Management of

Ocular Emergencies

7th Revised and Updated Edition

Raymond Stein, MD, FRCSC
Rebecca Stein, MD, FRCSC

Mediconcept

Library and Archives Canada Cataloguing in Publication

Stein, Raymond M., author
Management of ocular emergencies / Raymond Stein, MD, FRCSC,
Rebecca Stein, MD, FRCSC --7th revised edition.

Includes index.
ISBN 978-1-896825-33-5 (paperback)

1. Ophthalmologic emergencies--Handbooks, manuals, etc. 2. Ophthalmologic emergencies. 3. Eye-Diseases I. Stein, Rebecca, author II. Title.

RE48 9.S83 2016 617.7'1 C2016-901611-0

Applications and usage
The authors and publisher have exerted every effort to ensure that the applications and use of all drugs, devices, and procedures mentioned in this publication are in accord with current recommendations and practices. However, in view of ongoing research, changes in regulations, and the constant flow of information relating to ophthalmology, the reader is cautioned to consult the package insert of any product for the approved indications and dosage recommendations in Canada as well as for the incidence of adverse reactions and mortality.

The following are the respective registered products of:
Abbott Medical Optics: Blink® Tears; **Alcon:** Azarga®, Azopt®, Betoptic® S, Bion® Tears, Ciloxan®, Duotrav®, Flarex®, Iopidine®, Isopto® Carpine, Isopto® Carbachol, Maxidex®, Mydriacyl®, Nevanac®, Pataday®, Systane®, Systane® Preservative-Free, Systane® Ultra, Tears Naturale® P.M., Tobradex®, Tobrex®, Travatan Z®, Vigamox®, Voltaren®; **Allergan:** Acular®, Acuvail®, Alocril®, Betagan®, Lumigan®, Pred Forte®, Propine®, Refresh® Tears, Restasis®, Zymar®; **Bausch + Lomb:** Alrex®, Besivance®, Liposic®, Lotemax®, PreserVision®; **Baxter:** Osmitrol®; **Bristol-Myers Squibb:** Coumadin®; **CandorVision:** Hylo®; **Eli Lilly:** Ceclor®; Keflex®; **Merck:** Indocin®, Timoptic®, Trusopt®, Zostavax®; **Pfizer:** Viroptic®, Xalatan®; **Sanofi:** Brolene®; **TearLab:** TearLab® Osmolarity System; **TearScience:** Lipiflow®; **Wyeth:** Diamox®, Diamox® Sequels, Phospholine Iodide®; **Xanodyne:** Amicar®

Prepared and printed in Montreal Canada by:

Les Editions Optomed under license by Mediconcept.
421-5885 Marc Chagall,
Cote St Luc, Quebec
H4W 0C1 Canada
ISBN 978-1-896825-33-5

Contents

Introduction

This manual is designed to be a practical guide to the management of ocular emergencies, and presents the clinical principles used in our everyday ophthalmic practice. The material arose from a series of lectures given for emergency room physicians, medical students, and ophthalmology residents, and was enthusiastically received for its simplified approach and organization.

An attempt has been made to organize the material into clinically relevant sections. The first section highlights the essentials of the eye examination. The next section deals with emergency ocular diseases and is divided into those conditions in which the patient presents with either a red or white eye. The red eye conditions are those that may be non- traumatic or secondary to trauma; the white eye conditions are those that are associated with a decrease in vision or diplopia. The final section contains a series of appendixes that may be helpful for the differential diagnosis of a variety of emergency cases.

This seventh edition has been completely updated, which is a reflection of medical advances in diagnostic techniques and therapeutics. In addition, the color plates have been expanded in number and highlight the ocular conditions described in the text.

We dedicate this most recent edition of the book as a tribute to the memory of Dr. Harold Stein, a dedicated advocate of ophthalmic education through literature and scientific discourse. He approached patient care with a smile on his face and encouraging words.

We hope that this manual will serve as a practical guide to the diagnosis and management of ocular emergencies.

Raymond M. Stein, MD, FRCSC
Professor of Ophthalmology and Vision Sciences, University of Toronto
Medical Director, Bochner Eye Institute

Rebecca L. Stein, MD, FRCSC
Cornea and Refractive Surgery Specialist

Bochner Eye Institute
Toronto, ON, Canada

Ocular History and Examination

Editor's Note

A systematic approach to the history and examination of the ocular emergency patient allows for a correct diagnosis. Each clinical symptom and sign provides important clues to the underlying condition. Is there a history of trauma or did the condition appear spontaneously? Is this a recurrent problem? Is there a history of decreased vision, redness, pain, photophobia, tearing, itching, burning, diplopia, or floaters? The ocular history is important for establishing a provisional diagnosis. With a careful examination all the clinical signs can be detected. Is the vision affected? Are the pupils equal and reactive? Is there an afferent pupillary defect? Is there a full range of extraocular muscle movements? Is there evidence of a field defect on confrontation testing? Are there any abnormalities of the lids, conjunctiva, lacrimal puncta or sac, cornea, anterior chamber, iris, or lens? Are there any abnormalities of the vitreous, optic disc, retinal arteries or veins, macula, or peripheral retina? Is the intraocular pressure high? This chapter describes the important steps in the evaluation of the ocular emergency patient.

Introduction

The type and characteristics of the presenting symptoms can often suggest a provisional diagnosis prior to the examination. The nature of the symptoms should be recorded, including any precipitating factors, whether the episode is recurrent, constant, or intermittent, and whether the onset was gradual or acute. Common symptoms include decreased vision, redness, photophobia, tearing, itching, foreign body sensation, burning, pain, diplopia, and floaters.

A brief medical history should be obtained. A variety of systemic diseases can affect the eye, including diabetes mellitus, hypertension, thyroid disease, rheumatoid arthritis, and cancer (Appendixes A,C). All medications should be documented, as certain systemic medications can cause ocular complications (Appendix B). Drug allergies should be determined before eye drops are instilled or medications prescribed. Any family history of ocular diseases should be recorded.

An examination should then be conducted, and will usually include a test of visual acuity, pupils, motility, confrontation visual field, the anterior segment, the posterior segment, and intraocular pressure (Figs. 1-3, Plate 1).

Visual Acuity

Check the distance visual acuity (VA) for each eye. Vision measurement is crucial for proper diagnosis, management, and medical/legal documentation. If a patient is

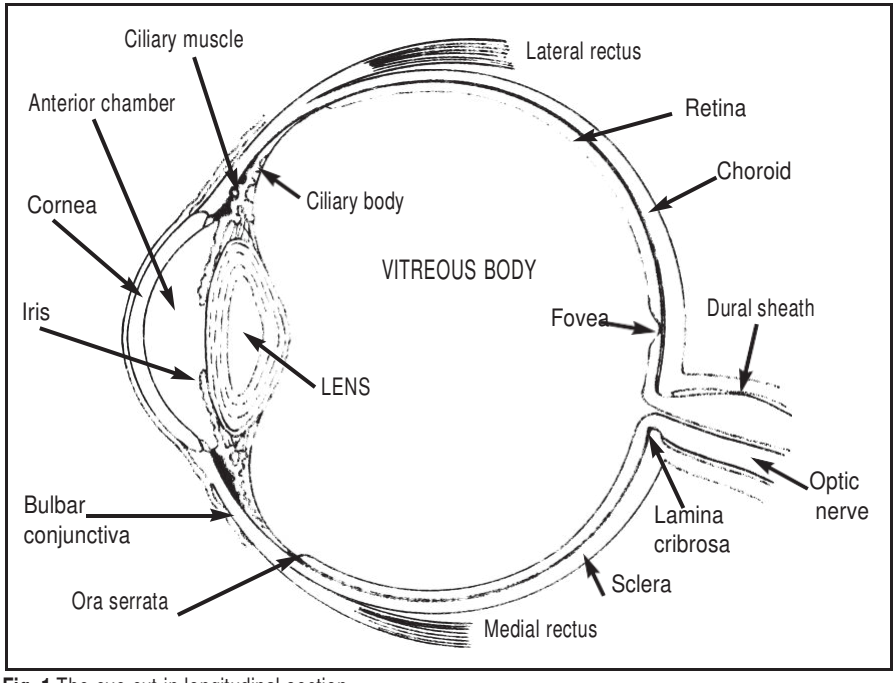


Fig. 1 The eye cut in longitudinal section

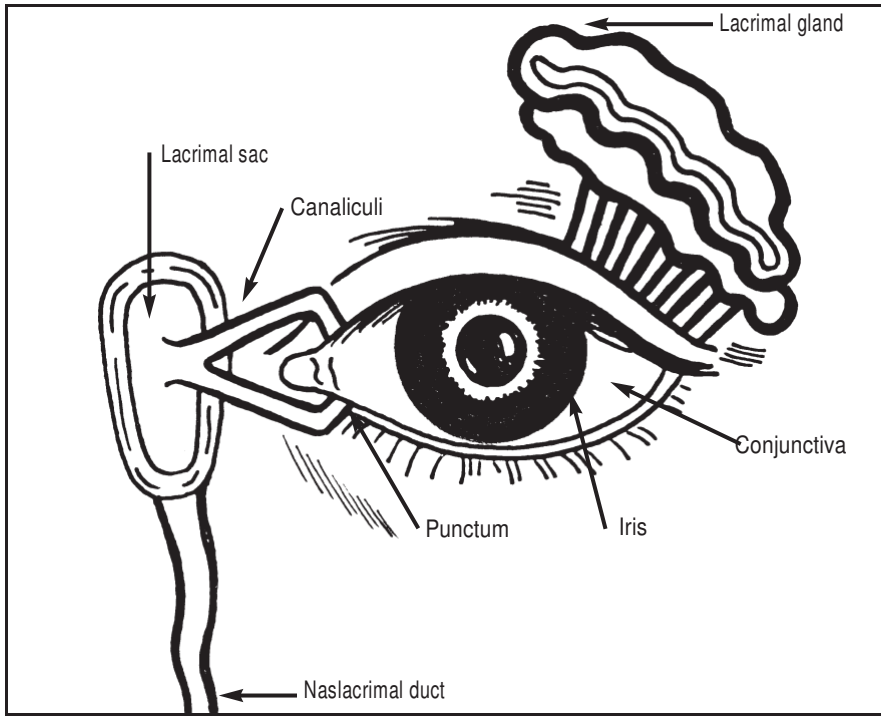


Fig. 2 Lacrimal apparatus. Tears produced by the lacrimal gland are drained through the punctum, lacrimal sac, and nasolacrimal duct into the nose.

unable to open his or her eye because of pain, a drop of topical anesthetic will usually reduce the pain enough to obtain a visual acuity.

Distance VA is usually checked at 20 feet (6 meters) using letters, numbers, or an illiterate E chart. In order of best to worst vision, acuities recorded are as follows: 20/15, 20/20, 20/25, 20/30, 20/40, 20/50, 20/60, 20/70, 20/80, 20/100, 20/200, 20/400, counting fingers, hand movements, light perception, and no light perception. The patient may view the visual acuity chart with both eyes either intentionally or unintentionally, if the examiner does not take care to see that one eye is completely occluded. If a patient has a VA of 20/60, this means that he sees at 20 feet what a person with normal vision sees at 60 feet. Similarly, a VA of 20/15 means that he sees at 20 feet what a person with normal vision sees at 15 feet. A patient will often read additional smaller letters on the chart with encouragement by the examiner, thereby increasing the accuracy of the examination data. For preschool-aged children, the "illiterate E" chart is used by having the child indicate the direction in which the legs of the "E" are pointing. Near vision is usually checked with a reading card held at 14 inches. This is the most convenient way to check vision in the hospitalized patient. The following are common abbreviations used to discuss visual acuity: OD (oculus dexter): right eye; OS (oculus sinister): left eye; OU (uterque): both eyes.

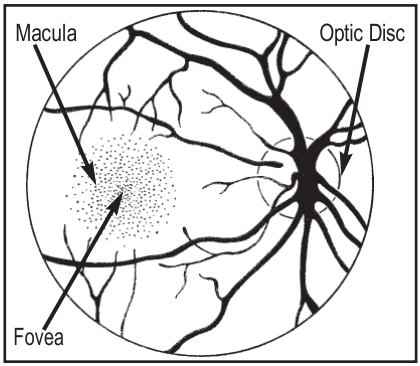


Fig. 3 Fundus diagram

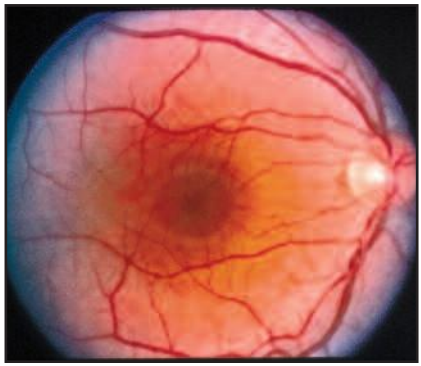


Plate 1 Normal fundus which shows the optic disc, blood vessels, macula, and fovea

If the patient has corrective lenses, they should be worn during testing. The examiner must try to determine optimum acuity. If the vision is less than 20/20,the potential for improved vision should be ascertained by having the patient look through a pinhole. Improved vision with a pinhole indicates that appropriate glasses or contact lenses would be beneficial; unimproved vision suggests that a non-refractive problem exists such as corneal edema, cataracts, a macular disorder,or an optic nerve problem. Visual acuity should be checked in each eye since some patients are unaware of an amblyopic eye. If the good eye of such a patient is patched, he may be at serious risk of a motor vehicle accident if he were to drive.

In estimating visual acuity in the uncooperative patient, withdrawal, or a change in facial expression in response to light or sudden movement indicates the presence of vision. A brisk pupillary response to light also suggests the presence of vision. The exception to this is the patient with cortical blindness, which is due to bilateral widespread destruction of the visual cortex. If there is any doubt, referral to an ophthalmologist is recommended.

Normal acuity does not ensure that significant vision has not been lost, since the entire visual field including peripheral vision must be considered. For instance, a patient who has lost all of the peripheral vision to one side — homonymous hemianopia — generally has normal visual acuity.

Pupils

The pupillary size and reaction to light stimulation should be checked, carefully noting the presence of a dilated or constricted pupil. The swinging flashlight test is used to determine the absence or presence of an afferent pupillary defect. It should be tested in all cases of decreased vision and head or eye trauma. Pupillary shape gives an indication of an eye's response to trauma. Eccentricity of pupillary shape after trauma can indicate serious ocular damage. A peaked or teardrop-shaped pupil may indicate a ruptured globe.

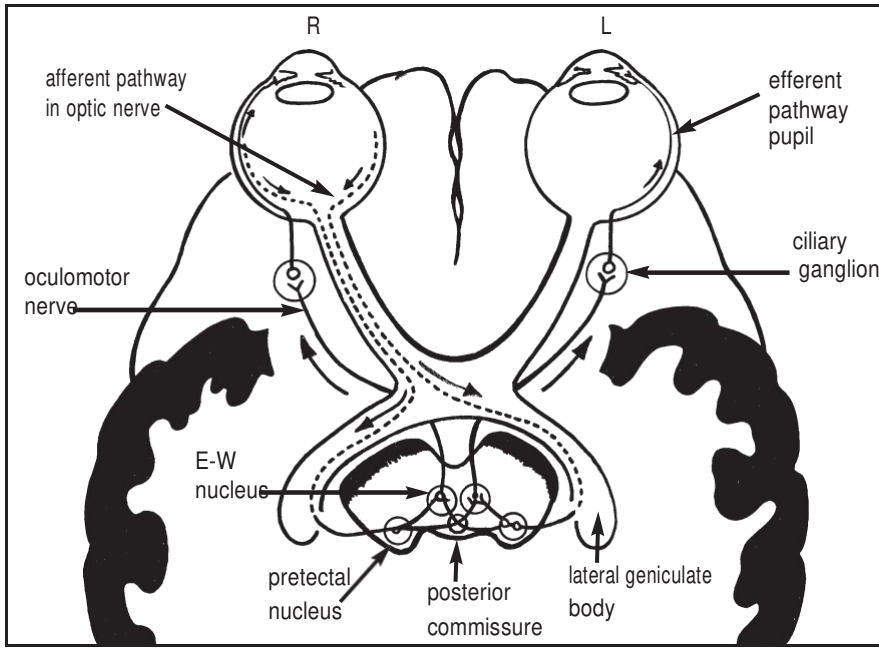


Fig. 4 Pupillary pathways

Figure 4 shows a cross section of the pupillary pathways. The solid line represents the efferent pathway, and the broken line represents the afferent pathway. Light stimulation of the left retina will result in impulses which travel up the left optic nerve and divide at the chiasm. Some impulses continue up to the left tract; some crossings continue up to the right tract. The nerve impulses arrive at each pretectal nucleus and stimulate cells which in turn send impulses down the third cranial nerve to the iris sphincter causing each pupil to constrict. It is because of the double decussation, the first in the chiasm and the second between the pretectal nuclei and the Edinger-Westphal nuclei, that the direct pupil response in the left eye equals the consensual response in the right eye.

Swinging Flashlight Test

During the swinging flashlight test the examiner projects the light on the right eye (for example), allowing the right pupil to constrict to a minimum size and subsequently escape to an intermediate size. The light is then quickly swung to the left eye, which constricts from an intermediate to a minimum size, subsequently escaping to an intermediate size. At this point the light is swung again to the right eye and a mental note is made of the intermediate (starting) pupil size and briskness of the response to light. These characteristics should be exactly the same in both eyes as the light is alternately swung to each eye.

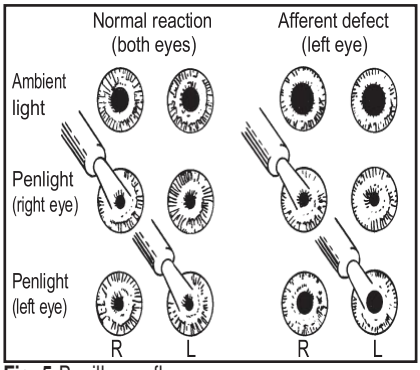


Fig. 5 Pupillary reflexes

Table I Extraocular muscle innervation

Innervation	Muscle	Primary Action
3rd nerve	superior rectus	up
3rd nerve	medial rectus	in
3rd nerve	inferior rectus	down
3rd nerve	inferior oblique	up and in
4th nerve	superior oblique	down and in
6th nerve	lateral rectus	out

Afferent Pupillary Defect (Marcus-Gunn Pupil)

The swinging flashlight test will determine if the amount of light transmitted from one eye is less than that carried via the fellow eye; when the light is swung to the defective eye, immediate dilatation of the pupil occurs instead of the normal initial constriction. This characterizes an afferent pupillary defect (Fig. 5). The differential diagnosis includes a retinal detachment, occlusion of a central retinal artery or vein, optic neuritis, and optic neuropathy.

N.B.: Cataract, hyphema, vitreous hemorrhage, corneal ulcer, and iritis are associated with a decrease in vision, but are *not associated with an afferent pupillary defect.*

Differential Diagnosis of a Dilated Pupil

A dilated pupil may be due to third nerve palsy, trauma, Adie's pupil, acute glaucoma, or may be drug-induced.

Third Nerve Palsy. If the dilated pupil is fixed, the cause may be third nerve palsy. This condition may be associated with ptosis and a motility disturbance, characterized by the eye being deviated out and down. The pupil responds to constricting drops, e.g., pilocarpine. This is a neurosurgical emergency, as the possibility of an intracranial mass lesion must be ruled out.

Trauma. Damage to the iris sphincter may result from a blunt or penetrating injury. Iris transillumination defects may be visible with the ophthalmoscope or slit lamp, and the pupil may have an irregular shape.

Adie's Pupil. The pupil responds better to near stimulation than to light. The condition is thought to be related to aberrant innervation of the iris by axons which normally stimulate the ciliary body.

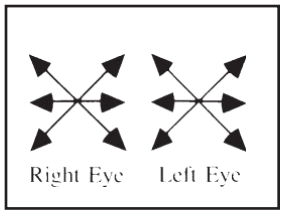


Fig. 6 Method for examining and recording ocular motility

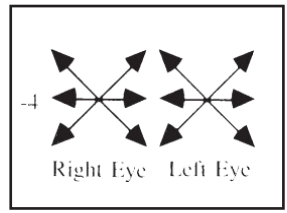


Fig. 7 Record of a sixth nerve palsy of the right eye

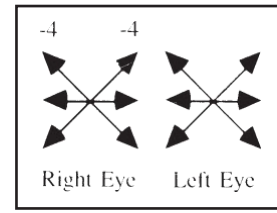


Fig. 8 Record of a right orbital blowout fracture and limited upgaze

Drug-Induced. Iatrogenic or self-contamination may occur with a variety of dilating drops, e.g., cyclopentolate hydrochloride (Cyclogyl®), tropicamide (Mydriacyl®), homatropine, scopolamine, and atropine. The pupil is fixed and dilated and, unlike in third nerve palsy, does not respond to constricting drops.

Acute Glaucoma. The patient may complain of pain and/or nausea and vomiting. The eye is red, the vision is diminished, the intraocular pressure is elevated, and the pupil is mid-dilated and poorly reactive.

Differential Diagnosis of a Constricted Pupil

A constricted pupil occurs in Horner's syndrome, iritis, and may be drug-induced.

Horner's Syndrome. Other signs of this condition include mild ptosis of the upper lid and retraction of the lower lid. The difference in pupillary size is more notable in dim light since adrenergic innervation to the iris dilator muscle is diminished.

Drug-Induced. Iatrogenic or self-induced pupillary constriction may be due to a variety of drugs, including pilocarpine, carbachol, and echothiophate iodide (Phospholine Iodide®).

Iritis. Slit-lamp examination shows keratic precipitates and cells in the anterior chamber, and there is a prominent ciliary flush. The intraocular inflammation stimulates pupillary constriction.

Motility

There are six extraocular muscles in each eye that are innervated by a total of three nerves. The action of a specific muscle can vary depending on the position of the eye when it is innervated. Table I shows the general relationships which apply. Trauma to the muscles and/or cranial nerves serving the muscles can result in asymmetric movement of the eyes resulting in double vision when both eyes are open.

The examiner should determine the range of ocular movements in all gaze positions (Fig. 6). Limited movement in any gaze position can be documented as –1 (minimal), –2 (moderate), –3 (severe), or –4 (total). For example, a patient with a complete right sixth nerve palsy can be recorded as shown in Figure 7. Figure 8 shows a patient with a blow-out fracture to the right orbit with entrapment of the inferior rectus muscle and limitation of upgaze.

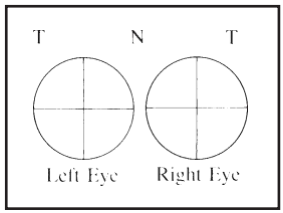


Fig. 9A A normal gross visual field test. T = temporal field; N = nasal field

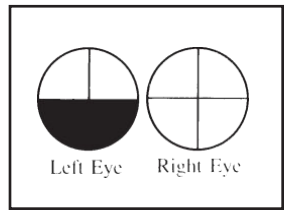


Fig. 9B An inferior field defect of the left eye; the right is normal.

