination. (Reproduced with permission from the American Academy of Ophthalmology.)

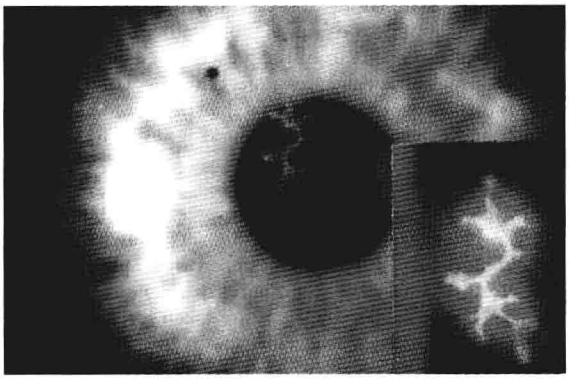

FIGURE 431-19. Herpes simplex corneal epithelial keratitis in diffuse light and in light passed through a cobalt blue filter after fluorescein staining *(inset)*. Note the dendritic staining pattern characteristic of herpes simplex.

TABLE 431-7 TOPICAL ANTIBIOTICS FOR EYE INFECTIONS

DRUG	TYPE	CONCENTRATION	DOSE
Moxifloxacin	Drops	0.5%	1 drop 4 times daily
Gatifloxacin	Drops	0.5%	1 drop 4 times daily
Ciprofloxacin	Drops	0.3%	1 drop 4 times daily
Gentamicin	Drops	0.3%	1 drop 4 times daily
Ofloxacin	Drops	0.3%	1 drop 4 times daily
Bacitracin	Ointment	500 U/g	Put in eye 4 times daily
Tobramycin	Ointment	0.3%	Put in eye 4 times daily
Erythromycin	Ointment	0.5%	Put in eye 4 times daily

Herpes Simplex Keratitis

Herpes simplex keratitis is the most common cause of central corneal ulcer (Fig. 431-19). Herpes simplex virus also can cause vesicular eyelid dermatitis. Initially, the main signs of primary herpes simplex keratitis are a red eye and a corneal epithelial dendritic ulcer. With appropriate antiviral therapy (e.g., trifluridine drops every 2 hours for 1 to 2 weeks), the keratitis usually heals without scarring. Recurrent herpes simplex keratitis may be precipitated by fever, menses, sunlight, irradiation, or stress. With recurrence, the disease may extend into the corneal stroma and cause a red eye, ocular discomfort, blurred vision, and corneal scarring. The treatment of stromal involvement is multifactorial and may not be successful. Corneal transplantation may be needed.

Herpes Zoster Ophthalmicus

Herpes zoster ophthalmicus (shingles, Chapter 383) has a propensity to involve one or more branches of the trigeminal nerve. The virus also can affect the uveal tract and, in immunosuppressed patients, the retina (i.e., acute retinal necrosis). When the trigeminal nerve is involved, spread to the inside of the eye (uveitis) is most likely if vesicles are present in the inner corner of the eyelids or on the nose, especially the tip of the nose. If the uvea is not involved, the skin lesions heal with some scarring but no long-term effects. In patients with moderate to severe skin involvement, treatment can be started with oral acyclovir (800 mg orally five times per day for 7 to 10 days). If uveitis develops, the treatment (e.g., prednisolone acetate drops 1% four times daily and scopolamine drops 0.25% three times daily) can be extended and difficult.

Pseudomonal and Gonococcal Keratitis

Keratitis, which is inflammation of the corneal stroma, can be caused by spread of pathogens internally from a corneal ulcer. *Pseudomonas aeruginosa* (Chapter 314), which causes a particularly virulent keratitis, is the most common gram-negative pathogen and is especially common in wearers of contact lenses. To avoid internal spread, urgent treatment is necessary (e.g., fortified tobramycin or gentamicin, 1.5 mg/mL every hour, alternating with fortified cefazolin, 50 mg/mL every hour, so a treatment is given each one-half hour around the clock). The dosage and duration of the treatment depend on the response.

Another gram-negative cause of a virulent keratitis is *Neisseria gonorrhoeae* (Chapter 306). Corneal infection is accompanied by copious weeping and a characteristic hyperpurulent discharge. Prompt treatment is essential in preventing corneal perforation.

Cytomegalovirus Retinitis

Cytomegalovirus retinitis (Chapter 384) is unusual except in immunosuppressed patients, especially patients with human immunodeficiency virus infection. Clinically, a central retinochoroiditis is seen. The presumptive diagnosis is made on the characteristic intense, retinal, wedge-shaped reaction, with considerable exudates and hemorrhages, giving the terms "pizza pie retinitis" and "hemorrhagic cottage cheese retinitis" to the entity. Treatment is antiviral drugs: ganciclovir (5 mg/kg intravenously twice daily, two to three times per week), foscarnet (90 mg/kg intravenously twice daily, twice per week), or cidofovir (5 mg/kg intravenously, weekly for 3 weeks) with follow-up maintenance.

Acanthamoeba Keratitis

Acanthamoeba species (Chapter 360) can cause a severe, blinding keratitis. Contact lens wearing is a major risk factor. A characteristic stromal ring infiltrate develops, and uveitis may occur. Using a confocal microscope, the acanthamebic parasite can be observed clinically as a pear-shaped cyst (11 to 15 μm). Numerous treatment protocols exist (e.g., polyhexamethyl biguanide 0.02% drops every hour). The duration and dose depend on the response, but the ideal treatment is not yet in hand.

Toxoplasmic Retinitis

Toxoplasma gondii (Chapter 357) causes both a congenital and acquired retinochoroiditis, which is more common in immunosuppressed patients. The lesions begin as an acute retinitis that atrophies centrally and pigments peripherally as it heals. The protozoa are found both in free and encysted forms within the retina. The condition may be self-limited and diagnosed as a healed incidental finding that does not need treatment. When active lesions are in the macula or a severe vitreitis causes at least a two-line decrease in vision, 4 to 6 weeks of quadruple therapy (pyrimethamine, 200 mg oral loading dose then 25 mg orally daily; folinic acid, 10 mg orally every other day; sulfadiazine, 2 g oral loading dose then 1 g four times daily; and oral corticosteroids, e.g., prednisone, 20 to 60 mg orally daily beginning at least 24 hours after antibiotic therapy is started and tapered 10 days before stopping antibiotics) usually produces good results. Alternative regimens may include clindamycin (150 to 450 mg orally three to four times daily) or atovaquone (1 g oral loading dose then 500 mg daily).

Fungal Endophthalmitis

Endophthalmitis is a potentially disastrous infection of the inside of the eye, often leading to blindness. The main fungal causes are *Candida, Coccidioides,* and *Aspergillus* species, which can gain access inside the eye either by traumatic introduction or through hematogenous spread. The patient presents with a red eye, ocular pain, and decreased vision. Multiple vitreous abscesses tend to be caused by fungi, whereas a solitary abscess is more likely caused