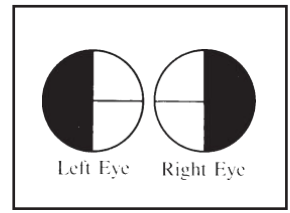


Fig. 9C A complete bitemporal visual field defect

Confrontation Visual Fields

A visual field defect may be caused by a disturbance of any of the neurologic pathways for light transmission. This includes the retina, optic disc, optic nerve, optic chiasm, optic tract, optic radiations, and occipital cortex.

A screening test for gross visual field loss can be performed as follows:

1. One eye of the patient is covered.
2. With the uncovered eye the patient must maintain fixation, e.g., on the tip of the examiner's nose.
3. The examiner randomly projects fingers in any quadrant, while standing 3 or 4 feet from the patient. For the detection of more subtle defects such as in optic neuritis, it is best to stand 10 to 20 feet away from the patient. The further one is from the patient, the greater the size of the scotoma.
4. The patient counts the projected fingers.

A normal response would be recorded as shown in Figure 9A. A patient with a retinal detachment of the superior retina would have an inferior field defect (Fig. 9B). A patient with a pituitary tumor may present initially with visual loss and a complete bitemporal defect (Fig. 9C). A more detailed evaluation of the visualfield requires an automated machine. The target size and luminosity can be varied, and the patient's response documented on a computer-generated printout.

Anterior Segment

Examination of the anterior segment should include an assessment of the lids, orbit, puncta, conjunctiva, sclera, cornea, anterior chamber, and lens (Fig. 10). In all suspected infectious cases, cotton swabs are used or gloves are worn to depress or elevate lids. This protects the examiner from contamination. Be careful not to put any pressure on the globe, especially in traumatic cases in which there is a possibility of an ocular perforation.

Examination of the orbit is important in trauma and in cases of periorbital swelling and erythema. In cases of trauma the orbital exam should include:
1. palpation for subcutaneous emphysema (air in subcutaneous tissues causing crepitus); 2. testing for localized areas of anesthesia (especially in the inferior orbit and cheek area); 3. palpation for defects in the orbital rim; 4. documenting the presence of proptosis or enophthalmos.

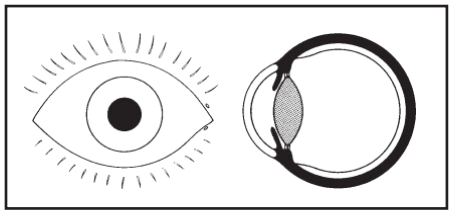


Fig. 10 Examination of the anterior segment includes an assessment of the lids, puncta, conjunctiva, sclera, cornea, anterior chamber, and lens

In a red eye, the conjunctiva should be examined to differentiate conjunctival hyperemia (blood vessel engorgement), subconjunctival hemorrhage (blood beneath the conjunctiva), a ciliary flush (injection of vessels around the cornea), or a combination of these. The examination should also check for the presence of conjunctival discharge, which, if detected, should be categorized as to its amount (profuse or scant) and character (muco-purulent or serous).

Pre-auricular lymph node enlargement and/or tenderness is frequently a sign of viral conjunctivitis. Usually such enlargement does not occur in acute bacterial conjunctivitis.

In a patient who presents with a chronic conjunctivitis, one should rule out the possibility of a lacrimal system obstruction. Pressure on the lacrimal sac may produce the reflex of a mucous or purulent discharge from the puncta.

Fluorescein stain can be used to detect de-epithelialized surfaces, e.g., corneal abrasions, erosions, dendrites, and ulcers; epithelial defects will stain green. Fluorescein is applied in the form of a sterile filter paper strip that is moistened with a drop of an artificial tear or saline solution and then touches the conjunctiva. A few blinks spread the fluorescein over the cornea. Viewing the eye under a cobalt blue light enhances the visibility of the green fluorescence. Fluorescein staining of the cornea should be assessed in all cases of "red eye" and trauma. Two precautions to keep in mind when using fluorescein are: 1. Use fluorescein-impregnated strips instead of stock solutions of fluorescein because such solutions are especially susceptible to contamination, with *Pseudomonas* species, and 2. Have the patient remove his or her contact lenses prior to application to avoid discoloration. Rose bengal stain also detects de-epithelialized surfaces, and in addition stains devitalized cells, as are found in the conjunctiva in keratitis sicca or chemical toxicity.

Superficial foreign bodies may be hidden under the inner surface of the upper lid or high in the cul-de-sac (formed by the junction of upper lid and conjunctiva of the globe). When a superficial keratitis occurs without evidence of a foreign body, the upper lid should be everted. To evert the upper lid the patient is asked to look downward and the examiner grasps the eyelashes of the upper lid between the thumb and the index finger. A cotton tip applicator may be used to press gently downward over the superior aspect of the tarsal plate as the lid margin is pulled upward by the lashes. Pressure is maintained on the everted upper lid while the patient is encouraged to keep looking downward. To return the lid to its normal position, the examiner releases the lid margin and the patient is instructed to look upward. Never perform this maneuver in the setting of a potentially ruptured globe.

The depth of the anterior chamber can be assessed. When the anterior chamber is shallow the iris is bowed forward over the lens. Under these conditions, the nasal iris is seen in shadow when the light is directed from the opposite side (Fig. 11). As

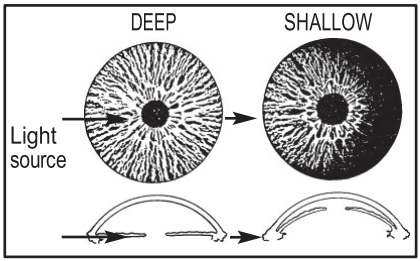


Fig. 11 Estimation of anterior chamber depth

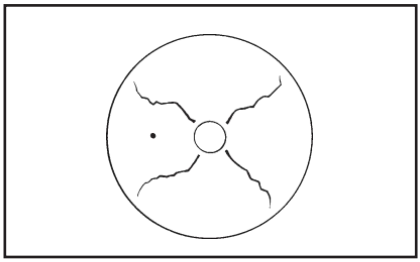


Fig. 12 Examination of the posterior segment includes an assessment of the vessels, macula, and disc as well as the vitreous and peripheral retina.

the shallowness of the anterior chamber increases, so does the shaded view of the nasal iris. A shallow anterior chamber may indicate:

1. a narrow angle that could close with pupillary dilation.
2. angle-closure glaucoma when associated with an elevation of intraocular pressure; or
3. traumatic ocular perforation or laceration.

Posterior Segment

Examination of the posterior segment should include an assessment of the vitreous, disc, vessels, macula, and peripheral retina (Fig. 12). Improved visualization of these structures is best appreciated through a dilated pupil. Dilation of the pupil should not be done in the following circumstances:

1. Do not dilate if assessment of anterior chamber depth suggests a shallow chamber and a narrow angle, because of the risk of precipitating angle-closure glaucoma.
2. Do not dilate if the patient has had a cataract extraction with implantation of an iris-supported intraocular lens. These implant lenses were popular over 35 years ago but are no longer used today. The dilation of the iris in these patients could result in a dislocated intraocular lens.
3. If the patient is undergoing neurological observation and pupillary signs are being followed (e.g., a head-injured patient), do not dilate until the neurologist or neurosurgeon thinks it is safe to do so.

Vitreous

The vitreous is a jelly-like substance located between the lens and retina. With aging, the vitreous shrinks and often pulls away from its attachments to the retina and disc. Tissue or cells may be displaced, causing the symptom of vitreous floaters, or vitreous movements against the retina may result in the experience of flashing lights.

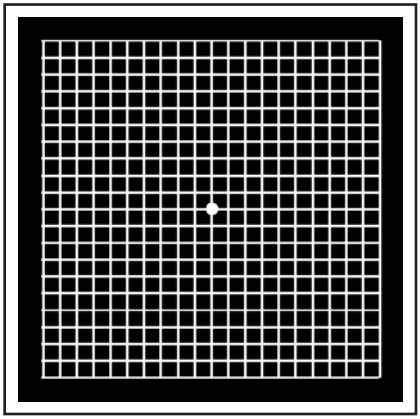


Fig. 13 Amsler grid testing. Shown here is the typical grid pattern of white lines against a black background.

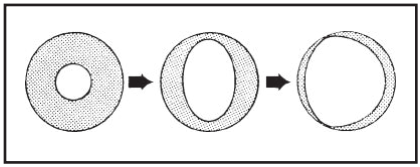


Fig. 14 Progressive cupping or increasing cup-to-disc ratio in the right eye in glaucoma.

Vessels

The retinal blood vessels are normally transparent, and their color is due to the blood. During arteriosclerosis the blood vessel wall becomes visible, progressing from a "copper-wire" appearance to a later "silver-wire" color. Where arterioles meet veins, a common sheath is found; thickening of the arteriole can cause indentation of the vein, or arteriovenous nicking. This can lead to thrombosis and vein occlusion.

Macula

The macula is the region of the retina responsible for central vision and color detection. In the center of the macula is a pit called the fovea which in young patients produces a well-defined reflex. If asymmetry in the foveal reflexes occurs in a patient with a visual disturbance, this suggests a retinal problem.

In addition to ophthalmoscopic examination, the macula can be evaluated by the Amsler grid test (Fig. 13). The test is carried out by having the patient look with one eye at a time at a central spot on a page with horizontal and vertical parallel lines making up a square grid pattern. The patient is asked to note irregularities in the lines. Irregularities may be reported as lines that are wavy, that seem to bow or bend, that are pure gray or fuzzy, or that are absent in certain areas of the grid. With the chart held at a normal reading distance of 30 centimeters from the eye, the Amsler grid measures 10 degrees on each side of fixation. This allows the entire macula to be evaluated.

Optic Disc

The normal optic disc is slightly oval in the vertical meridian and has a pink color due to capillary blood supply. There is a central depression on the surface of the disc called the physiologic cup. The size of the physiologic cup varies among individuals. The pigmented coats of the eye — the retinal pigment epithelium and the choroid — frequently fail to reach the margin of the optic disc, producing a hypopigmented crescent. Such crescents are especially common in myopic eyes on the temporal side of the optic disc. Conversely, an excess of pigment may be seen in some eyes, producing a heavily pigmented margin along the optic disc. The retinal nerve fibers are ordinarily nonmyelinated at the optic disc and retina, but, occasionally, myelination may extend onto the surface of the disc and retina, producing a dense, white, superficial opacification.

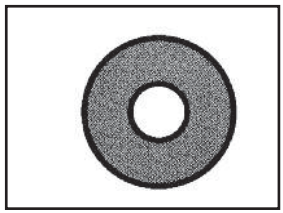


Fig. 15A The normal neural rim is pink

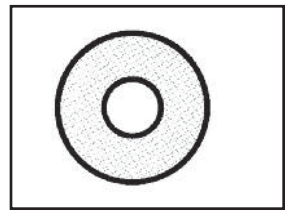


Fig. 15B the neural rim is pale in an old ischemic optic neuropathy.

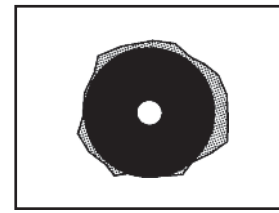


Fig. 16 Hyperemia of the disc and indistinct disc margins may occur in papilledema, optic neuritis, or ischemic optic neuropathy.

The optic disc should be evaluated as follows:

1. *The Cup-to-Disc Ratio.* Most normal patients have a cup-to-disc (C/D) ratio of less than 0.5. A higher C/D ratio or asymmetry between the discs is suggestive of glaucoma. Figure 14 shows progressive cupping in glaucoma.
2. *The Color of the Neural Rim.* A normal neural rim will have a pink color and should be similar in both eyes (Fig. 15A). In an old ischemic optic neuropathy the neural rim will be pale (Fig. 15B).
3. *Contour of the Disc Margins.* The disc margin contour should be distinct. In the acute stages of papilledema, optic neuritis, or ischemic optic neuropathy the disc may appear swollen and the margins indistinct (Fig. 16).

Intraocular Pressure

In an emergency case, the most important reason for checking intraocular pressure is to rule out acute angle closure glaucoma, in which condition the pressure is often greater than 40 mmHg. A pressure over 22 mmHg is generally considered above average and should prompt further investigation. The methods which can be used to check intraocular pressure include tactile tension, Schiotz tonometry, applanation tonometry, air-puff tonometry, and digital tonometry.

Tactile Tension

The pressure of the two eyes can be compared and approximated by digital pressure on the globes through closed lids. The eye of a patient with angle closure glaucoma will be firm compared to the opposite eye.

Schiotz Tonometry

Schiotz tonometry is an older technique and may be available in emergency departments. The patient is supine, a topical anesthetic is instilled in the eyes, and the lids are separated, with care being taken to avoid putting pressure on the globe. The tonometer is used initially with a 5.5-gram weight in place. The tonometer is held as perpendicular as possible and is gently lowered onto the central cornea for a few seconds to determine the scale reading. The less the corneal indentation, the

lower the scale reading and the higher the intraocular pressure. If the scale reading is 3 units or less for either eye, the measurement should be repeated. Add weights to the 5.5-gram weight to a total of 7.5, 10.0, or 15.0 grams as necessary to reach the scale reading in the range of 3.5 to 8.0 units. Refer to the calibration scale for Schiotz tonometers to determine the intraocular pressure for a particular plunger load (Appendix D).

Carefully clean the tonometer with an alcohol swab after each use to prevent transmission of disease such as viral conjunctivitis to other patients. The tonometer should not be re-used until it is dry; otherwise a chemical keratoconjunctivitis can be induced.

Applanation Tonometry

The equipment for this test is not as readily available as the Schiotz tonometer. It is a slit-lamp attachment that takes more experience to master, but results in a more accurate measurement.

Air-Puff Tonometer

The air-puff tonometer is an expensive instrument which is not readily available. It does not require use of a topical anesthetic but is not as accurate as applanation or digital tonometry.

Digital Tonometry

An easy-to-use, expensive portable device (Tono-Pen) that applanates a small surface of the cornea of 1.5 mm. A digital reading is obtained. This device is advantageous when dealing with irregular or scarred corneas. It has become a popular device with ophthalmologists and recently has been introduced in many emergency departments.

Nontraumatic Red Eye

Editor's Note

Conditions discussed in this section are those that present with a red eye without a history of trauma. These disorders may be secondary to an infection (bacterial, viral, chlamydia, fungal, or parasitic), inflammation, toxicity, allergy, hereditary or traumatic weakness of corneal epithelial attachments, or narrow anterior chamber angle. The clinician that follows a systematic history and examination will detect the important symptoms and signs leading to the correct diagnosis. The differential diagnosis of nontraumatic red eye conditions are listed in Appendix E. The primary clinician can often initiate treatment. In many cases ophthalmic referral is recommended to initiate treatment with antibiotics, antivirals, steroids, nonsteroidals, a bandage soft contact lens, or perform a peripheral YAG iridotomy in the case of angle closure glaucoma. In some situations a workup may be performed such as a corneal or conjunctival scraping for culture, a CT scan or MRI, or evaluation of possible underlying systemic disease.

Pre-Septal Cellulitis

Description

Pre-septal cellulitis is characterized by erythema and swelling of the eyelids (Plate 2). The infection is confined to the anterior structures of the periorbita.

Predisposing factors include a history of an upper respiratory tract infection, trauma to eyelids, bug bites, or an external ocular infection. It must be differentiated from an orbital cellulitis, which can result in a permanent loss of vision. Orbital cellulitis is most often caused by extension of infection from adjacent sinuses especially the ethmoid sinus. In pre-septal cellulitis the patient has normal vision, no proptosis, normal ocular motility, and no pain with eye movements. *Hemophilus influenzae* is the organism most commonly associated with this condition in children under five years of age, and *Staphylococcus aureus* and *Streptococcus sp.* in adults.

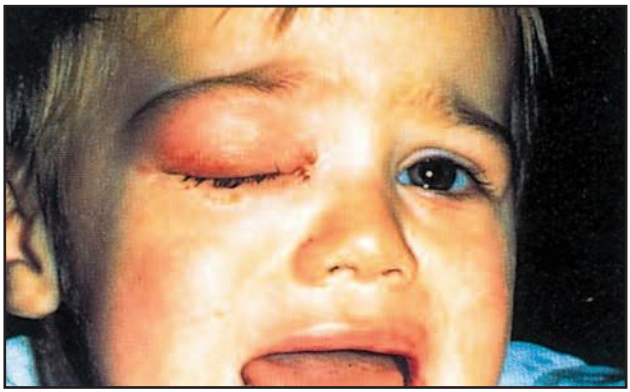


Plate 2
Lid swelling and erythema in a patient with preseptal cellulitis

Workup

- Cultures are obtained from the nasopharynx, conjunctiva, and blood.
- The patient should be examined by an ophthalmologist to rule out orbital involvement.
- If the patient is unable to cooperate for the examination, or if there is any suspicion of orbital cellulitis, then a computed tomography (CT) scan or MRI should be ordered.

Treatment. In mild to moderate cases the prescribed therapy for pre-septal cellulitis is oral antibiotics:

- In adults, e.g., cephalexin hydrochloride (Keflex®) 250 mg q. 6 h. for 10 days.
- In children, e.g., cefaclor (Ceclor®) 40 mg/kg/day (maximum 1 gm/day) q. 8 h. for 10 days.

In severe cases intravenous antibiotics are administered. For example:

- In adults, ceftriaxone 1 to 2 g, IV, q. 12 h. and vancomycin 0.5 to 1 g, IV, q. 12 h.
- In children, ceftriaxone 100 mg/kg/day in two divided doses and vancomycin 40 mg/kg/day in three to four divided doses.

Chalazion

Description

Chalazion may be manifested initially as diffuse eyelid swelling which results from blockage of the duct of a meibomian gland (Plate 3). Acutely, the obstruction may be secondary to infection by *Staphylococcus sp.* When the infection resolves, a painless nodule may remain which points to the skin or conjunctival side. Recurrent chalazia are often seen in association with blepharitis; appropriate treatment will decrease the incidence of this condition.

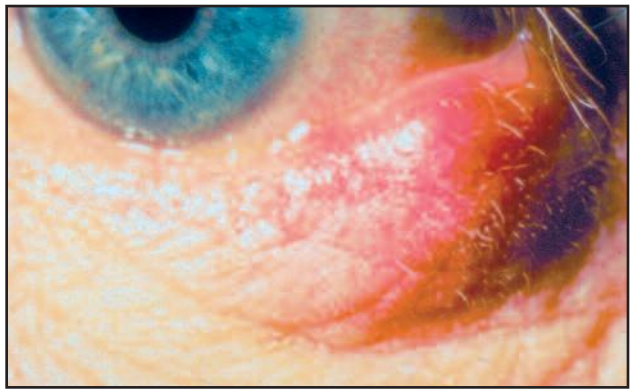


Plate 3
Chalazion characterized by localized lid swelling due to obstruction of a meibomian gland

Workup. There is effectively no workup for the treatment of this condition.

Treatment

- Warm compresses can be applied for 10 minutes four times a day.
- Topical antibiotic such as moxifloxacin (Vigamox®), besifloxacin (Besivance®) or gatifloxacin (Zymar®) can be applied t.i.d.
- If the condition does not resolve in four to six weeks and is of cosmetic concern to the patient, the affected area can be incised and drained under local anesthesia; infants and children usually require general anesthesia. The incision is usually made on the conjunctival side of the tarsal plate, which obviates a skin incision and resultant scar.

Acute Dacryocystitis

Description

Acute dacryocystitis is a blockage of the lacrimal duct which impedes the flow of tears through the lacrimal drainage system. Stasis occurs, which can result in a secondary bacterial infection leading to swelling and tenderness of the lacrimal sac (Plate 4). Conjunctival injection and preseptal cellulitis often occurs in conjunction with acute dacryocystitis. Epiphora (i.e., tearing) is invariably present. The organism most commonly associated with this condition in children under five years of age is *Hemophilus influenzae*, and in adults is *Staphylococcus aureus* (usually penicillinase-resistant).

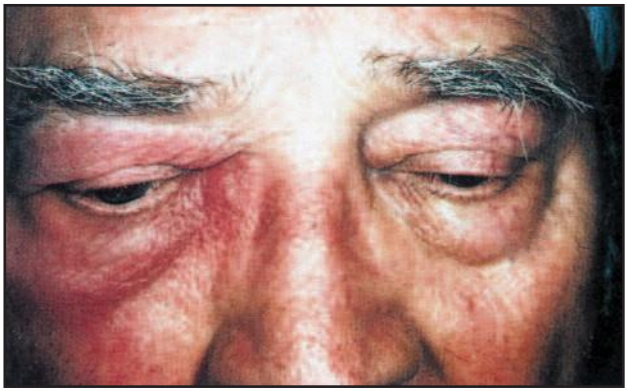


Plate 4
Dacryocystitis with swelling over the lacrimal sac and tearing

Workup. Pressure is applied to the lacrimal sac to express material through the puncta, and a conjunctival culture is prepared.

Treatment. In mild to moderate cases oral antibiotics are prescribed:
- In adults, Keflex® 250 mg q. 6 h. for 10 days.
- In children, Ceclor® 40 mg/kg (maximum 1 g/day) in divided doses every 8 hours for 10 days.

In severe cases intravenous antibiotics are administered as for pre-septal cellulitis:
- Adults may receive nafcillin 1.5 g and penicillin 3 million units q. 4 h.
- Children may receive ampicillin 200 mg/kg/day and chloramphenicol11 mg/kg/day. If the organism is sensitive to ampicillin, the chloramphenicol is discontinued.
- When the infection resolves, a dacryocystorhinostomy is recommended to provide a drainage channel for the tears.

Blepharitis

Description

Blepharitis is characterized by scales on the eyelashes, erythema of lid margins, and misdirection or loss of lashes (Plate 5). It may be associated with conjunctivitis, keratitis, or neovascularization of the cornea (Plate 6). Blepharitis may be seborrheic and/or secondary to *Staphylococcus sp.* Rarely, the lids may be infected by pubic lice (pediculosis).

Treatment of Staphylococcal and/or Seborrheic Blepharitis
- Warm compress should be applied b.i.d. to the eyelids to remove scales, and the lid margins cleansed with diluted Johnson's® Baby Shampoo applied with a cotton swab or a specially formulated eyelid cleanser such as Lid & Lash™.
- Ciloxan® ointment may also be applied to the eyelids at bedtime.

Plate 5
Blepharitis as characterized by erythema of the lid margins and scales on the lashes

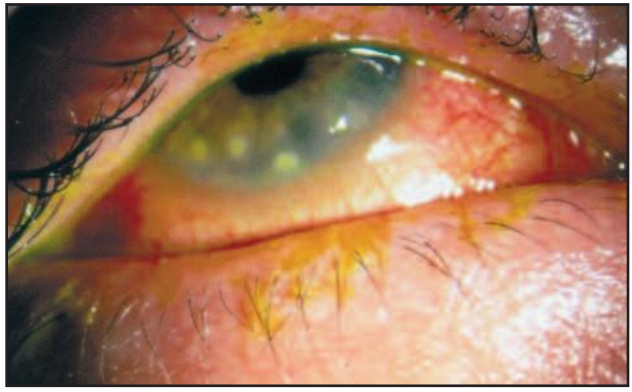


Plate 6
Blepharo-keratitis with sterile immune infiltration of the peripheral cornea

- Artificial tears are used if there is associated keratitis or dry eye, e.g., Systane® Ultra, Refresh Tears®, Blink Tears®, etc. applied q.i.d. and p.r.n. Preservative-free tears are also available without risk of toxicity, e.g., Systane® Preservative-Free, Bion Tears®, Hylo®, Hydrasense®.
- If these measures fail to resolve the problem, then the patient should be referred to an ophthalmologist.
- A short course of a topical steroid/antibiotic combination such as dexamethasone/tobramycin (Tobradex®) may be useful if there is significant inflammation (Appendix F).
- Tetracycline or doxycycline administered orally is useful in refractory cases, especially in those associated with acne rosacea.

Treatment of Pediculosis-Associated Blepharitis. Pubic lice (pediculosis) involvement of the eyelids requires a distinct treatment:

- A 20% fluorescein solution applied to lashes will cause the adult lice to fall off.
- Eggs must be removed manually.
- A 30% incidence of other venereal diseases exists, and this should be ruled out by appropriate testing.

Dry Eye

Description

Dry eye is a common ocular condition characterized by irritation, burning, and tired eyes affecting approximately 15% of the population and affects far more women than men (Appendix G). Systemic medications such as β-adrenergic-blocking, anti-anginal, and anti-hypertensives; tricyclic anti-depressants; oral antihistamines; alkylating immunosuppressives; and diuretics increase the incidence of dry eye. Systemic diseases are also associated with increased incidence, i.e., diabetes can increase the incidence of dry eye by up to 50%. Symptoms are often worse when blinking is reduced during activities such as reading, watching TV, and driving. Contact lens tolerance becomes reduced as the contact lenses and eyes become dry. The eyes may have a lackluster appearance, redness, and a decrease in the tear film (Plate 7) (Appendix H).

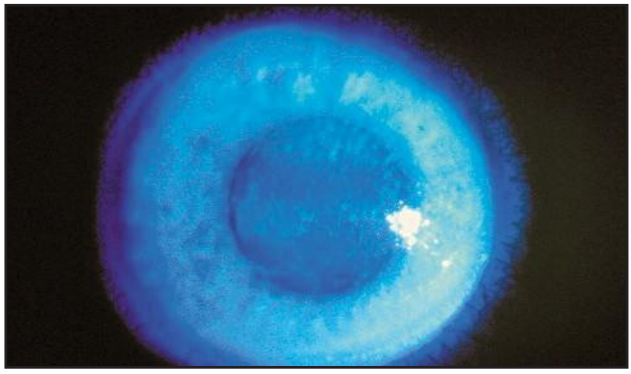


Plate 7
Severe dry eye syndrome with a diffuse punctate keratitis that is best seen with fluorescein dye under cobalt blue light

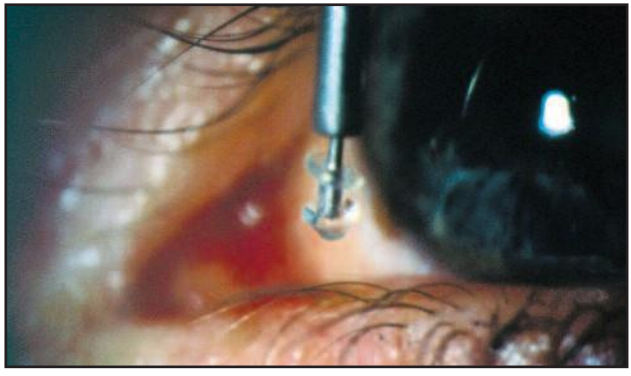


Plate 8
Silicone punctum plug used to enhance the tear film in dry eyes

Workup

- Rose Bengal will stain devitalized cells of the cornea and conjunctiva in advanced cases of a dry eye condition.
- Schirmer strips will measure the amount of tear production. The strips are placed in the inferior conjunctival fornix and draped over the lid margin. The amount of wetting is determined after a five minute period. Less than 10 mm of wetting is suggestive of a dry eye.
- TearLab™ osmolarity system is intended to measure the osmolarity of tears to aid in the diagnosis of dry eye. An elevated tear osmolarity (increased salt in the tears) causes dry-eye surface disease.

Treatment

- Artificial tears are recommended a few times per day to every hour (e.g., Systane® Preservative-Free, Bion Tears®, Hylo®, and Hydrasense® drops).
- Lubricating ointment can be given at bedtime (e.g., Tears Naturale® P.M., Liposic® gel, Hylo® gel).
- Humidifier may be helpful at home and in the workplace.
- Punctal plugs can be inserted to enhance the tear film (Plate 8). This is similar to placing a stopper in a bathtub which will decrease the outflow.
- Increase oral consumption of Omega-3 fatty acids found in fish, walnuts, flaxseed, canola oil, and soybean oil.
- Xiidra® (Lifitegrast 5%) or Restasis® (Cyclosporin) drops may be used b.i.d. to increase tear production in patients whose tear production is presumed to be suppressed dueto ocular inflammation.
- Dry eye secondary to meibomian gland dysfunction may benefit from the Lipiflow® treatment, which involves the application of heat to the inside of the lids to liquefy the lipid within the meibomian glands and ducts.

Allergic Conjunctivitis

Description

Itching is the hallmark of allergic conjunctivitis. Other symptoms include tearing, redness, and chemosis (swelling of the conjunctiva). The condition may be unilateral or bilateral (Plate 9). The patient often has a history of atopy (allergic symptoms, rhinitis, asthma, eczema). There may be a history of allergies to dust, pollen, grass, cats, dogs, etc.

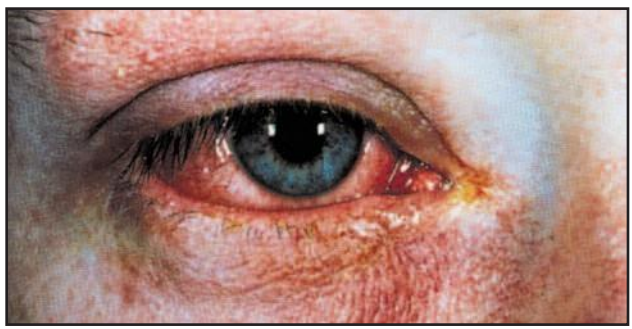


Plate 9
Allergic conjunctivitis with swelling of the lids and conjunctiva

Workup
- Conjunctival scraping is optional.
- Giemsa stain may show eosinophils.

Treatment
- Mast cell stabilizers/antihistamine combinations can be prescribed,e.g., olopatadine hydrochloride (Pataday®), nedocromil (Alocril®) b.i.d.
- Cold compresses and topical antihistamines can be applied, e.g., naphazoline and pheniramine (Naphcon-A®) t.i.d.
- Topical nonsteroidals may be effective alone or in combination with the above drugs, e.g., ketorolac (Acular®), ketorolac preservative-free (Acuvail®), nepafenac (Nevanac®), or diclofenac (Voltaren®).
- If highly symptomatic, the patient can be referred to an ophthalmologist.
- A short course of a mild topical steroid could be prescribed, e.g., fluorometholoneacetate (Flarex®), loteprednol 0.2% (Alrex®), or loteprednol etabonate (Lotemax®) qid.

Adenoviral Conjunctivitis

Description

Adenoviral conjunctivitis is a highly contagious disease (for up to 10 days) characterized by redness, tearing, and a variable degree of photophobia (Plate 10). Follicular hypertrophy of the conjunctiva, which is difficult to detect in the absence of a slit lamp, microscopically represents focal collections of lymphocytes. Keratitis may be absent or limited to superficial punctate keratitis or subepithelial infiltrates. Enlarged pre-auricular lymph nodes may be palpated and their presence is helpful in confirming the diagnosis, as they are never seen in bacterial conjunctivitis except with the gonococcal organism.

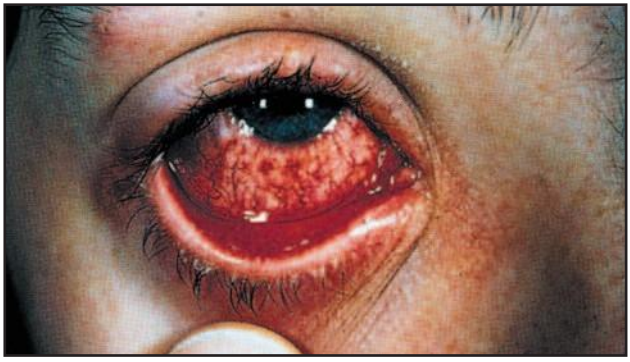


Plate 10
Adenoviral conjunctivitis with lid swelling, conjunctival injection, and tearing

Workup. Cultures are unnecessary since diagnosis is based on clinical evaluation.

Treatment
- No specific antiviral therapy is available.
- Cold compresses can be applied for patient comfort.

- Artificial tears (e.g., Systane® Preservative-Free, Bion Tears®, Hylo® drops q.i.d and p.r.n.) or astringents (e.g., Naphcon-A®) can be used.
- Povidone-Iodine diluted at a concentration of 1:10 is a potential option to reduce infectivity of adenovirus.
- Prophylactic precautions should be observed by the patient's family members and friends.
- Children should stay away from school for 7 to 10 days.
- If the examiner is uncertain of the diagnosis, it should be assumed that the cause is bacterial, and the condition treated with a topical antibiotic.

Bacterial Conjunctivitis

Description

The symptoms of bacterial conjunctivitis are redness, purulent discharge, and "crusty" eye upon morning waking (Plate 11). There is no pre-auricular node enlargement except in cases of gonococcal conjunctivitis. This condition is less common than viral conjunctivitis.

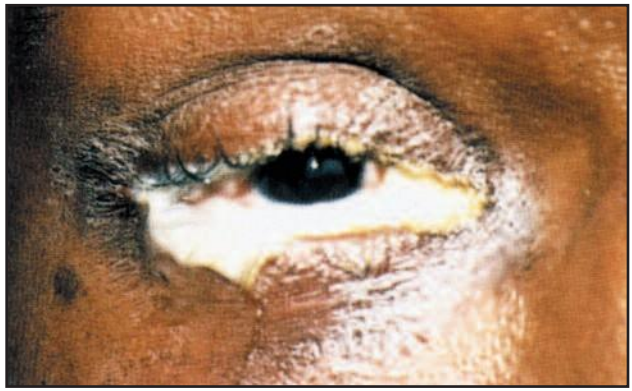


Plate 11
Bacterial conjunctivitis characterized by a purulent discharge that was due to gonorrhea

Workup. In severe cases or those involving a neonate, a Gram stain and culture can be prepared.

Treatment

- Broad-spectrum fluoroquinolone antibiotics (e.g., Vigamox®, Besivance®, Zymar® q.i.d.) are prescribed.
- In children under five years of age infection may be by *Hemophilus influenza* and a fluoroquinolone antibiotic can be used as treatment.
- In cases of gonococcal conjunctivitis, the patient may be given ceftriaxone 1 g in a single dose. If corneal involvement exists or cannot be excluded because of eyelid swelling and chemosis then the patient should be hospitalized and treated with ceftriaxone 1 g IV q. 12 h. to 24 h. In penicillin-allergic patients consideration can be given to ciprofloxacin 500 mg p.o. single dose. Topical fluroquinolone drops q. 2 h. and or topical bacitracin ointment.

Chlamydia

Description

This is a venereal disease which is usually seen in young sexually active adults. The ocular symptoms of chlamydial infection include redness and mucoid discharge, with or without photophobia. The pre-auricular lymph nodes may be enlarged. Follicular hypertrophy of the conjunctiva is characteristically seen by slit-lamp examination, and later in the disease course a superior micropannus of the cornea may develop (Plate 12). This condition is refractory to topical eye medications, and unlike adenoviral conjunctivitis which usually resolves in less than one month, it may become chronic if not treated.

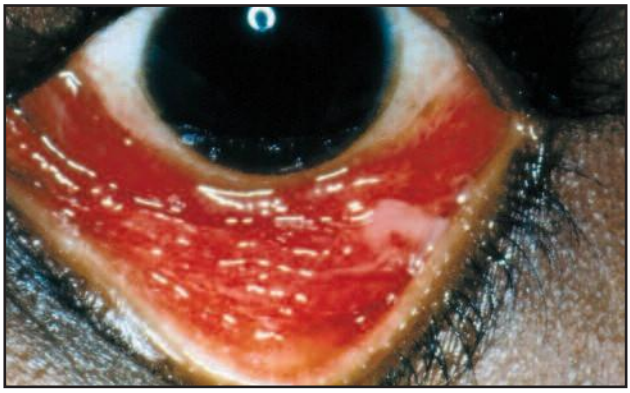


Plate 12
Chlamydial infection characterized by redness, mucoid discharge, and follicular hypertrophy

Workup

- The patient should be referred to an ophthalmologist.
- Clinical diagnosis is made based on the signs and chronicity.
- A Giemsa stain, culture, and fluorescent antibody stain can be performed, but false negatives may occur.

Treatment

- Doxycycline 100 mg p.o. b.i.d for one week; or a single dose of azithromycin 1 gram.
- The patient's sexual partner must be similarly treated for the same duration.

Herpes Simplex

Primary Herpes Simplex

Description

The first exposure to herpes simplex virus in 90% of cases results in subclinical, usually mild disease. Resistance increases with age, so that primary infection is exceedingly rare in early adult life. Characteristically, the young child is infected by

salivary contamination from an adult who has labial herpes. The incubation period is three to nine days. The clinical features of herpes simplex are both ocular and nonocular.

Ocular disease. Characteristics are vesicular eruption (especially lower lid and medial canthus) (Plate 13), conjunctivitis, regional lymphadenopathy, and occasional corneal epithelial disease. Symptoms are frequently unilateral.

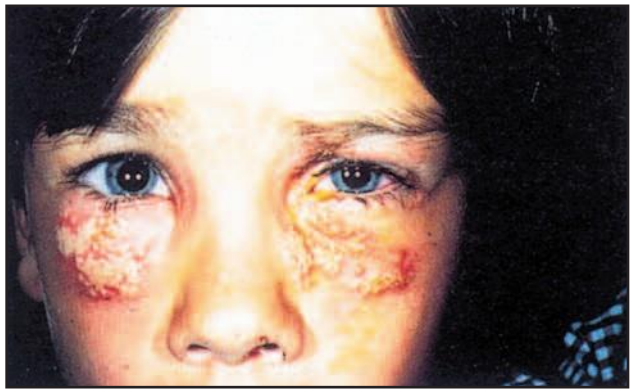


Plate 13
Primary herpes simplex dermatitis with bilateral facial involvement

Nonocular Disease. The following forms of the disease may be present:

- Gingivostomatitis — The symptoms are fever, malaise, cervical lymphadenopathy, and sore throat.
- Pharyngitis — In college students, a primary attack of herpes simplex virus frequently results in a pharyngitis with vesicles on the tonsils.
- Cutaneous disease — Generally, type I occurs above the waist and type II below the waist. This disease may be seen in wrestlers, rugby players, and as aherpetic whitlow in dentists.
- Genital infection — Type II of the infection is more common than type I and is characterized by balanitis in males and cervicitis/vulvovaginitis in females. Patients may exhibit fever, myalgia, extensive vesicular lesions, and inguinal and pelvic lymphadenopathy.

Recurrent Herpes Simplex

Description

The virus develops a "symbiosis" with man, and trigger mechanisms such as trauma, fever, sunlight, emotional stress, steroids, and menses provoke viral shedding, and immunological functions may be overcome. The trigeminal ganglion is a reservoir for the type I disease. The virus has a 50% recurrence rate over five years, and the recurring condition may be highly localized on the lips, nose, chin, eyes (lids, conjunctiva, corneal epithelium, corneal stroma, uvea), and genitals.

Workup. Since this is a clinical diagnosis, cultures are usually unnecessary.

Treatment. The various forms of herpes simplex require specific treatment:

- **Blepharitis** may occur without conjunctival or corneal disease. If it is recurrent, this is consistent with herpes simplex; herpes zoster does not recur. If there is skin but no lid involvement, no topical antiviral treatment is necessary. If the lid margin is involved, then prophylactic antivirals (e.g., Valcyclovir or Acyclovir)
- **Conjunctivitis** may occur without lid or corneal disease and the patient may have an enlarged pre-auricular lymph node. Ophthalmic referral is recommended and an antiviral (e.g., Valcyclovir or Acyclovir).
- **Keratitis** occurs in the following forms:

(i) **Punctate keratitis** is characterized by raised clusters of opaque epithelial cells, as evidenced with fluorescein stain. Referral to an ophthalmologist is recommended. If diagnosis is unequivocal, an oral antiviral should be prescribed (e.g., Valcyclovir or Acyclovir) in the case of an equivocal diagnosis, treatment should be deferred, and the patient followed closely.

(ii) **Dendritic keratitis** is recognized by desquamation in the center of plaques of swollen epithelial cells (Plate 14). The typical linear branching ulcer (stains with fluorescein) has overhanging margins of swollen opaque cells, which are laden with virus (stains with rose bengal). Ophthalmic referral is recommended and an oral antiviral (e.g., Valcyclovir or Acyclovir) should be used.

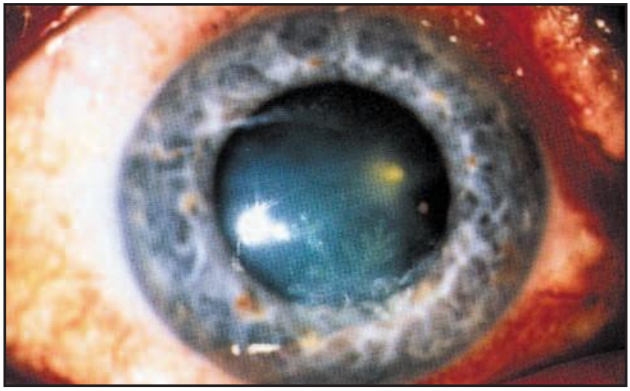


Plate 14
Herpes simplex dendritic keratitis

(iii) **Geographic keratitis** results from progression of dendritic keratitis; a geographic epithelial defect (stains with fluorescein) is lined by heaped-up opaque cells (stains with rose bengal) and may be associated with steroid use in dendritic keratitis. Ophthalmic referral is recommended and an antiviral (e.g., Valcyclovir or Acyclovir) should be prescribed.

(iv) **Stromal keratitis** is an immunologic disease characterized by corneal stromal infiltrates and/or edema. Corneal inflammation that may be associated with iritis and keratic precipitates results from antibodies directed at viral antigens.

Ophthalmic referral is recommended. If the epithelium is intact, a topical steroid, such as fluorometholone acetate (Flarex®), loteprednol 0.5% (Lotemax®), or prednisolone acetate (Pred Forte®) 5x/day. If the stromal keratitis is associated with an epithelialdisease, an oral antiviral (e.g., Valcyclovir or Acyclovir) should be taken until the epithelium heals (approximately 14 days), after which a topical steroid can be added.

- The typical dose above for oral Valcyclovir is 500 mg po TID and Acyclovir is 400 mg 5 times per day for 10 to 14 days. If the disease is resistant (no improvement after 2 weeks), then consideration should be given to compounding topical Ganciclovir 0.15% gel.
- Oral acyclovir (400 mg p.o. b.i.d. for 1 year) has been shown to reduce the epithelial recurrence of ocular simplex in patients who had one or more recurrent episodes. This medication has not been shown to prevent the development of stromal keratitis or uveitis.

Herpes Zoster

Description

Herpes zoster tends to occur in children under 14 and in adults over 40 years of age. Its incidence is five times greater in those over 80 years of age than in adults between 20 and 40. Almost one out of three people in North America will develop herpes zoster during their lifetime.

The varicella virus which causes chickenpox can lie dormant in the sensory ganglia and later reactivate as shingles or herpes zoster (Plate 15). Causes of reactivation are unknown but may be related to aging, immune compromise (e.g., AIDS, lymphoproliferative diseases, systemic steroids), and trauma to the involved ganglion. Although chickenpox is contagious, it should not cause herpes zoster; however, children and adults who have not had chickenpox can contract the disease from herpes zoster patients. Once the virus is reactivated, it may be contained (zoster sine herpete), or spread to the brain, skin, eye, or enter the bloodstream. The virus has a predilection for dermatome T3-L3, but the most common site is the trigeminal nerve. Cutaneous lesions of herpes zoster are histopathologically identical to varicella but have a greater inflammatory reaction which can cause scarring.

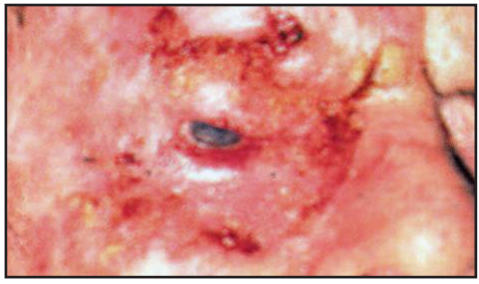


Plate 15 Herpes zoster ophthalmicus with trigeminal nerve distribution

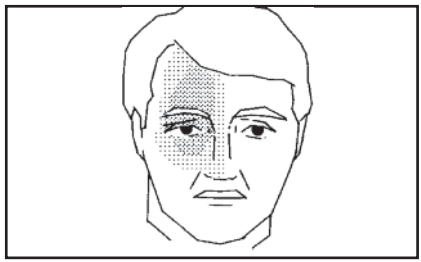


Fig. 17 Herpes zoster ophthalmicus is characterized by vesicular skin eruptions in the distribution of any of the branches of the trigeminal nerve.

The dermatome pattern of herpes zoster may occur in three sites supplied by branches of the trigeminal nerve:

- The ophthalmic nerve distribution (V1) where it occurs 20 times more frequently than at the V2 or V3 sites. Frontal involvement is the most common, including the upper lid, forehead, and superior conjunctiva, which are supplied by supraorbital and suprtrochlear branches (Fig. 17). Alternatively, it may spread to the lacrimal and nasociliary area which
- supplies the cornea, iris, ciliary body, and the tip of the nose.

The virus may affect none, any, or all of these branches. Involvement of the nasociliary nerve often leads to infection of the eye. Hutchinson's rule (1860s) states that ocular involvement is frequent if the side of the tip of the nose is involved.

Clinically, herpes zoster is characterized by a prodrome, skin disease, and ocular complications. The patients may experience pain, burning, itching, hyperesthesia in the dermatome area, followed by erythema, macules, papules, and vesicles which become confluent and may form deeply pitted scars (dermis affected by necrotic process). Ocular complications include lid scarring and exposure, muscle palsies, conjunctivitis, episcleritis, scleritis, keratitis, uveitis, and retinitis.

A vaccination is available to reduce the risk of developing herpes zoster. Individuals aged 50 years and above should receive a pair of Shingrix vaccine doses, spaced 2 to 6 months apart. Those aged 19 years and older, who currently have compromised immune systems due to illness or medical treatment, should likewise be administered two doses of Shingrix. In the event it is necessary, individuals with weakened immune systems can opt to receive their second dose 1 to 2 months subsequent to the initial dose.

Workup. Systemic evaluation for underlying malignancy is not indicated since the yield is low.

Treatment

- Compresses can be applied to the affected areas of the skin.
- Medication for pain relief can be given.
- Antivirals may be prescribed: Famciclovir 500 mg 3 times a day for 7 days and Valacyclovir 1 g 3 times a day for 7 days have better bioavailability with oral dosing than acyclovir, and therefore for herpes zoster, they are generally preferred to oral Acyclovir 800 mg 5 times a day for 7 to 10 days. It is more effective if initiated within 72 hours of the onset of the disease. Corticosteroids do not decrease the incidence of postherpetic neuralgia.
- If the patient has lesions close to the eye, they should be referred to an ophthalmologist to rule out ocular involvement.

- Topical steroids (e.g., Flarex®, Lotemax®, Pred Forte® q.i.d.) will improve comfort and decrease the chance of corneal scarring when there is corneal stromal inflammation.
- Lubrication with preservative-free artificial tears (e.g., Systane® Preservative-Free, Bion Tears®, Hydrasense®, Hylo®) q. 1 – 2 h. may be helpful.
- Cycloplegic agents (e.g., Cyclogyl® 2% b.i.d.) will relieve ciliary spasm in corneal and anterior chamber inflammation making the patient more comfortable and dilating the pupil to prevent posterior synechiae (iris-lens adhesions).
- Pain may be severe during the first two weeks and analgesics (e.g., acetaminophen with or without codeine) may be required. An antidepressant (e.g., amitriptyline 25 mg p.o. t.i.d.) may be beneficial as depression frequently develops during the acute phase of HZV infection. Antidepressants also may help post-herpetic neuralgia. Management of post-herpetic neuralgia should involve the patient's primary medical doctor.
- Patients with active herpes zoster can transmit the virus that causes shingles to a person who has never had chickenpox, but only through direct contact with the shingles rash. If a person who has never had chickenpox is infected with the varicella zoster virus, he or she will develop chickenpox, not shingles.

Toxic Conjunctivitis

Description

This condition may be secondary to a topical medication (e.g., antibiotics, glaucoma drops, etc.), from a consumer product such as eye make-up or moisturizing creams, or from *molluscum contagiosum*. The patient will often complain of irritation and redness. If the cause is secondary to *molluscum contagiosum* the focal lesions may be seen on the face and/or lids (Plate 16 A & B).

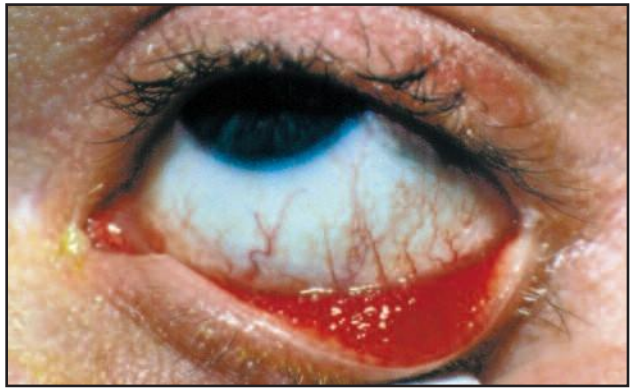


Plate 16A
Toxic conjunctivitis secondary to *molluscum contagiosum*

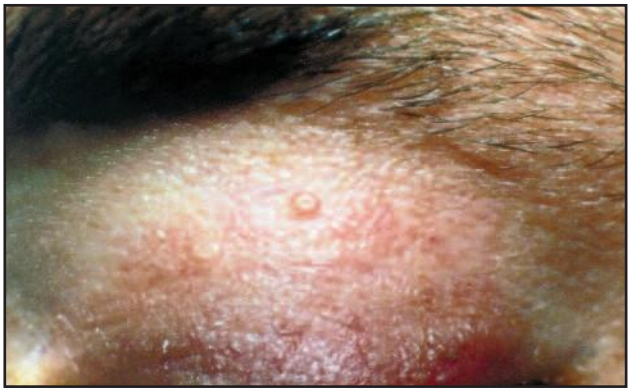


Plate 16B
Molluscum lesions of the upper lid

Workup. No workup is required.

Treatment

- If drug-induced, it is important to discontinue the topical medications. If the patient has a history of glaucoma then alternative medications need to be prescribed.
- If the patient is not taking any topical medications, then consideration should be given to the potential irritative effects from a consumer product. Discontinuing eye make-up and creams may be effective in resolving the symptoms.
- If the diagnosis is *molluscum contagiosum* then the small skin lesions can either be excised, lasered, or treated with cryotherapy. In addition, Cantharidin, a chemical compound can be applied, causing them to form a blister and eventually fall off.

Recurrent Corneal Erosions

Description

Patients with recurrent corneal erosions experience pain, photophobia, and redness, but have no acute history of trauma. Corneal erosion, which stains with fluorescein, is due to the lack of strong corneal epithelial attachments. The erosion frequently occurs on awakening, since the corneal epithelium becomes more edematous during eyelid closure and more susceptible to focal sloughing. Predisposing factors for this condition may be an old traumatic injury (e.g., fingernail, tree branch, paper), corneal dystrophy, and bullous keratopathy (i.e., corneal edema).

Workup. Fluorescein dye will stain the erosions. The dye is also useful if the epithelium is intact, as it will show an irregular epithelial surface characterized by a rapid breakup of the tear film overlying the area of concern.

Treatment

- Antibiotic drops (e.g., Vigamox®, Besivance®, Zymar®) and a cycloplegic agent (e.g., Cyclogyl® 1%) should be prescribed.
- A pressure patch or a bandage soft contact lens should be applied.
- Ophthalmic referral is recommended.

- Hypertonic drops and/or ointment, e.g., sodium chloride 5% (Muro 128 5%) drops during the day and ointment at bedtime to be used over a period of weeks to months to dehydrate the epithelium and decrease the risk of erosions.
- A bandage soft contact lens can be used for a period of weeks to months to decrease the chance of epithelial erosions.
- Anterior stromal puncture can be performed if the patient continues to develop erosions in the same location. A 25-gauge needle can be used to make multiple punctures into the anterior stroma in the area of the erosion. This allows for the development of stronger adhesions and decreases the risk of erosions. However, the technique is contraindicated in erosions that occur close to the pupillary axis.
- In patients who continue to have recurrent corneal erosions, the excimer laser can be used to perform a phototherapeutic keratectomy. After the epithelium is mechanically removed, five to ten microns of stromal tissue are ablated. This allows for greater epithelial adherence to the somewhat roughened surface and causes essentially no change in refractive error.
- Another technique that has shown success is debridement of the epithelium followed by polishing Bowman's layer with a diamond burr. A bandage soft contact lens is then inserted and worn until the epithelium becomes intact and clinically smooth. This may take 4 or 5 days or up to a month.

Subconjunctival Hemorrhage

Description

A ruptured vessel with blood accumulation in the subconjunctival space describes a subconjunctival hemorrhage (Plate 17). It is often accompanied by a history of coughing, vomiting or straining. The patient may be taking warfarin sodium (Coumadin®), aspirin, or another blood thinner.

Plate 17
Subconjunctival hemorrhage as evidenced by a bright red color

Workup

- If the patient's history is negative for Valsalva's maneuver, a blood pressure reading should be taken.
- The patient on Coumadin® or other blood thinners should undergo tests to ensure that the rate of blood clotting is in the desired range.
- In the case of recurrent subconjunctival hemorrhage, a complete blood count should be taken to rule out a blood dyscrasia.

Treatment. Reassuring the patient is all that is necessary since the hemorrhage will resolve spontaneously.

Phlyctenule

Description

A phlyctenule is a small, pinkish-white nodule in the center of a hyperemic area of conjunctiva (Plate 18). Although it is seen most frequently near the limbus, it may occur anywhere on the bulbar conjunctiva. Less commonly, it involves the cornea where it is associated with vascular ingrowth. The patient's history should be used to rule out the possibility of any foreign body. Phlyctenules represent a collection of white blood cells (primarily polymorphonuclear leukocytes and lymphocytes) and is caused by a hypersensitivity reaction to an antigenic stimulus such as *Staphylococcus aureus* or the tubercle bacilli.

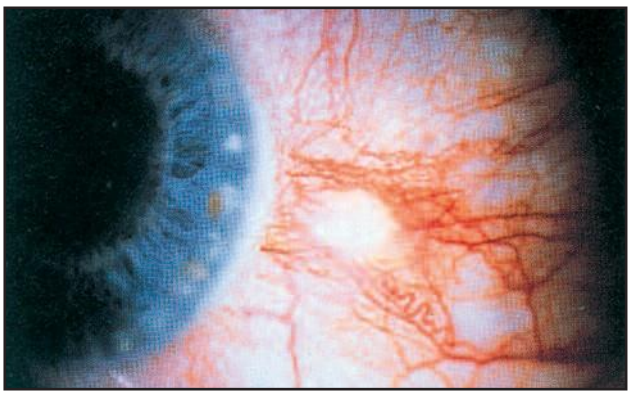


Plate 18
Phlyctenule of the conjunctiva with an elevated white nodule surrounded by conjunctival injection

Workup

- The patient should be referred to an ophthalmologist.
- A tuberculin skin test and chest X ray is recommended if the patient is in a high-risk group.

Treatment

- A topical steroid (e.g., Flarex®, Pred Forte®, Lotemax®, q.i.d.) may be prescribed.
- Any associated staphylococcal blepharitis should be treated.

Episcleritis

Description

Episcleritis is characterized by a salmon-pink hue of the superficial layer of the eye, with involvement of the conjunctiva and episclera (Plate 19). At least one-third of the lesions are tender to touch. Simple episcleritis may be sectoral in 70% or generalized in 30% of patients. In nodular episcleritis, unlike in nodular scleritis, the nodules which form are moveable with a cotton swab. Most cases of episcleritis are idiopathic; however, up to one third of cases may have an underlying systemic condition.

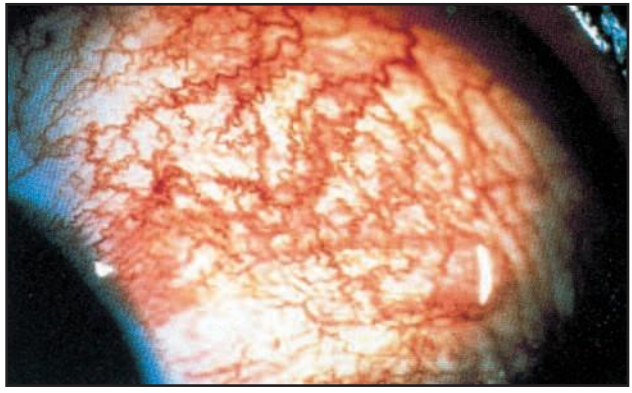


Plate 19
Episcleritis with sectorial injection of the conjunctiva and episcleral tissue

Workup. Ophthalmic referral is recommended.

Treatment. A topical steroid (e.g., Flarex®, Pred Forte®, or Lotemax® q.i.d.) will cause resolution of the inflammation.

Scleritis

Description

Scleritis is frequently bilateral and, characteristically, associated with severe pain. The disease may involve the anterior (visible segment) and/or posterior segment (less frequent). The ocular surface has a purplish hue with involvement of the deep episcleral vessels (Plate 20). Systemic diseases, such as collagen vascular, ulcerative

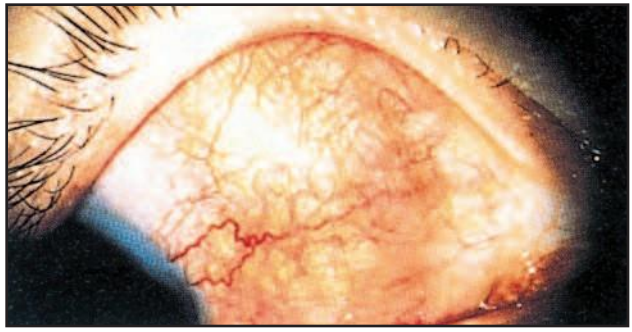


Plate 20
Scleritis with diffuse involvement of the deep episcleral vessels

colitis, Crohn's, and sarcoidosis, are present in 50% of patients. The eight-year mortality rate is 30%, with death usually due to a vascular disease. Scleritis may be classified as simple (in its most benign form), nodular (the nodule is immobile when pushed with a cotton swab), or necrotizing (the majority of these patients have rheumatoid arthritis).

Workup

- Ophthalmic referral is recommended.
- The patient should be evaluated for an underlying systemic disease.

Treatment

- A topical steroid (e.g., Flarex®, Pred Forte®, Lotemax®) may be prescribed to reduce the inflammation.
- A systemic nonsteroidal anti-inflammatory medication is recommended, e.g., indomethacin (Indocin®) 25 mg p.o. t.i.d.
- If there is no significant improvement, then systemic steroids can be prescribed with a tapering dose.

Corneal Ulcers

Description

Patients with corneal ulcers may experience redness, pain, photophobia, and tearing. The cornea will have a whitish infiltrate with an overlying epithelial defect that will stain with fluorescein (Plate 21). Hypopyon (layered pus in the anterior chamber) may be associated with a corneal ulcer. Patients most at risk are those who wear contact lenses, those with blepharitis and dry eyes, or those who have experienced corneal trauma. The most common causes are bacterial infections, e.g., by *Pseudomonas, Staphylococcus aureus*, or *Streptococcus pneumoniae*. Less common organisms include fungi and *Acanthamoeba*. Infection with the latter is relatively rare and is seen predominantly in contact lens wearers. Risk factors for acquiring *Acanthamoeba* include swimming with contact lenses and rinsing lenses with tap water or home-made saline.

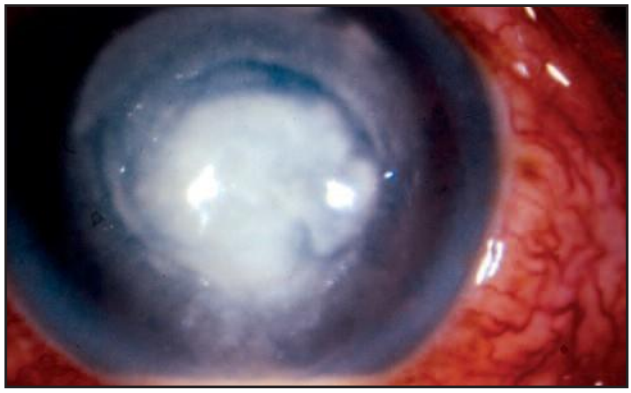


Plate 21
Corneal ulcer with central white infiltrate and a hypopyon

Workup

- Ophthalmic referral is recommended.
- The cornea should be scraped for Gram stain and culture if the infiltrate is in the visual axis, is greater than 2 mm, or is progressive despite antibiotic treatment.

Treatment

- Vigamox®, Besivance®, or Zymar® alone or in combination with Tobrex® may be used for the majority of small corneal ulcers (< 2 mm).
- Topical fortified antibiotics, e.g., tobramycin (1 4 mg/mL) and cefazolin (50 mg/mL) should be considered for advanced corneal ulcers (> 2 mm).
- If a fungal infection is noted with a Gram stain or culture, then topical natamycin and miconazole can be started.
- If *Acanthamoeba* is detected, then the patient can be placed on propamidine isethionate 0.1% (Brolene®) drops and ointment, polyhexamethylene biguanide (0.02%), or chlorhexidine (0.02%).

Iritis

Description

Iritis is characterized by redness, photophobia, tearing, and decreased vision. A ciliary flush is prominent, and the pupil is constricted secondary to the inflammation (Plate 22). Slit-lamp examination shows an anterior chamber reaction manifested by inflammatory cells, flare (protein leakage), and keratic precipitates. If treatment is not initiated early then adhesions of the iris to the lens (posterior synechiae) may occur. Testing with fluorescein stain should be done to rule out corneal abrasion and a herpes simplex dendrite.

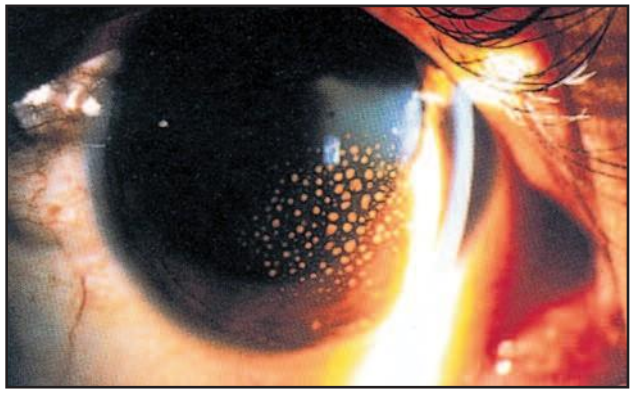


Plate 22
Iritis with granulomatous (mutton-fat) keratic precipitates

Workup

- Ophthalmic referral is recommended.
- If the condition is persistent or recurrent, underlying systemic disorders (e.g., ankylosing spondylitis, sarcoidosis) should be ruled out.

Treatment. A topical steroid (e.g., Maxidex® or Pred Forte® q.1-2 h.) and a cycloplegic agent (e.g., Cyclogyl® 1% or homatropine 5% q. 6 h.) should be prescribed. A NSAID (e.g., Nevanac®, Acular®, Voltaren®) may be used if there is a contraindication to steroid drops; for example, a history of steroid-induced glaucoma.

Acute Angle Closure Glaucoma

Description

Acute angle closure glaucoma is characterized by redness, severe pain, photophobia, and decreased vision (Plate 23, Fig. 18); the patient may also experience nausea and vomiting. Elevated intraocular pressure, corneal edema, and a nonreactive mid-dilated pupil may also be present. This tends to occur more frequently in the hyperopic (farsighted) eye due to a relatively shallow anterior chamber.

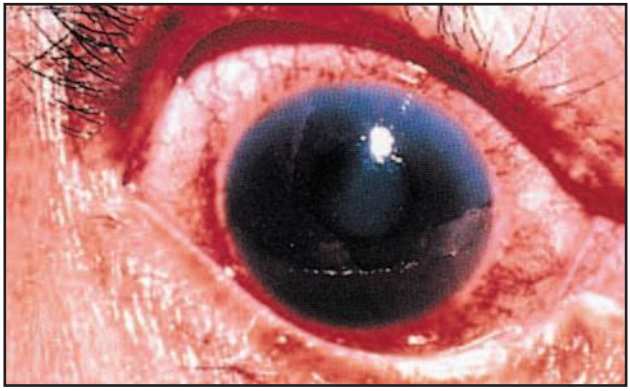


Plate 23
Acute angle closure glaucoma with a ciliary injection, swollen cornea, and marked elevation in intraocular pressure

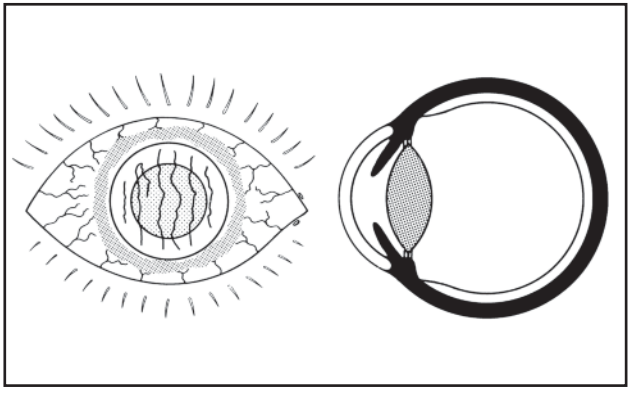


Fig. 18 Acute angle closure glaucoma is manifested by elevated intraocular pressure, and is associated with redness, ciliary injection, corneal edema, a nonreactive mid-dilated pupil, and a relatively narrow anterior chamber angle.

Workup

- Tonometry should be performed to confirm diagnosis.
- Ophthalmic referral is recommended.

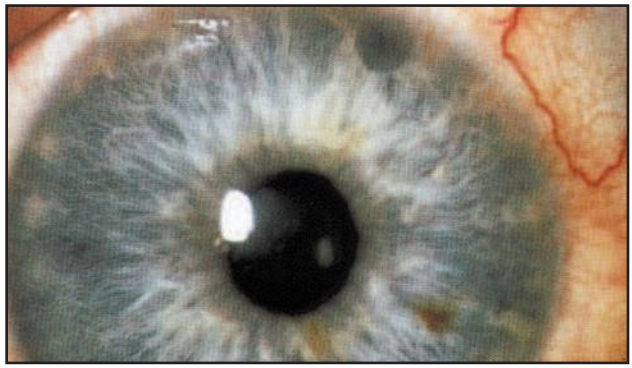


Plate 24
Laser peripheral iridotomy noted superiorly in a patient who had angle-closure glaucoma

Treatment

- Effective medications include pilocarpine 2% q. 5 min. x4, then q.1 h., pressure lowering drop (e.g., Travatan Z®, Xalatan®, or Lumigan®) drops q.12 h., isosorbide 1-2 gm/kg p.o. x1, acetazolamide (Diamox®) 250 mg p.o. q. 6 h. or 500 mg IV, and mannitol IV 20% 1-2 gm/kg.
- Laser iridotomy should be performed in the affected eye and a prophylactic iridotomy in the contralateral eye (Plate 24)

Traumatic Red Eye

Editor's Note

Patients with a traumatic red eye are usually highly symptomatic and typically are seen on the same day as the injury. Injuries may be due to blunt trauma (superficial or intraocular damage), a perforating injury, ultraviolet light, chemical toxicity, or contact lens or solution related mechanisms. The most common injuries are corneal abrasions, metallic corneal foreign bodies, and contact lens solution reactions. If there is a history of a possible metallic foreign body but no foreign body is detected an x-ray is indicated to rule-out an intraocular foreign body. Contact lens related injuries are most commonly secondary to a toxic or hypersensitivity reaction to the contact lens solution. Less common injuries are an ultraviolet light keratitis, chemical injuries, intraocular foreign bodies, blow-out fracture, hyphema, blunt trauma with intraocular damage, or a laceration or perforation.

Corneal Abrasions

Description

The patient with a corneal abrasion has a history of trauma caused, for example, by a tree branch, fingernail, or contact lens. The patient will usually complain of pain, light sensitivity, redness, and blurred vision. Most corneal abrasions heal well without adverse sequela. Some patients may develop recurrent corneal erosions.

Workup. The diagnosis is confirmed by demonstrating an epithelial defect with fluorescein dye under cobalt blue light (Plate 25).

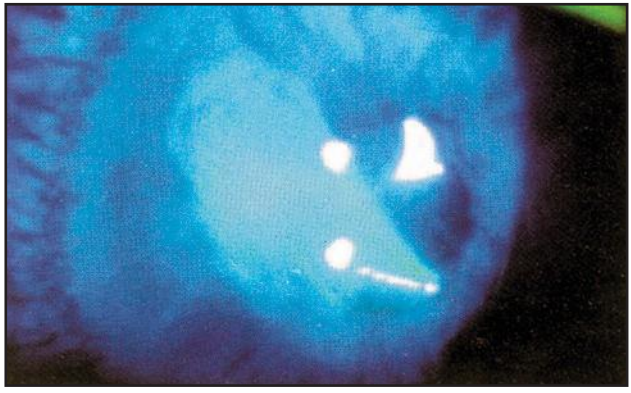


Plate 25
Corneal abrasion stains green with fluorescein dye under cobalt blue light

Treatment

- Antibiotic drops (e.g., Vigamox®, Besivance®, or Zymar®) should be instilled if the eye is patched, or prescribed if no patching, along with a cycloplegic agent (e.g., Cyclogyl® 1% or homatropine 5%).
- Pressure patching of the eye is optional as this has not been shown to reduce pain or improve healing rates. Patching should not be performed in patients at high risk of infection such as those who wear contact lenses (risk of *Pseudomonas* and amebic keratitis) and those with trauma caused by vegetable matter (risk of fungal keratitis).
- Oral analgesic medication may make the patient more comfortable.
- Topical anesthetics should never be prescribed as an analgesic, as this will inhibit corneal epithelial healing.
- Patient follow-up is recommended on a daily basis to determine epithelial healing and ensure the absence of an infection.

Contact Lenses

Description

Contact lens wearers may develop a red eye due to a variety of pathophysiologic causes: mechanical, hypoxic, immunologic, chemical toxicity, and infection. The

most important concern is the possibility of the development of an infected ulcer that can lead to corneal scarring and a permanent decrease in vision (Plate 26).

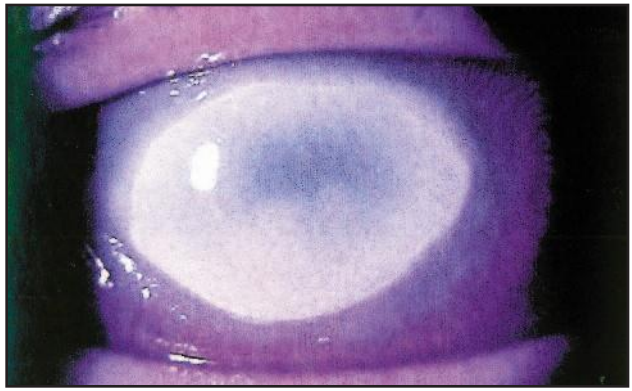


Plate 26
Acanthamoeba corneal ulcer secondary to contact lens wear

Workup. Refer to Appendix I for the differential diagnosis of red eye in contact lens wearers.

Treatment

- All patients with a red eye should remove their contact lenses.
- Referral to an ophthalmologist is necessary to determine the cause of red eye.

Ultraviolet Keratitis

Description

Ultraviolet keratitis is usually bilateral and is characterized by redness, photophobia, tearing, and blepharospasm (Plate 27). Usually the patient has been welding or using a sun lamp without proper eye protection. Typically, the symptoms appear 6 to 10 hours after exposure. The pain is usually out of proportion to the clinical findings.

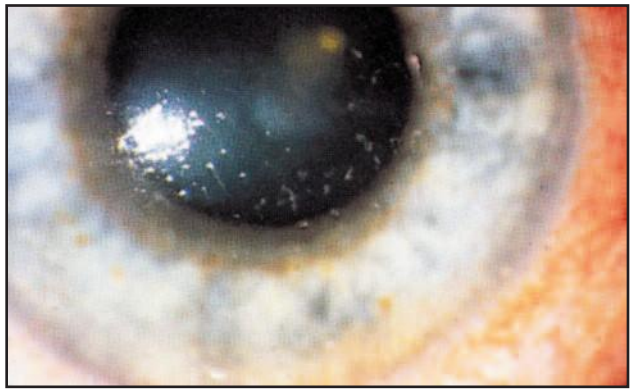


Plate 27
Ultraviolet light burn to the eye with evidence of an epithelial punctate keratitis

Workup. Fluorescein staining will reveal the presence of superficial punctate keratitis.

Treatment

- An antibiotic drop or ointment (e.g., Vigamox®, Besivance®, or Zymar®) should be instilled along with a cycloplegic agent (e.g., Cyclogyl® 1%).
- The more severely affected eye should be patched, and the patient instructed to apply a patch to the less affected eye at home.
- A pain medication can be prescribed.
- Follow-up is recommended to ensure epithelial healing.

Chemical Injuries

Description

Alkali injuries are often more severe than acid injuries due to the fact that acids tend to coagulate tissue and inhibit further penetration into the cornea. Clinical findings of chemical injury vary with severity of the injury: a mild injury is characterized by conjunctivitis, superficial punctate keratitis, and an epithelial defect of the cornea and conjunctiva (Plate 28); a severe injury exhibits blanching of limbal blood vessels and opacification of the cornea (Plate 29).

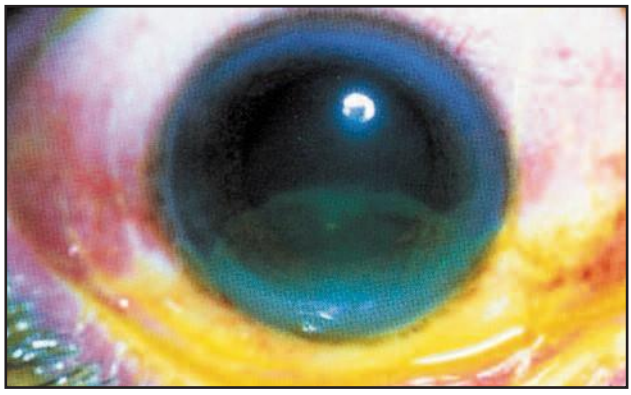


Plate 28
Mild chemical injury with fluorescein staining of the inferior cornea and conjunctiva

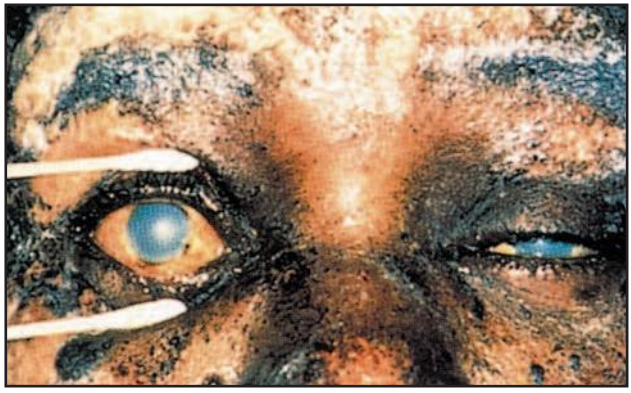


Plate 29
Severe chemical injury to the eyes that resulted in corneal opacification

Alkali Agents

- Ammonia — Commonly found in household ammonia (7% cleaning agent), fertilizer, and refrigerant (strongest concentration is 29%). Penetration of the eye occurs in less than a minute, which makes the injury difficult to treat by irrigation.
- Lye (sodium hydroxide) — Commonly found in drain cleaners (e.g., Draino), it ranks second to ammonia in severity of injury induced.
- Hydroxides — Common forms are potassium hydroxide (found in caustic potash) and magnesium hydroxide (found in sparklers and flares). The chemical burns are similar to those caused by sodium hydroxide.
- Lime ($CaOH_2$=calcium hydroxide) — This is one of the most common substances involved in ocular burns and is found in plaster, cement, mortar, and whitewash. However, because it reacts with the epithelial cell membrane to form calcium soaps which precipitate, it penetrates the eye poorly.

Acid Agents

- Sulfuric Acid (H_2SO_4) — Commonly found in batteries and industrial chemicals, injuries are often due to battery explosions with resultant lacerations, contusions, and foreign bodies. When H_2SO_4 comes into contact with the water in the corneal tissue, heat is released charring the tissue and causing severe injury.
- Sulfur Dioxide (SO_2) — Commonly found combined with oils in fruit and vegetable preservatives, bleach, and refrigerants. It forms sulfurous acid (H_2SO_3) when it combines with water in corneal tissue. Injury is caused by the H_2SO_3 rather than the freezing effect of SO_2; it denatures proteins, inactivates numerous enzymes, and penetrates tissue well because of its high lipid and water solubility.
- Hydrofluoric Acid (HF) — Commonly used for etching and polishing glass or silicone, frosted glass, refined uranium and beryllium, alkylation of high-octane gasoline, production of elemental fluoride, inorganic fluorides, and organic fluorocarbons. Much of the damage to the eye is caused by the fluoride ion.
- Other Acids — These include chromic acid, hydrochloric acid, nitric acid, and acetic acid.

Workup. Since this emergency situation requires immediate treatment, no workup is recommended.

Treatment. The emergency physician should initiate the following procedures:

- Initial vision testing is deferred for chemical injuries until the irrigation and pH testing has been completed.
- Topical anesthetic drops are instilled to facilitate irrigation.
- The eye should be irrigated with a nontoxic liquid (water, ionic solutions, buffered solutions), but acidic solutions are not recommended as they are too risky. An IV drip for at least 30 minutes, with at least one liter of fluid, is recommended with the eyelids retracted. The pH can be checked using litmus paper, applied to the conjunctival surface to assess the adequacy of irrigation in achieving a neutral pH.

- Any particulate matter should be removed from the fornices. A moistened cotton-tip applicator can be used to remove chemical matter. Any particles in the fornices can act as a reservoir and cause severe damage if not identified and removed promptly.
- A cycloplegic agent (e.g., Cyclogyl® or homatropine) will decrease the risk of posterior synechiae and alleviates ciliary spasm.
- An antibiotic drop or ointment can be applied, along with a pressure patch.

The ophthalmologist may initiate the following treatment:
- The patient should be followed closely to ensure healing of the epithelium.
- The intraocular pressure should be lowered, if it is elevated (e.g., prescribe Travatan Z®, Lumigan®, Xalatan®, or a Beta blocker, and/or Diamox®).
- Topical steroids may be used to decrease inflammation but should be limited to no more than two weeks in the case of a persistent epithelial defect. Prolonged steroid use in the presence of an epithelial defect can cause the cornea to melt.
- Bandage contact lenses may be used, if the epithelium is not healed by patching.
- If the cornea heals with scarring and vascularization, the prognosis for restoring vision using a corneal transplant is poor because of the high incidence of graft rejection and failure.

Corneal Foreign Bodies

Description

A corneal foreign body (e.g., metal, glass, wood, plastic, sand) may be present in the patient exhibiting redness, foreign-body sensation, photophobia, and a history of trauma (Plate 30).

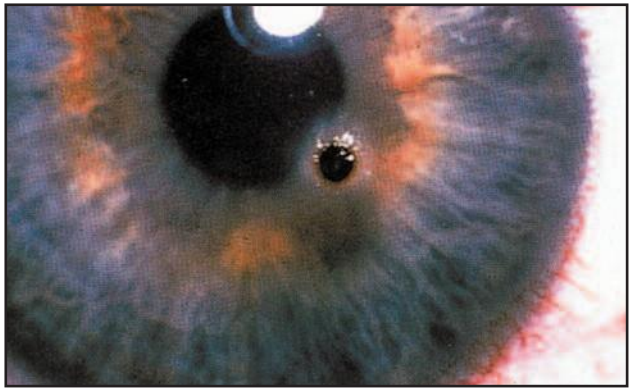


Plate 30
Metallic foreign body of the superficial cornea

Workup. Rule out full thickness corneal penetration that if present should prompt urgent ophthalmic referral.

Treatment

- A topical anesthetic should be instilled, and the foreign body removed with aneedle (e.g., 22-gauge) or a burr drill if done under slit-lamp magnification.

- If the foreign body cannot be seen, the upper lid should be everted to examine the lid margin and the palpebral conjunctival surface (Plate 31).
- It is best to remove all the associated rust, but not essential. It tends to diffuse out of the cornea with time.
- Be extremely gentle when removing foreign bodies overlying the pupil as excessive manipulation can lead to corneal scarring and a decrease in vision (Plate 32).
- A cycloplegic agent (e.g., Cyclogyl® 1%) should be instilled, along with an antibiotic drop or ointment (e.g., Vigamox®, Besivance®, or Zymar®).
- A pressure patch should be applied.
- Follow-up is recommended to determine epithelial healing, and to ensure the absence of infection or residual rust.

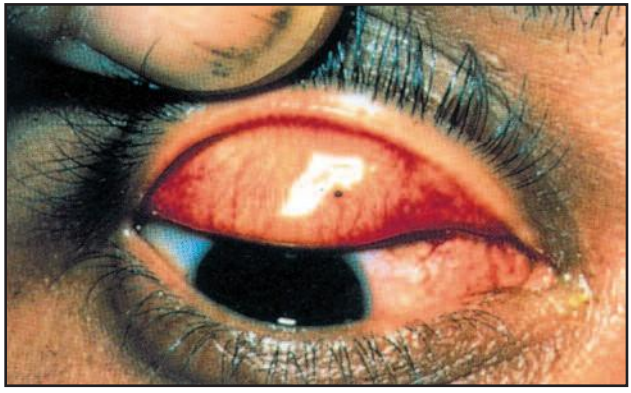


Plate 31
Superficial keratitis of the cornea secondary to a retained foreign body beneath the upper lid

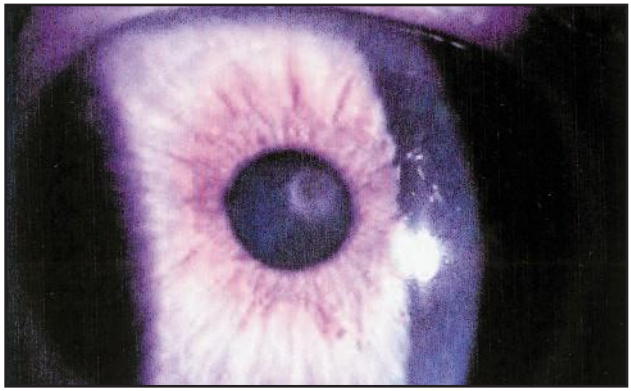


Plate 32
Central corneal scar following foreign body removal

Intraocular Foreign Bodies

Description

A small foreign body travelling at a high speed can penetrate the eye without the patient's awareness. The symptoms are highly variable, depending on the site of penetration and intraocular structures affected. All foreign bodies made of iron should be removed, since they can cause significant intraocular damage (siderosis bulbi) (Plate 33). However, glass, aluminum, gold, and silver are inert and usually cause little or no chronic intraocular damage (Fig. 19).

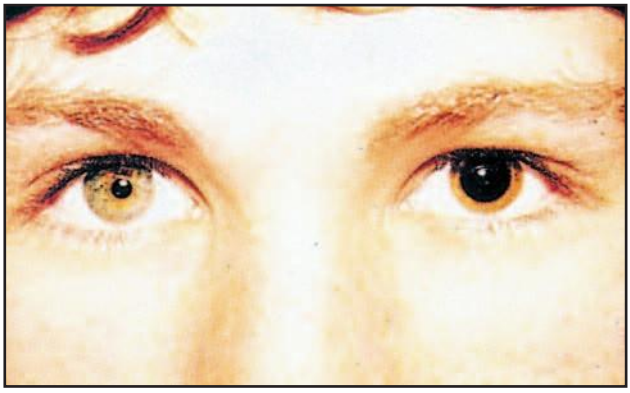


Plate 33
A retained intraocular foreign body that has resulted in siderosis with discoloration of the iris, dilated pupil, and retinal toxicity

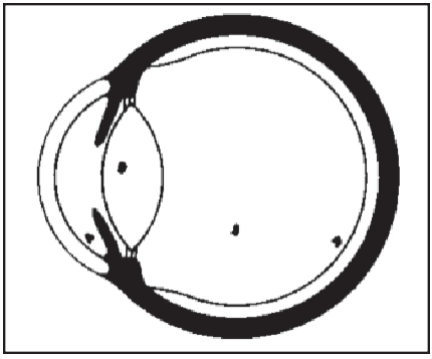


Fig. 19 Intraocular foreign bodies can be found in a variety of sites: in the anterior chamber, lens, vitreous, or retina.

Workup

- An X ray (Waters' view and lateral) and/or B scan ultrasound should be ordered if an intraocular foreignbody is suspected.
- Ophthalmic referral is recommended.
- If the foreign body cannot be visualized on examination, a CT scan should be ordered to determine whether the foreign body is intraocular or extraocular.

Treatment. To extract an intraocular foreign body, magnetic extraction or vitrectomy with foreign body instrumentation is usually indicated. Most foreign bodies outside of the eye in the orbit can usually be left without adverse sequelae.

Blow-Out Fracture

Description

A blow to the periorbital structures can cause a fracture of the orbital floor and result in periorbital ecchymosis, infraorbital nerve anesthesia, and limitation of upgaze (Plate 34). There are two theories as to the mechanism of a blow-out fracture: (1) That a blow to the orbit causes a sudden increase in intraorbital pressure which results in the fracture, and (2) A blow to the inferior orbital rim results in a buckling of the orbital floor.

Other fractures to the orbit are less common. A medial fracture of the thin ethmoidal bone may be associated with subcutaneous emphysema of the eyelids. A fracture at or near the optic canal through which the optic nerve and ophthalmic artery pass may cause damage to the optic nerve, resulting in visual loss.

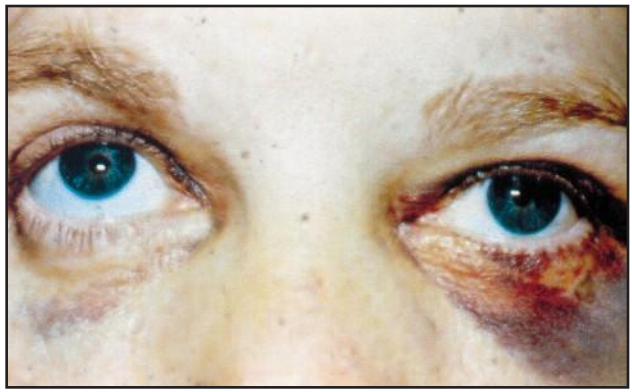


Plate 34
Blow-out fracture to the left orbital floor with periorbital ecchymosis and limitation of upgaze

Workup

- An X ray (Waters' view and lateral) and a CT scan (anteroposterior and coronal views of orbits) should be taken.
- Ophthalmic referral is recommended.
- The eye should be checked for any associated intraocular damage (e.g., hyphema, scleral rupture, traumatic cataract, macular edema, choroidal rupture, retinal tears, or retinal detachment).

Treatment

- Patients should try to refrain from nose-blowing and coughing to prevent pressure from pushing the sinus contents in to the orbit.
- Systemic antibiotics should be prescribed (e.g., Keflex® 250 mg p.o. q.i.d.x 10 days).
- Surgical repair of the orbital fracture is dependent on the CT scan findings and/or clinical signs during the subsequent one to two weeks.
- Surgery is indicated in cases of soft tissue entrapment associated with diplopia, enophthalmos greater than 2 mm, or fractures involving more than one-half of the orbital floor.

Hyphema

Description

Hyphema is caused by blunt or penetrating trauma and is characterized by decreased vision, ciliary injection, and a view of the fundus which is hazy due to the presence of blood (Plate 35, Fig. 20). A ruptured globe must be ruled out. Children often have an unreliable history, and it is important to rule out any intraocular foreign body. A tear in the ciliary body or iris usually occurs in the area of the angle. The incidence of rebleeds is 20% to 25%, usually between the third and fifth days.

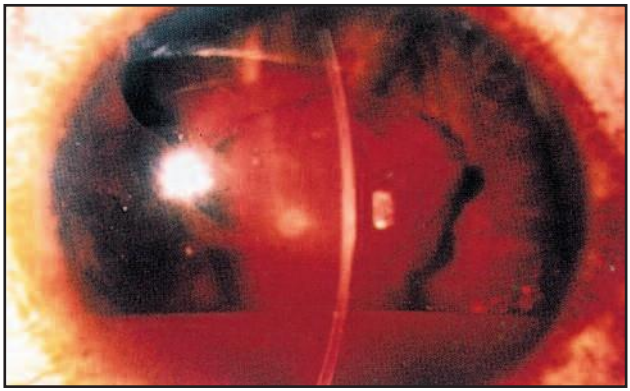


Plate 35
Traumatic hyphema with layering of blood inferiorly and a clot covering the pupil

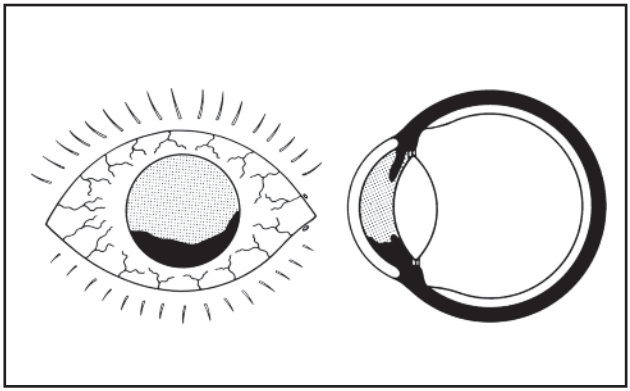


Fig. 20 Usually the result of blunt trauma, hyphema (or blood in the anterior chamber) is characterized by decreased vision, ciliary injection, and a hazy view of the fundus due to the presence of blood.

Workup. No workup is recommended.

Treatment

- Ophthalmic referral is recommended.
- A protective eye shield should be applied.
- Confined either to bedrest with bathroom privileges or to limited activity. No strenuous activity allowed.

- A cycloplegic agent and antiglaucoma medication should be prescribed. No aspirin-containing products should be given.
- Topical steroids e.g., Flarex®, Pred Forte®, Maxidex® q.i.d. should be prescribed to reduce intraocular inflammation.
- Aminocaproic acid (Amicar®) reduces the incidence of secondary hemorrhage and may be taken orally in a dosage of 50 mg/kg every 4 hours for 5 days. Adverse effects such as hypotension, nausea, and hepatic toxicity limit its use. The medication retards clot lysis by preventing plasmin from binding to lysine in the fibrin clot. This medication may be used in hyphemas occupying 75% or less of the anterior chamber because the clot may persist in the anteriorchamber for an increased period during administration of the drug.
- Patients should be told that they are at an increased risk for the development of glaucoma secondary to damage to the angle, as well as for retinal detachment. Patients therefore should be followed on a regular basis for the rest of their lives.

Blunt Trauma Injury

Description

Hyphema, cataract, iridodialysis (Plate 36), scleral rupture (Plate 37), traumatic mydriasis, choroidal rupture, optic neuropathy, retinal tears and/or retinal detachment may be present in blunt trauma injuries. Retinal hemorrhages in children older than one month are a strong indicator of the shaken baby syndrome (Plate 38).

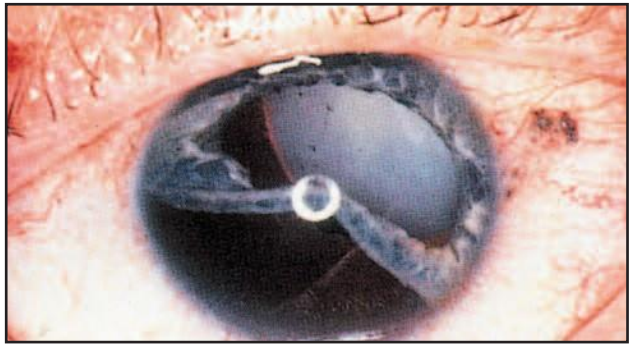


Plate 36
Traumatic iridodialysis and cataract

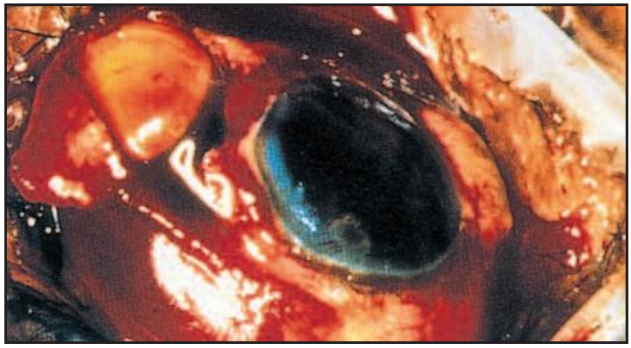


Plate 37
Severe blunt trauma that resulted in a scleral rupture with extrusion of the lens

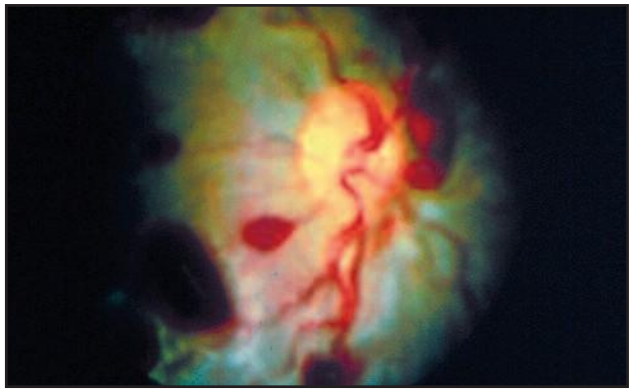


Plate 38
Scattered retinal hemorrhages secondary to head trauma in a case of child abuse

Workup. No workup is required.

Treatment
- Ophthalmic referral is recommended.
- A protective eye shield should be applied.
- Cataracts, scleral ruptures, retinal tears, and/or retinal detachments should be surgically managed.
- Any associated hyphema should be treated as previously described.
- Any case of suspected child abuse should be reported to the appropriate authorities.

Lacerations and Perforations

Description
Most ocular injuries present with obvious redness and pain. However, some injuries provide few warning signs. For example, a sharp perforation may produce minimal redness and escape attention. In all cases of perforating lid injuries the possibility of an ocular perforation should be considered (Plate 39). Failure to recognize a perforating eye injury and to initiate antibiotic therapy may result in loss of the eye from endophthalmitis, a severe infection within the globe.

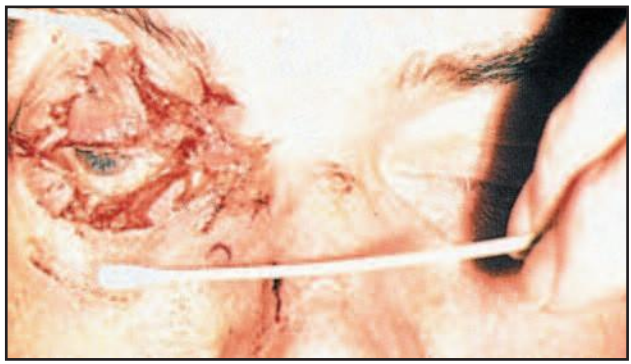


Plate 39
A severe injury to the lids from a dog bite

Workup. No workup is required.

Treatment

- Ophthalmic referral is recommended.
- A protective eye shield should be applied.
- Tetanus toxoid injection is given if updating required.

The following are the treatment options depending on the affected site:

(i) Lid — if the lid margin is involved, a suturing technique is critical to prevent notching.
(ii) Tear Drainage System — repair includes re-approximation of the severed canaliculi ends with internal stents to prevent chronic tearing.
(iii) Conjunctiva — if an isolated injury, repair is usually unnecessary.
(iv) Sclera — always suspect a puncture or laceration when the conjunctiva is involved; scleral laceration requires sutures and treatment with IV antibiotics to prevent endophthalmitis.
(v) Cornea — full-thickness lacerations require sutures, and puncture wounds that leak can be glued with tissue adhesives.
(vi) Lens — cataract extraction is indicated for this injury.

Decreased Vision in a White Eye

Editor's Note

The ability to diagnose a sudden decrease in vision is a crucial skill for emergency clinicians. There are various severe conditions that can endanger eyesight by affecting the retina, optic nerve, or brain. Some potential underlying risk factors include hypertension and atherosclerosis (which can lead to vein or artery occlusion), high myopia, a history of cataract surgery, or trauma (which may cause retinal detachment), diabetes, advanced age (which could result in maculopathy or vitreous hemorrhage), multiple sclerosis (associated with optic neuritis), giant cell arteritis (linked to optic neuropathy), stroke (causing cortical blindness), or elevated intraocular pressure (causing glaucoma). Timely intervention for conditions like central retinal artery occlusion, ischemic optic neuropathy caused by giant cell arteritis, glaucoma, or retinal detachment can significantly preserve eyesight

Vein Occlusion

Description

The presence of scattered superficial retinal hemorrhages dilated tortuous retinal veins, and cotton-wool spots may indicate a central retinal vein occlusion (CRVO) or a branch retinal vein occlusion (BRVO) (Plates 40, 41, Fig. 21). In CRVO the hemorrhages are located primarily at the posterior pole but may be seen throughout the fundus; in BRVO the hemorrhages are located in the distribution of the occluded vein. Vein occlusions are often encountered in older patients with hypertension and arteriosclerotic vascular disease. Carotid occlusion may produce a similar fundus picture. In rare cases, diseases that alter blood viscosity, such as polycythemia vera, sickle-cell disease, and leukemia induce retinal vein occlusions.

The acute hemorrhages and disc swelling resolve with time; however they may be followed by the development of shunt vessels from the choroidal circulation to the retina and ocular neovascularization.

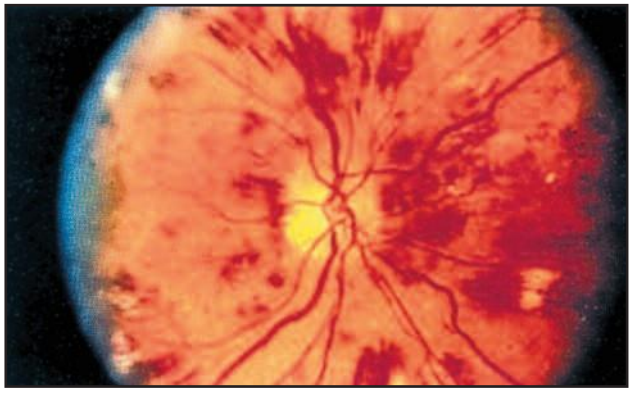


Plate 40
Central vein occlusion with scattered hemorrhages and cotton-wool spots

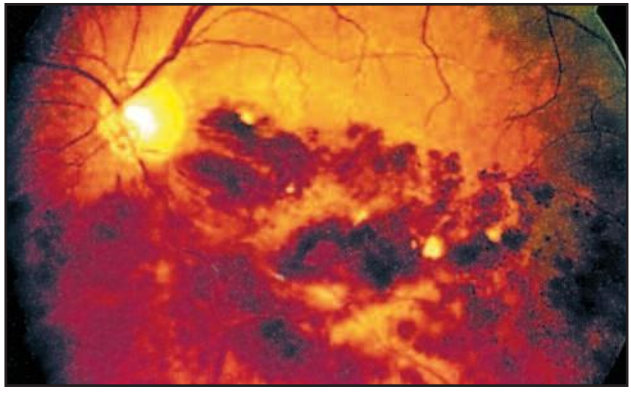


Plate 41
Branch vein occlusion with scattered hemorrhages throughout the inferior retina

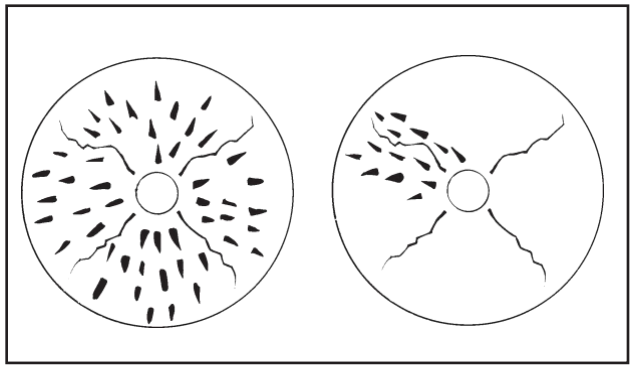


Fig. 21 Scattered superficial retinal hemorrhages are indicative of central retinal vein occlusion (left) or branch retinal vein occlusion (right).

Workup

- Ophthalmic referral is recommended.
- The intraocular pressure in both eyes should be taken, since patients with vein occlusions have a higher incidence of glaucoma.
- Fluorescein angiography may be performed to determine the extent of retinal ischemia and/or macular edema. This technique involves the intravenous injection of fluorescein dye to demonstrate the integrity of the retinal and choroidal vasculature.
- Optical coherence tomography is a less invasive test that can identify the extent of macular edema.

Treatment

- Intravitreal injection of anti-vascular endothelial growth factor (VEGF) drugs is the primary treatment today to reduce the risk of macular edema.
- In CRVO, pan retinal laser photocoagulation is indicated if neovascularization develops of the iris or angle. Macular grid laser photocoagulation has not been found to improve vision in those with macular edema.
- In BRVO, macular grid laser photocoagulation may be indicated for chronic macular edema. If neovascularization of the retina develops, then focal laser photocoagulation may resolve the neovascular tufts and prevent a vitreous hemorrhage.

Artery Occlusion

Description

Both central retinal artery occlusions (CRAO) and branch retinal artery occlusions (BRAO) are characterized by ischemic whitening of the retina (Plates 42, 43). Permanent visual loss may be preceded by periods of transient monocular visual loss called amaurosis fugax. The report of a period of visual loss in one eye lasting for several minutes should prompt an investigation of the ipsilateral carotid circulation to seek the presence of an atheroma, which may be the source of emboli that transiently interrupt blood flow to the retina. These patients should be referred to an ophthalmologist, neurologist, or vascular surgeon. In CRAO the fovea appears as a cherry-red spot, since the choroidal vasculature is easily visible through this relatively thinned retinal area (Fig. 22). Central visual acuity may rarely be normal

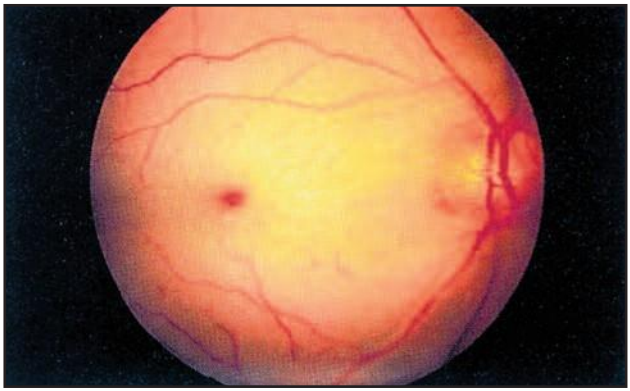


Plate 42
Central retinal artery occlusion as characterized by a cherry-red spot on the fovea

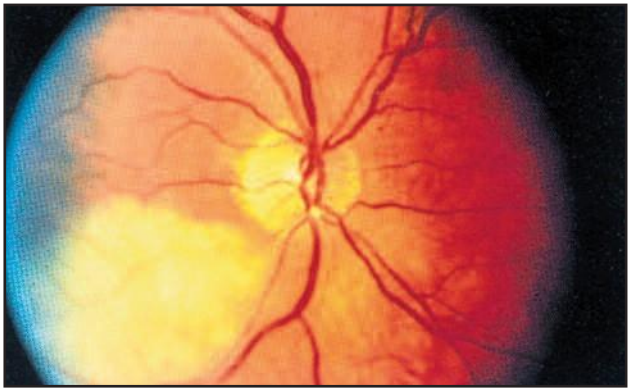


Plate 43
Branch retinal artery occlusion as characterized by an embolus on the disc and ischemic whitening of the inferotemporal retina

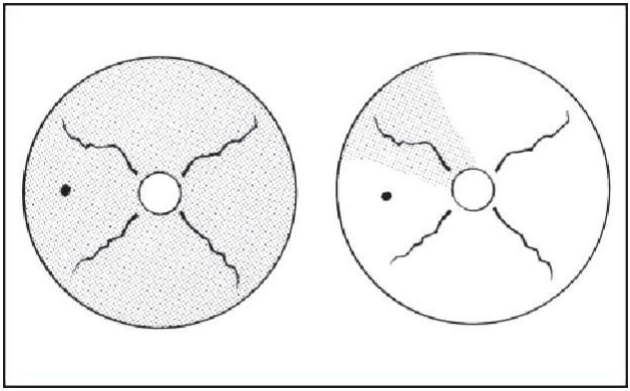


Fig. 22 Ischemic whitening of the retina is indicative of a central retinal artery occlusion (left) or a branch retinal artery occlusion (right).

in CRAO, if the blood supply from the choroidal vasculature to the fovea is maintained by a small retinal artery (cilioretinal artery). Most occlusions are caused by emboli that may be seen on the disc in CRAO or in an artery in BRAO.

A chronic cherry-red spot is also a feature of metabolic storage diseases such as Tay-Sachs disease and a variant of Niemann-Pick disease in which the ganglion cells become swollen as a result of the deposition of metabolic substances.

The disc, which is supplied by other branches of the ophthalmic artery, does not swell unless the occlusion is in the ophthalmic or carotid artery, proximal to the origin of the central retinal artery. The characteristics of the eye's vascular supply explain the preservation of some vision in the presence of a complete CRAO. If part of the retina derives its blood supply from the choroidal circulation via a cilioretinal artery, its function is spared. Since cilioretinal arteries are relatively common anomalies, present in up to 25% of eyes, small islands of vision may be preserved after CRAO. If the territory of the cilioretinal artery includes both the macula and the disc, a visual acuity of 20/20 is possible but there is severe loss of peripheral field. After a CRAO the retinal edema slowly resolves, and the death of the ganglion cells and their axons leads to optic atrophy. Months later, the characteristic ophthalmoscopic appearance is a pale disc and a blind eye.

Workup

- The patient's history should be taken to determine whether cerebral transient ischemic attacks have occurred.
- The carotid arteries and the heart should be evaluated to determine the source of the emboli.

Treatment

- Less than four hours by history is a true emergency.
- Carbogen therapy (CO_2/O_2 mixture); CO_2 dilates retinal arterioles and O_2 increases oxygen delivery to ischemic tissues.
- The patient should be given an ocular massage, which can cause fluctuation in intraocular pressure to mechanically dislodge a clot.
- Pressure lowering drop (e.g., Travatan Z®, Lumigan®, Xalatan®, Betagan®), Diamox® 500 mg p.o.or i.e., and mannitol 20% 200 mL
- Ophthalmic referral is recommended.
- An anterior chamber paracentesis may be performed to rapidly lower the intraocular pressure in the hopes of dislodging the emboli.

Retinal Detachment

Description

When a retinal tear or hole develops, fluid from the vitreous cavity may accumulate beneath the retina resulting in a retinal detachment (Plate 44). The patient with a retinal detachment may have a history of floaters and flashing lights and then a shade over the vision in one eye. A detachment that is extensive enough to reduce visual function will produce a relative afferent pupillary defect in the involved eye. A visual field deficit is present and the retina appears white when elevated (Fig. 23).There is an increased risk of retinal detachment in patients with high myopia, aphakia, pseudophakia (especially if complicated by vitreous loss during surgery), or previous ocular trauma (e.g., paintball injury, boxing, or bungee jumping).

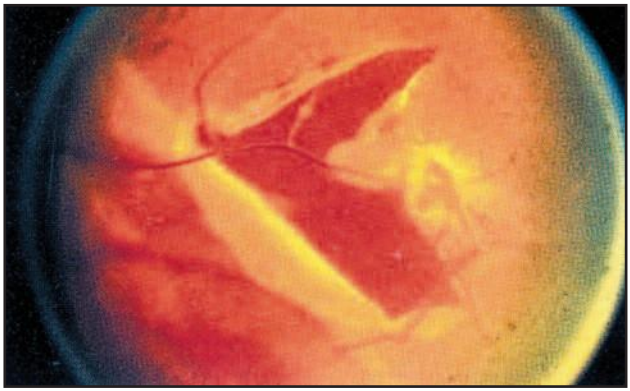


Plate 44
Large retinal tear with an associated retinal detachment

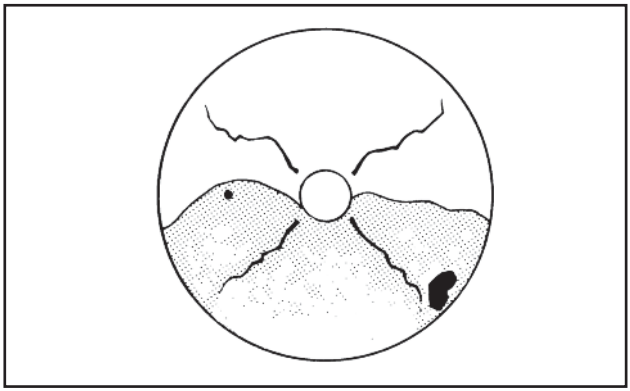


Fig. 23 In retinal detachment the retina appears white when elevated. If the macula is detached, the central vision will be diminished.

Workup. No workup is required.

Treatment

- Urgent ophthalmic referral is recommended.
- Surgical repair is required.
- If the patient's central vision is diminished (i.e., the macula is detached), there appears to be no difference in final visual acuity whether the surgery is performed immediately or after two or three days.
- Scleral buckling is the primary technique in which a silicone band indents the eye to approximate the retina. The tear is closed with cryotherapy or laser.
- Another surgical technique is the use of intraocular gas (i.e., pneumatic retinopexy) to tamponade the detachment with close follow-up of the intraocular pressure.
- Intraocular repair with pars plana vitrectomy may be necessary in complicated tractional and exudative detachment.

Maculopathy

Description

A decrease in vision often associated with metamorphopsia (wavy vision) suggests a macular problem. The macula may be affected acutely by edema, hemorrhage, and/or exudates. Unless the macular disease is extensive, a relative afferent pupillary defect is usually not present. Differential diagnosis includes diabetes mellitus (Plate 45), histoplasmosis, central serous retinopathy, and macular degeneration.

The visual changes in age-related macular degeneration (AMD) may be secondary to drusen, to degenerative changes in the retinal pigment epithelium, and to subretinal neovascular membranes with leakage (Plate 46). In the early stages of AMD, the transport of nutrients and wastes by the retinal pigment epithelium slows down. As waste products accumulate under the retina, they form yellowish deposits called drusen. The drusen may be small and discreet or larger with irregular shapes and indistinct edges. Patients with drusen alone tend to have normal or near-normal visual acuity with minimal metamorphopsia. Degenerative changes in the retinal pigment epithelium may occur with or without drusen. These degenerative changes are manifested as clumps of hyperpigmentation and/or depigmented atrophic areas (referred to as geographic atrophy). The effects on visual acuity vary.

Age-related macular degeneration can be categorized into two types: dry (non-neovascular) and wet (neovascular). The term "neovascular" pertains to the abnormal growth of new blood vessels in areas like the macula, where they shouldn't naturally occur. Roughly 10% of instances involving dry AMD evolve into the more severe and harmful wet AMD. The likelihood of AMD development rises as one gets older, exceeding 30% by the time they reach 75 years of age.

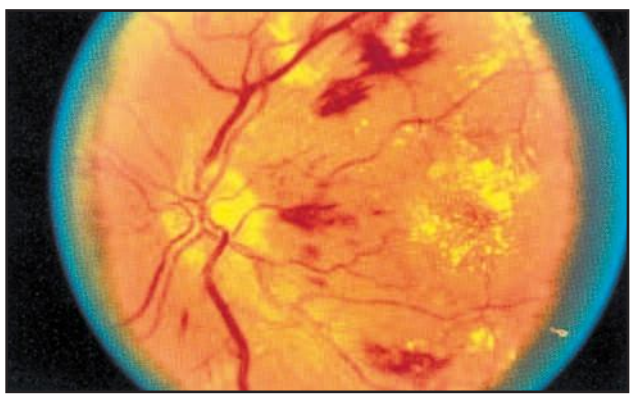


Plate 45
Background diabetic retinopathy as characterized by scattered retinal hemorrages and exudates

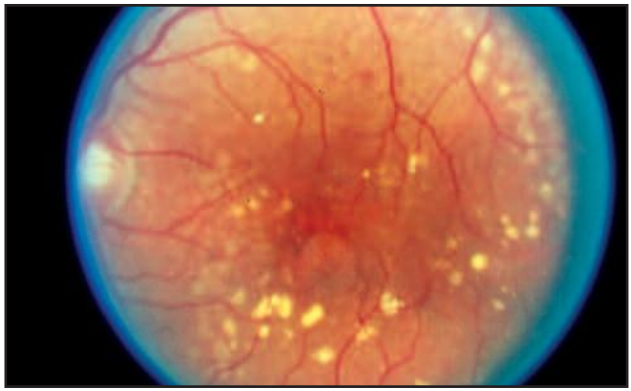


Plate 46
Macular drusen and pigment changes as seen in age-related macular degeneration

Workup

- Ophthalmic referral is recommended.
- Fluorescein angiography may be performed to determine the source of macular leakage.
- Optical coherence tomography (OCT) is a noninvasive test which records the features of the retina and displays this information in cross-sectional views.

Treatment

- If leaking vessels and/or microaneurysms are identified in diabetic patients, laser photocoagulation can be performed.
- In central serous retinopathy, a fluorescein angiogram will often identify a focal leakage point of the retinal pigment epithelium which causes an accumulation of fluid beneath the retina. The majority of cases resolve spontaneously. However, the course can be shortened by using a laser to seal the defect in the pigment epithelium. Acetazolamide (Diamox®) has been suggested to hasten the resolution of subretinal fluid.
- In the case of choroidal neovascularization in macular degeneration or histoplasmosis, laser photocoagulation can be applied if the vessels are not directly beneath the fovea otherwise photodynamic therapy is the preferred option.
- The most popular treatment for choroidal neovascularization is the injection of anti-VEGA agents into the vitreous cavity to prevent blood vessel growth and leakage. These drugs are required every 1 to 2 months.
- Nutritional supplements may help prevent or slow progression of macular degeneration (e.g., I Caps® AREDS Multivitamin, I Caps® with Lutein & Zeaxanthin, or PreserVision®).

Vitreous Hemorrhage

Description

Patients with a vitreous hemorrhage may complain of cloudy vision or the perception of shadows with cobwebs or floaters. Vitreous hemorrhage is characterized by a hazy view of the fundus with a reduced or altered red reflex (Plate 47). The most common causes are posterior vitreous detachment, proliferative diabetic retinopathy, vein

occlusion with neovascularization of the retina, retinal tear without detachment, macroaneurysm of the retina, and trauma. Treat as suspected ruptured globe if vitreous hemorrhage occurs in setting of trauma.

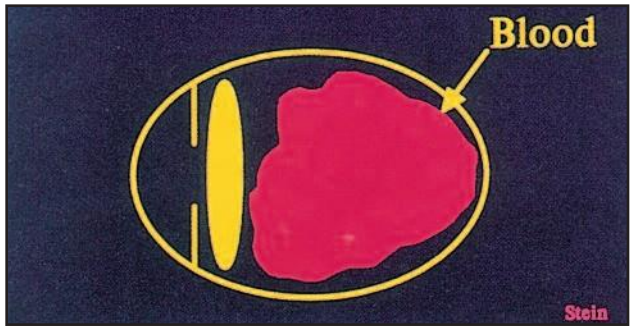


Plate 47
Diagram showing a vitreous hemorrhage that obscures visibility of the fundus

Workup

- Ophthalmic referral is recommended.
- A B-scan ultrasound should be performed to rule out an associated retinal detachment and/or mass lesion such as a malignant melanoma.

Treatment

- The majority of hemorrhages will resolve spontaneously in a few weeks to months.
- Vitrectomy may be indicated in non-clearing vitreous hemorrhages. It may also be combined with laser photocoagulation if there is an associated retinal tear or neovascularization, or a scleral buckle if there is a retinal detachment.

Optic Neuritis

Description

Optic neuritis is usually seen in patients between 20 and 50 years of age, who typically complain of a decrease in vision and pain with eye movement. Patients may complain of vision loss exacerbated by heat or exercise (Uhthoff phenomenon). Patients usually have decreased color vision. In acute optic neuritis the optic disc is swollen (Plate 48, Appendix J) in one-third of cases or it may appear normal in retrobulbar optic neuritis (Fig. 24) as seen in two-thirds of cases. Of these patients, over 50% will develop multiple sclerosis.

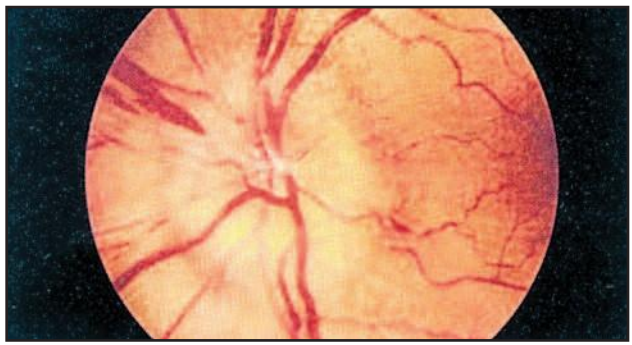


Plate 48
Swollen optic disc in optic neuritis. Papilledema has a similar appearance except this condition is bilateral and visual acuity is usually unaffected.

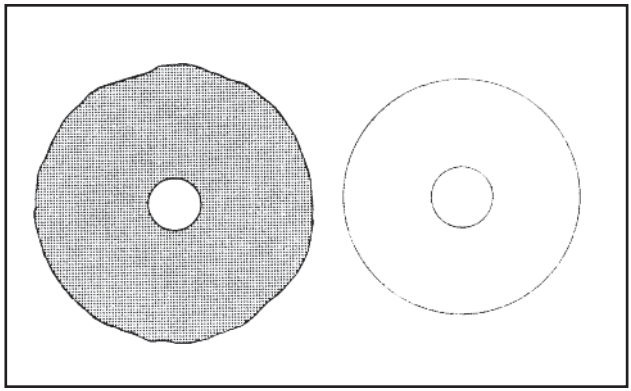


Fig. 24 Optic neuritis is characterized by a swollen optic disc and indistinct disc margins (left) or a normal appearing disc in retrobulbar optic neuritis (right).

Workup

- Ophthalmic referral is recommended.
- The visual field should be checked, and a follow-up test performed to determinethe course of visual loss.
- Brain MRI should be performed to rule out demyelination.

Treatment

- No specific ocular therapy is generally indicated.
- Controlled studies of systemic steroids have failed to demonstrate any difference in long-term visual outcome between treated and untreated groups.
- Intravenous steroids (methylprednisolone) may decrease the short-term risk of development of multiple sclerosis in patients with CNS white matter plaques but have no long-term protective benefit from multiple sclerosis.
- Although intravenous steroids do not affect the ultimate visual acuity in patients with optic neuritis, they do speed the rate recovery. Some clinicians advocate intravenous methylprednisolone 250 mg q6h for 3 days, followed by oral prednisone 1 mg/kg/day for 11 days, in patients with severe visual loss or bilateral visual loss.

Ischemic Optic Neuropathy

Description

Ischemic optic neuropathy is usually seen in patients over 50 years of age and is characterized by a sudden decrease in vision and a swollen optic disc (Fig. 25, Plate 49). The disease process is idiopathic in the majority of cases. The cause is thought to be on the basis of atherosclerosis with its effect on the circulation of the optic nerve head. Occasionally the condition is secondary to giant cell arteritis (GCA). This is an inflammatory condition of medium to large arteries with a predilection for extradural cranial arteries including the ophthalmic vessels. The symptoms and signs of GCA are headache, jaw claudication, and temporal artery tenderness, but it is important to note that patients with GCA may exhibit only visual symptoms.

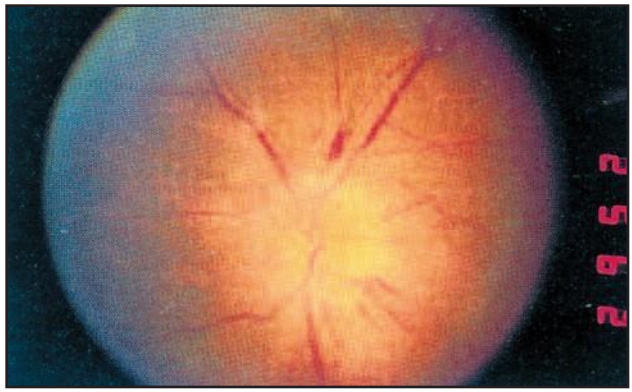


Plate 49
Ischemic optic neuropathy with swelling of the optic disc and a few surrounding hemorrhages

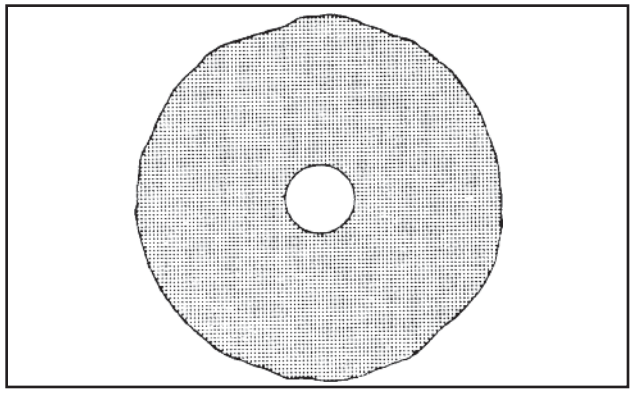


Fig. 25 Ischemic optic neuropathy is accompanied by a pale swelling of the optic disc and indistinct disc margins.

Workup

- The erythrocyte sedimentation rate (ESR) should be obtained to rule out GCA. In this condition the ESR is usually elevated.
- Other blood tests, such as the C-reactive protein (CRP), have been found useful in diagnosing giant cell arteritis.
- Temporal artery biopsy is the definitive test to confirm the diagnosis. Biopsy of the temporal artery may demonstrate the findings of giant cells, fragmentation of the elastica with surrounding chronic inflammation and occlusion of the vessel.
- Bilateral temporal artery biopsy should be considered if giant cell arteritis is still suspected despite an initial negative result of the temporal artery biopsy.
- Biopsy should be performed within 4 weeks of initiation of steroid treatment, although positive biopsy results can be obtained months after steroids have begun.

Treatment

- No specific treatment exists for idiopathic ischemic optic neuropathy.
- Ophthalmic referral is recommended.
- If GCA is suspected, systemic steroids should be started immediately to protect the patient from bilateral visual loss.
- Systemic steroids do not compromise biopsy results and may protect fellow eye while awaiting confirmation of diagnosis by biopsy.
- Steroids are usually tapered and maintained for a minimum of 9 to 12 months.
- Consultation with a rheumatologist is advisable if the diagnosis of giant cell arteritis is confirmed.
- Numerous complications of steroid use require medical monitoring with the help of a primary care physician or internist.

Cortical Blindness

Description

Patients with cortical blindness present with acute loss of vision in both eyes, resulting from a stroke to the occipital or visual cortex of the brain. Because the pathways serving the pupillary light reflex are separate from those carrying visual information, the patient with cortical blindness has normal pupillary reactions. This finding along with a normal ophthalmoscopic examination helps establish the diagnosis. Some patients with cortical blindness show some improvement; others expire due to severe neurological damage.

Workup

- CT scan of the brain.
- Neurologic referral.
- Ophthalmic referral to document extent of visual loss.

Treatment. No specific treatment is available.

Understanding Glaucoma: An Overview

Description

The term glaucoma refers to a group of diseases that have in common involvement of the optic nerve associated with loss of visual function. Elevated intraocular pressure (IOP) is the primary risk factor for the disease. There is no clear IOP level below which IOP can be considered "normal" or safe and above which can be considered "elevated" or unsafe. It has been shown that some eyes undergo damage to the optic nerve at IOP of 18 mmHg or less, whereas others tolerate IOPs in the 30s. Despite these findings IOP is considered the primary risk factor for glaucoma and the only factor that can be effectively altered with medical or surgical treatment.

Occasionally the IOP can be normal despite progressive changes to the optic nerve and visual field. These cases are referred to as normal-tension glaucoma. In order to make an accurate diagnosis of low-tension glaucoma one needs to take into consideration the artifact in IOP measurements caused by variation in central

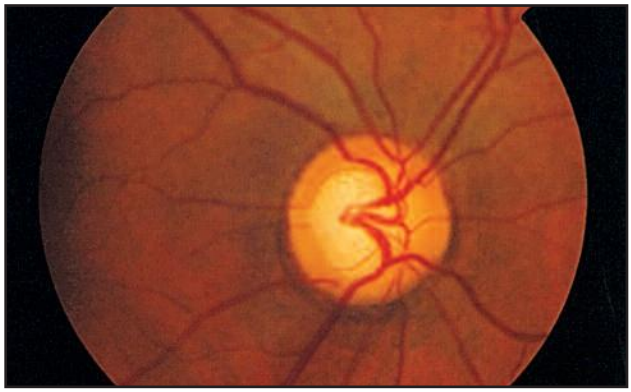


Plate 50
Glaucoma in a large physiologic disc. Patient has a large cup and a large disc. However, the superior and inferior rim tissue are thin by comparison to the temporal rim tissue.
Source: Litwak AB, ed. Glaucoma Handbook. Butterworth-Heinemann, 2001

corneal thickness (normal variation versus thinning or thickening secondary to an underlying disease or previous surgery) and that occurs with the diurnal fluctuation in pressure. In normal individuals, IOP varies 2-6 mmHg over a 24-hour period. A diurnal fluctuation of IOP of greater than 10 mmHg is suggestive of glaucoma.

Clinical evaluation of the optic discs allows for the diagnosis of glaucoma (Plate 50). Findings suggestive of glaucoma are:

- large optic cup
- asymmetry of optic cups between the patient's eyes
- progressive enlargement of optic cup
- focal enlargement (notching) of optic cup
- cupping to the rim margin
- splinter, or nerve fiber layer, hemorrhages on or near the disc
- nerve fiber layer loss

The two most common forms of glaucoma are primary open-angle glaucoma (POAG) and acute angle closure glaucoma. Patients with POAG usually have no symptoms until the late stage of the disease when the central vision is affected. The diagnosis is typically made on a routine visit to the eye doctor at which time the IOP is found to be elevated. There may be changes to the optic disc, a characteristic visual field defect, and/or a reduction in the thickness of nerve fiber layer of the retina. With a diagnosis of POAG patients are started on topical medication to reduce the IOP.

If a diagnosis of POAG is made in an emergency department it is usually because the patient was aware of loss of central vision. This is an unfortunate situation since the vision is irrevocably lost. It is important for all patients to have a comprehensive eye evaluation by an eye doctor to detect the early stages of disease. POAG if detected early can be successfully managed with either medical or surgical treatment to preserve vision.

The other most common form of glaucoma is primary angle closure glaucoma. Patients usually present with severe eye pain, nausea, and occasionally vomiting. There is usually diminished vision, redness, corneal swelling, mid-dilated pupil, and elevated IOP. Patients at higher risk for angle closure glaucoma include those

Table II Classification of glaucoma

Type	Characteristics
Open-angle glaucoma	
Primary open-angle glaucoma (POAG)	Usually associated with elevated IOP; not associated with other ocular or systemic conditions
Normal-tension glaucoma	IOP not elevated, No secondary causes
Juvenile open-angle glaucoma	Open-angle glaucoma diagnosed at a young age (Usually 10 to 30 years of age)
Glaucoma suspect	Elevated IOP with normal optic disc and visual field
Secondary open-angle glaucoma	Elevated IOP secondary to a variety of conditions such as pigmentary glaucoma, photolytic glaucoma, steroid-induced Glaucoma, pseudoexfoliation, angle-recession Glaucoma.
Angle-closure glaucoma	
Acute angle-closure glaucoma	Peripheral iris in contact with trabecular meshwork. anterior chamber becomes very shallow; movement of aqueous humor from posterior chamber to anterior chamber is restricted
Subacute angle closure	Repeated brief episodes of angle closure with mild symptoms and elevated IOP; often a prelude to acute angle closure
Chronic angle closure	IOP elevation occurs gradually as portions of the anterior chamber angle closed by peripheral anterior synechiae
Secondary angle-closure glaucoma	May be secondary to a swollen cataract or secluded pupil that is bound down to the lens (posterior synechiae)
Plateau iris syndrome	An anatomic variation in the iris root in which narrowing of the angle occurs independent of pupillary block
Childhood glaucoma	
Primary congenital glaucoma	Present from birth to first few years of life
Glaucoma associated with congenital anomalies	Secondary to ocular disorders e.g., aniridia, anterior segment dysgenesis
Secondary glaucoma in infants and children	Secondary to retinoblastoma or trauma

Modified from American Academy of Ophthalmology, Basic and Clinical Science Course, Glaucoma, 2020

that are hyperopic (smaller axial length of the eye and narrower anterior chamber), or of Inuit or Asian descent. As the anterior chamber decreases in depth and volume with age, acute angle closure glaucoma is most common between the ages of 55 and 65 years, although it can occur in young adults and has been reported in children.

The treatment of acute angle closure glaucoma is a true medical emergency. Urgent treatment of the elevated IOP should be initiated and then the patient promptly seen by an ophthalmologist for a laser iridotomy. If the IOP is significantly elevated for a long duration, this results in greater corneal edema, which makes the iridotomy procedure more difficult to perform. The initial emergency room treatment may

Table III Glaucoma medications

Class/ Compound	Brand Name	Strength	Method of Action	Method of Action	IOP Decrease
Prostaglandin analogs					
Travoprost	Travatan Z®	0.004%	qid	Increase uveoscleral outflow	25-32%
Bimatoprost	Lumigan®	0.01%	qid		
Latanoprost	Xalatan®	0.005%	qid		
Tafluprost	Zioptan®	0.0015%	qid		
B-adrenergic antagonists (beta-blockers)					
Betaxolol	Betoptic S®	0.25%	b.i.d.	Decrease aqueous production	15-20%
Levobunolol	Betagan®	0.25/0.5%	b.i.d		
Timolol	Timoptic®	0.25/ 0.5%	b.i.d		
Metipranolol	OptiPranolol	0.3%	b.i.d		
Adrenergic Agonists					
Dipivefrin HCL	Propine®	0.1%	b.i.d.	Improve aqueous outflow	15-20%
A2-adrenergic agonists					
Apraclonidine HCl	Iopidine®	0.5, 1.0%	b.i.d., t.i.d.	Decrease aqueous production	20-30%
Brimonidine	Alphagan®	0.2%	b.i.d., t.i.d.	Decrease episcleral venous pressure Decrease aqueous production Increase uveal scleral flow	
Parasympathomimetic (miotic) agents					
Pilocarpine HCl	Isopto Carpine®	0.2-4%	b.i.d., q.i.d.	Increase trabecular outflow	15-25%
Carbachol	Isopto Carbachol®	5-3%	b.i.d., t.i.d.	Same as above	15-25%
Carbonic Anhydrase Inhibitors					
Acetazolamide	Diamox®	62.5, 125, 250 mg	b.i.d., q.i.d.	Decrease aqueous production	15-20%
	Diamox Sequels®	500 mg	qd, b.i.d.		up to 23.5%
Methazolamide	Neptazine®	25,50 mg	b.i.d, t.i.d.		
Brinzolamide	Azopt®	1%	b.i.d., t.i.d.	Decrease aqueous humor secretion	
Dorzolamide	Trusopt®	2%	t.i.d.		
Hyperosmotic Agents					
Mannitol (i.v.)	Osmitrol®	20%	2 g/kg body wt.	Osmotic gradient dehydrates vitreous	
Glycerin (oral)		50%	4-7 oz	Same as above	
Fixed Combinations					
Timolol/Travoprost	Duotrav®	0.5%/0.004	qid	Same as nonselective beta-blocker and travopost	32-38%
Timolol/Latanoprost	Xalacom®	0.5%/0.005%		Same as nonselective beta-blocker and Latanoprost	
Timolol/bimatoprost	Ganfort®	0.5%/0.03%		Same as nonselective beta-blocker and Bimatoprost	
Timolol/ Brinzolamide	Azarga®	0.5%/1.0%	b.i.d.	Decrease aqueous humor secretion Reduce aqueous humor formation and a slight increase in outflow facility	up to 35%

consist of pilocarpine 2% q. 5 min x 4, then q. 1h., a pressure lowering drop (e.g., Travatan Z®, Lumigan®, Xalatan®) q. 12 h., isosorbide 1-2 gm/kg p.o. x 1, acetazolamide (Diamox®) 250 mg q. 6 h. or 500 mg i.v., and mannitol i.v. 205 1-2 gm/kg.

Glaucoma can be classified into one of three categories as described in Table II.Table III outlines the available classes of medications to treat glaucoma. The list is not inclusive as it is intended to demonstrate the different classes of medications followed by one or two examples.

Careful follow-up and monitoring of the glaucoma patient is important to determine the success of treatment i.e., stability or progression of the disease. In addition to a simple clinical evaluation (checking IOP and evaluation of optic disc), sophisticated automated tests can be utilized to determine any progressive changes of the optic disc or nerve fiber layer.

In the treatment of open-angle glaucoma if medical therapy is not successful in lowering the IOP or if there is progressive damage to the optic nerve then a laser trabeculoplasty is considered. Laser trabeculoplasty is a technique whereby laser energy is applied to the trabecular meshwork in discrete spots, usually one half of the circumference of the trabecular meshwork (180 degrees) per treatment. Approximately 80% of patients with medically uncontrolled open-angle glaucoma experience a drop in IOP for a minimum of 6-12 months following a laser trabeculoplasty. Additional laser treatment may be helpful in some patients, especially if the entire angle has not been treated previously.

Glaucoma incisional surgery is indicated for the treatment of glaucoma when a patient whose optic nerve function is failing on the maximum tolerated medical therapy and has not achieved a satisfactory response in IOP to laser treatment. The goal of filtration surgery is to create a new pathway for the flow of aqueous humor from the anterior chamber through a surgical opening in the sclera into the subconjunctival and sub-Tenon spaces.

Diplopia

Editor's Note

When evaluating a patient who reports experiencing double vision, it is crucial to ascertain whether this phenomenon occurs when viewing with a single eye or when both eyes are used together, and whether the double vision vanishes when one eye is covered. Double vision experienced with one eye is referred to as monocular diplopia, and it typically arises from non-neurological causes such as irregularities in the cornea (for example, epithelial basement membrane dystrophy) or lens issues like cataracts.

In contrast, genuine neurologic diplopia manifests as the perception of two distinct and equally bright images, where one image disappears upon closing one eye. Neurological sources of diplopia often involve impairments in the 3rd, 4th, or 6th cranial nerves. Swift identification of a nerve palsy and appropriate intervention can have life-saving implications. Imaging techniques like CT scans or MRIs might be necessary to investigate the cause further. It is important to rule out the possibility of an intracranial mass lesion, such as an aneurysm or tumor, which could be responsible for the nerve dysfunction.

Third Nerve Palsy

Description

The third nerve (oculomotor nerve) innervates four eye muscles: the superior rectus, the medial rectus, the inferior rectus, and the inferior oblique. It carries the parasympathetic fibers to the sphincter of the iris and innervates the levator muscle of the upper lid.

In third nerve palsy there may be diplopia, ptosis, and/or a dilated pupil (Plate 51). The eye is deviated out and down. In addition, with pupillary involvement there is paralysis of accommodation which causes blurred vision for near objects. The etiology of this condition is as follows: aneurysm 20%, vascular disease (diabetes, hypertension, athero-sclerosis) 20%, tumors 15%, trauma 15%, and miscellaneous and undetermined 30%.

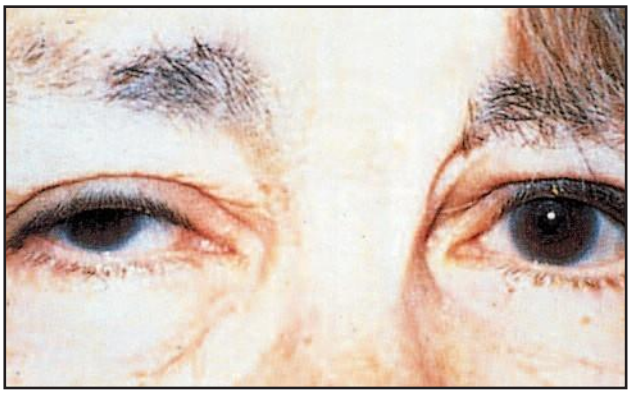


Plate 51
Third nerve palsy characterized by ptosis, dilated pupil, and deviation of the eye laterally

Workup

- If the pupil is fixed and dilated, other causes should be ruled out (e.g., Adie's pupil, or contamination with a dilating drop).
- If the pupillary dilatation is secondary to third nerve palsy, this constitutes a medical emergency and prompt neurosurgical referral is required. Cerebral angiography and CT scan should be ordered to rule out an intracranial aneurysm or neoplasm. MRI is a more sensitive imaging technique than a CT scan for identifying small brainstem lesions, such as infarction, small abscess, or tumor. If the pupil is not involved, diabetes, hypertension, collagen vascular disease, and GCA (if the patient is more than 55 years of age)should be ruled out.

Treatment

- Ophthalmic referral is recommended.
- The eye can be patched to alleviate diplopia.
- The majority of third nerve palsies not involving the pupil resolve within six months.
- If muscle weakness persists for more than 12 months, then surgery can be performed to improve cosmesis.

Fourth Nerve Palsy

Description

The fourth nerve (trochlear nerve) supplies the superior oblique muscle which moves the eye downward and inward. The palsy causes elevation of the eye with resultant vertical diplopia and involves a torsional component making images appear tilted. Congenital fourth nerve palsies may initially be asymptomatic and a head tilt (towards the opposite shoulder to minimize the diplopia) may be the only symptom; as image fusion ability diminishes over time, diplopia results. Acquired fourth nerve palsies are usually secondary to head trauma. The long course of the trochlear nerve makes it especially susceptible to injury in association with severe head trauma.

Workup

- If the fourth nerve palsy is isolated, it is not necessary to test for any underlying systemic diseases.

Treatment

- Ophthalmic referral is recommended.
- Prismatic correction in eyeglasses or surgical intervention may be indicated, depending on the severity and the duration of the palsy.

Sixth Nerve Palsy

Description

The sixth nerve (abducens nerve) innervates the lateral rectus muscle which moves the eye out. The palsy is characterized by horizontal diplopia (images side-by-side), most prominent in the field of gaze of the underactive lateral rectus muscle. The patient may be partially or completely unable to move the eye laterally (Plate 52).

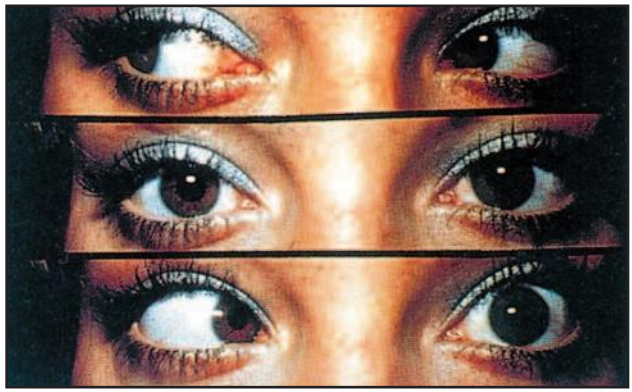


Plate 52
Sixth nerve palsy of the left eye characterized by limited motility on gaze to the left side (lower sequence)

Workup

- Obtain a patient history; children often have a history of a recent viral illness or immunization.
- In adults, diabetes, hypertension, collagen vascular disease, and GCA (if over the age of 55) should be ruled out.
- If sixth nerve palsy is not isolated (i.e., associated with other nerve palsies) or if the patient has papilledema, then a CT scan and/or MRI is indicated to rule out a neoplastic process.

Treatment

- Ophthalmic referral is recommended.
- Isolated sixth nerve palsies usually resolve spontaneously within six months.
- An eye patch can be placed over the affected eye, or if the patient wears eyeglasses, tape can be placed over the lateral portion of the lens.
- If muscle weakness persists for more than 12 months, then surgery can align the eyes in primary gaze.

Myasthenia Gravis

Description

Myasthenia gravis (MG) is an autoimmune condition that interferes with neuromuscular transmission in skeletal muscles. Although it can affect any muscle, ptosis and/or double vision are the presenting signs in approximately 50% of patients. MG may mimic any other ocular motility disorder including 3rd, 4thand 6th nerve disease. A history of intermittent diplopia or ptosis that is worse later in the day is suggestive of MG.

Workup

- Neurologic referral is suggested.
- Ophthalmic referral is recommended.
- Patients with unexplained diplopia and/or ptosis should have an edrophonium chloride (Tensilon) test to rule out myasthenia gravis.
- With severely drooping eyelids the ice pack test may be performed. Ice packs are placed on the lids with the eyes shut in a dark room for a few minutes. If the drooping improves after the ice application, then this is suggestive of MG.
- About 85% of people with MG have unusually high levels of acetylcholine receptor antibodies in their blood. Approximately 6% patients have muscle-specific kinase (MuSK) antibodies. Antibodies may not be detected in less than 10% of MG patients.
- Electromyogram (EMG): An EMG measures the electrical activity of muscles and nerves. This test detects communication problems between nerves and muscles.

Treatment

- Systemic medication for MG to be initiated by an internist.

Orbital Disease

Description

There are a variety of orbital diseases that can result in diplopia. Graves' disease is characterized by inflammation of the extraocular muscles leading to a restriction in ocular motility (Plate 53). Patients with Graves' disease may have proptosis, periorbital and conjunctival edema, eyelid retraction, and eyelid lag. Primary or secondary tumors of the orbit can also result in proptosis with limited motility. Unlike a third, fourth, or sixth nerve palsy, other associated findings of orbital diseases may include loss of vision, a relevant afferent pupillary defect, and proptosis.

Orbital tumors can be benign or malignant and arise primarily within theorbit or secondarily from an adjacent source, such as the eyelid, paranasal sinus, or intracranial compartment. In adults the most common benign tumors are meningiomas, mucoceles, and cavernous hemangiomas. Common malignant tumors include lymphoma, squamous cell carcinoma, and metastatic disease.

Workup

- Ophthalmic referral is recommended
- CT scan or MRI of the orbits should be performed

Treatment

- Varies depending on the presumed diagnosis
- Ocular management of mild cases of Graves' disease is treated with lubricant eye drops. Severe cases threatening vision (corneal exposure or optic nerve compression) are treated with steroids or orbital decompression. Double vision can be corrected with prism glasses and surgery (the latter only when the process has been stable for a while).
- Eyelid muscles can become tight with Graves', making it impossible to completely close the eyes. Difficulty closing the eyes can be treated with lubricant gel at night, or with tape on the eyes to enable full sleep. Eyelid surgery can be performed on upper and/or lower eyelids to reverse the effects of Graves'.
- Orbital decompression can be performed to enable bulging eyes to be retracted again. In this procedure, bone is removed from the skull behind the eyes, and space is made for the enlarged muscles and fatty tissue to be moved back into the skull.
- Sphenoid wing meningiomas are treated with debulking via craniotomy when symptomatic, sometimes followed by a course of radiation therapy. Because meningioma cells infiltrate bone of the skull base, complete resection usually

is not possible. Mucoceles are treated by draining the offending lesion into the nose, because they most commonly arise from the ethmoid or frontal sinus. Cavernous hemangiomas are excised. Lymphomas involving the orbit are typically B-cell and characteristically low grade. Lymphomas can be bilateral and simultaneous and can be part of a systemic process or exist in the orbit in isolation. Radiation therapy effectively treats orbital lymphomas with few adverse effects, although the addition of monoclonal antibodies against a surface receptor (CD20) on the lymphocyte is also effective. Most squamous cell carcinomas arise from the adjacent paranasal sinuses. Surgery, radiation therapy, or both form the backbone of therapy. Metastatic disease is usually treated with radiation therapy. Metastatic disease involving the orbit is usually an unfavorable prognostic sign, with carcinoid tumors being a notable exception.

Appendixes

Editor's Note

This section should stimulate the clinician to improve their diagnostic skills. The appendixes include important summaries or differential diagnoses of a variety of ophthalmic complications or conditions. The astute clinician will recognize the ocular complications from systemic diseases or medications, identify lifesaving ocular signs, and understand the potential complications from ophthalmic surgical procedures. A knowledgeable clinician will know the differential diagnosis of a variety of conditions such as the patient who presents with a nontraumatic red eye (acute conjunctivitis, acute iritis, acute glaucoma), conjunctivitis (viral, bacterial, allergy), or swollen optic disc (optic neuritis, optic neuropathy, papilledema).

Appendix A Ocular complications of systemic diseases

Disease	Possible Ocular Findings
Diabetes mellitus	Background retinopathy: retinal hemorrhages, exudates and microaneurysms.
	Preproliferative retinopathy: cotton-wool spots, intraretinal microvascular abnormalities.
	Proliferative retinopathy: neovascularization, preretinal hemorrhage, vitreous hemorrhage, retinal detachment.
Graves' disease	Lid retraction, lid lag, exposure keratopathy, chemosis and injection, restriction of eye movements, proptosis, compressive optic neuropathy.
Hypertension	Sclerosis of vessels in longstanding disease; narrowing of vessels, retinal hemorrhages, and/or exudates in severe hypertension.
Rheumatoid arthritis & other collagen vascular diseases	Dry eye, episcleritis, scleritis, peripheral corneal ulceration and/or melting.
Cancer	Metastatic disease to choroid may result in retinal detachment; disease in the orbit can result in proptosis and restriction of eye movements. (e.g., breast, lung cancer)
Sarcoidosis	Dry eye, conjunctival granulomas, iritis, retinitis.
AIDS	Kaposi's sarcoma, cotton-wool spots of retina, cytomegalovirus retinitis.

Appendix B Ocular complications of systemic medications

Medication	Ocular Complications
Amiodarone	Superficial whorl-like keratopathy
Chlorpromazine	Anterior subcapsular cataracts
Corticosteroids	Posterior subcapsular cataracts, glaucoma
Digitalis	Blurred vision, disturbed color vision
Ethambutol	Optic neuropathy
Hydroxychloroquine	Superficial keratopathy and bull's-eye maculopathy
Indomethacin	Superficial keratopathy
Isoniazid	Optic neuropathy
Nalidixic acid	Papilledema
Sildenafil	Optic neuropathy (controversial)
Tamsulosin	Cataract surgery complications 2º to floppy iris and intraoperative miosis
Tetracycline	Papilledema
Thioridazine	Pigmentary degeneration of the retina
Vitamin A	Papilledema

Appendix C Life-threatening ocular signs	
Findings	**Clinical Significance**
White pupil	In an infant retinoblastoma must be ruled out.
Aniridia (iris appears absent)	May be autosomal dominant (2/3s) or sporadic inheritance. In sporadic cases where the short arm of chromosome 11 is deleted, there is a 90% risk of developing Wilms' tumor; the risk in other sporadic cases is approximately 20%.
Thickened corneal nerves (slit lamp)	Part of the multiple endocrine neoplasia syndrome type IIB. Must rule out medullary carcinoma of the thyroid, pheochromocytoma, and parathyroid adenomas.
Retinal angioma	May be part of the Von Hippel-Lindau syndrome. Autosomal dominant inheritance with variable penetrance. Must rule out hemangioblastomas of the central nervous system, renal cell carcinoma, and pheochromocytoma.
Multiple pigmented patches of fundus	Lesions represent patches of congenital hypertrophy of the retinal pigment epithelium. May be part of Gardner's syndrome characterized by multiple premalignant intestinal polyps together with benign soft tissue tumors (lipomas, fibromas, sebaceous cysts) and osteomas of the skull and jaw. A complete gastrointestinal investigation is indicated. If a diagnosis of Gardner's syndrome is made, prophylactic colectomy is indicated because of the potential for malignant degeneration of colonic polyps.
Third nerve palsy with a dilated pupil	Must rule out an intracranial aneurysm or neoplastic lesion. CT scan and/or MRI should be performed on an emergency basis.
Papilledema	Must rule out an intracranial mass lesion. CT scan and/or MRI should be performed on an emergency basis.
Pigmentary degeneration of the retina and motility disturbance	May represents the Kearns-Sayre syndrome. Must rule out a cardiac conduction disturbance with an annual electrocardiogram. May develop an intraventricular conduction defect, bundle block, bifascicular disease, or complete heart block. Patient must be prepared for the possible need to implant a pacemaker.

Appendix D Calibration scale for Schiotz tonometers

Scale Reading (scale units)	Plunger Load (gm) 5.5	7.5	10.0	15.0
	Intraocular Pressure (mmHg)			
0	41	59	82	127
0.5	38	51	75	118
1.0	35	50	70	109
1.5	32	46	64	101
2.0	29	42	59	94
2.5	27	39	55	88
3.0	24	36	51	82
3.5	22	33	47	76
4.0	21	30	43	71
4.5	19	28	40	66
5.0	17	26	37	62
5.5	16	24	34	58
6.0	15	22	32	54
6.5	13	20	29	50
7.0	12	19	27	46
7.5	11	17	25	43
8.0	10	16	23	40
8.5	9	14	21	38
9.0	9	13	20	35
9.5	8	12	18	32
10.0	7	11	16	30
10.5	6	10	15	27
11.0	6	9	14	25
11.5	5	8	13	23
12.0		8	11	21
12.5		7	10	20
13.0		6	10	18
13.5		6	9	17
14.0		5	8	15
14.5			7	14
15.0			6	13
15.5			6	11
16.0			5	10
16.5				9
17.0				8
17.5				8
18.0				7

Appendix E

1. Differential diagnosis of nontraumatic red eye

	Condition		
Feature	**Acute Conjunctivitis**	**Acute Iritis**	**Acute Glaucoma**
Symptoms	Redness, tearing +/- purulent discharge, itching	Redness, pain, photophobia	Redness, severe pain, nausea, vomiting
Appearance	Conjunctival injection	Ciliary injection	Diffuse injection
Vision	Normal, can be blurred secondary to discharge	Moderate reduction	Marked reduction, halo vision
Cornea	Clear	May see keratic Precipitates	Hazy secondary to edema
Pupil	Normal	Small, sluggish to light	Semi-dilated nonreactive
Secretions	Tearing to purulent	Tearing	Tearing
Test & Comments	Smears may show etiology: bacterial infection = polycytes; viral infection = monocytes; allergy = eosinophils	Slit lamp will show cells and flare in the anterior chamber	Elevated intraocular pressure
Treatment	Antibiotic Vigamox® Besivance® Zymar®	Steroids Cycloplegics	Pilocarpine Travatan Z® Duotrav® Azarga® Diamox® Mannitol Laser surgery

2. Differential diagnosis of viral, bacterial, and allergic conjunctivitis

Feature	**Viral**	**Bacterial**	**Allergy**
Discharge	Watery	Purulent	Watery
Itching	Minimal	Minimal	Marked
Pre-auricular lymph node	Common	Absent	Absent
Stain & smear	Monocytes Lymphocytes	Bacteria Polycytes	Eosinophils

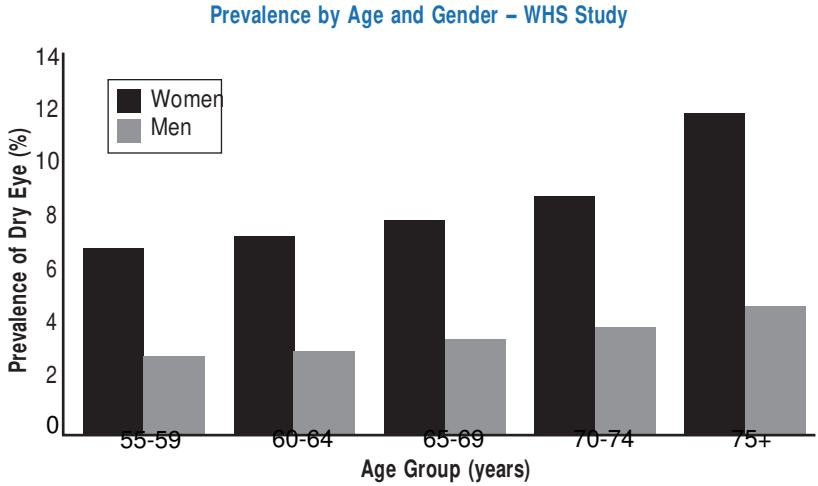
Appendix F Ocular complications of topical corticosteroids

Topical corticosteroids alone or in combination with antibiotics should not be administered to the eye by a primary-care physician. They can be helpful when used under the close supervision of an ophthalmologist.

Topical corticosteroids have three potentially serious ocular side effects:

1. **Keratitis**. Herpes simplex keratitis and fungal keratitis are both markedly potentiated by corticosteroids. These agents may mask symptoms of inflammation making the patient feel better while the cornea may be melting or even perforating.
2. **Cataracts**. Prolonged use of corticosteroids, either locally or systemically, will often lead to cataract formation.
3. **Elevated intraocular pressure**. Local application of corticosteroids may cause an elevation of intraocular pressure. Optic nerve damage and loss of vision can occur. The combination of a corticosteroid and an antibiotic carries the same risk.

Appendix G Prevalence of dry eye



Prevalence of Dry Eye

- Salisbury Study = 14.4%
- Melbourne Study = 5.5%
- Beaver Dam Study = 14.4%
- WHS Study = 6.7%

Although, percent of individuals who experience signs and symptoms of dry eye at one time or another due to environmental factors = 100%

Schaumberg et al. Epidemiology of Dry Eye Syndrome. Adv Exp Med Biol 2002; 506(Pt B): 989-998.

Appendix H Influential factors of dry eye

Drying Systemic Medications

- β-Adrenergic-blocking, anti-anginals, and anti-hypertensives
  (e.g., Atenolol, Practolol, Propranolol)
- Tricyclic anti-depressants
  (e.g., Amittriptyline, Doxepin)
- Oral antihistamines
  (e.g., Loratadine, Clemastine, Hydroxyzine)
- Alkylating immunosuppressives
  (e.g., Busulfan, Cyclophosphamide)
- Diuretics
  (e.g., Triamterene)

Other Drying Factors

- Age
- Gender
- Arthritis
- Osteoporosis
- Gout
- Lens surgery
- Contact lens wear
- Blink disorders
- Disorders of lid aperture
- Nutritional problems
- Rheumatoid arthritis
- Thyroid problems
- Time of day
- LASIK surgery
- Cosmetic surgery
- Gender
- Mechanical disturbances
- Exposure keratitis
- Entropion
- Ectropion
- Symblepheron formation
- Large lid notches
- Lagophthalmos
- Incomplete blinking
- Dellen formation
- Illumintation
- Temperature
- Humidity
- Air movement
- Allergies
- Change in environment
- Reading
- Watching movies
- Sleep

Prause JU, Norn M. Relation between blink frequency and break-up time. Acta Ophthalmol 1983; 61: 108-116.
Cho P, Cheung P, Leung K, Ma V, Lee V. Effect of reading on non-invasive tear break-up time and inter-blink interval. Clin Exp Optom 1997; 80: 62-68.
Tsubota K, Seiichiro H, Okusawa Y, Egami F, Ohtsuki T, Nakamori K. Quantitative videographic analysis of blinking in normal subjects and patients with dry eyes. Arch Ophthalmol 1996; 114(6): 715-720.
Nally L, Ousler GW, Abelson MB. Ocular discomfort and tear film break-up time in dry eye patients: a correlation. IOVS 2000; 41(4): 1436; *Collins M, Seeto R, Campbell L, Ross M.* Blinking, and corneal sensitivity. Acta Ophthalmologica 1989; 67(5): 525-531.
Abelson MB, Holly FJ. A tentative mechanism for inferior punctate keratopathy. Am J Ophthalmol 1977; 83: 866-869; *Doane MG.* Dynamics of the human blink. Ber Disch Ophthalmol Ges 1980; 77: 13-17.
Kaneko K, Sakamoto K. Spontaneous blinks as a criterion of visual fatigue during prolonged work on visual display terminals. Perceptual and Motor Skills 2001; 92(1): 234-250.

Appendix I Differential diagnosis of red eye in contact lens wearers

Diagnosis	Findings	Mechanism	Treatment
Corneal abrasion	Epithelial defect Stains with fluorescein	Mechanical Hypoxia	Antibiotic drops
Superficial punctate keratitis	Punctate corneal staining	Mechanical Chemical toxicity	Artificial tears
Giant papillary conjunctivitis	Papillary reaction of superior tarsal conjunctiva	Immunologic Mechanical	Mast cell stabilizer/ Antihistamine
Sterile infiltrates	Corneal infiltrate Epithelium usually intact	Immunologic	Antibiotic drops (assume infected)
Infected ulcer	Corneal infiltrate with ulceration stains with fluorescein	Pseudomonas, Staphylococcus aureus, etc.)	Corneal scraping Gram stain and culture. Antibiotic drops

Appendix J Differential diagnosis of swollen optic disc

	Optic Neuritis	Optic Neuropathy	Papilledema
Age	< 50 years	> 50 years	Any age
Visual acuity	Decreased	Decreased	Normal
Other symptoms	Pain, especially with eye movement	May have symptoms of giant cell arteritis, headache, jaw claudication, and scalp tenderness	May have associated neurological signs if there is a lesion, e.g., nerve palsy, weakness, or sensory loss in limbs
Disc appearance	Swollen occasionally Normal in retrobulbar optic neuritis.	Swollen	Swollen
Workup	Visual field — if atypical or if no improvement in follow-up a CT scan to rule out mass lesion	Stat ESR. If elevated perform temporal artery biopsy to confirm diagnosis.	Stat CT scan to rule out intracranial mass lesion
Treatment	None	If elevated ESR use systemic steroids. If temporal artery biopsy negative discontinue steroids.	Neurosurgical consult to consider surgery

Appendix K Postoperative ocular complications

Surgery	Symptoms	Possible Diagnosis	Ophthalmic Referral
Cataract	Redness, pain, decreased vision in the first week after surgery	Endophthalmitis (infection within the eye)	Stat
	Foreign body sensation, discharge	Loose suture	Urgent
	Diminished vision months after surgery	Opacified posterior capsule	Non-urgent
		Retinal detachment	Urgent
Corneal transplant	Redness, pain, decreased vision in the first week after surgery	Endophthalmitis (infection within the eye)	Stat
	Foreign body sensation, discharge	Loose suture	Urgent
	Decreased vision months after surgery	Graft rejection	Urgent
Retinal detachment (scleral buckle)	Decreased vision	Recurrent retinal detachment Vitreous hemorrhage	Urgent Urgent
Photorefractive keratectomy (PRK) or Corneal cross-linking (CXL)	Pain in first 48 hours	UV radiation effect and corneal abrasion	Urgent
		Corneal ulcer	Stat
Laser assisted in situ keratomileusis (LASIK)	Pain and decreased vision	Displaced corneal flap, infection, or interstitial keratitis	Stat
Blepharoplasty	Lid hemorrhage, pain, diminished vision	Periorbital/orbital hemorrhage	Stat
		Compression of optic nerve	Stat
Dacryocystorhinostomy	Periorbital hemorrhage	Bleeding into soft tissues	Stat

Note: Stat referral = seen within hours; Urgent referral = seen within 24 hours;
Non-urgent referral = seen within a few days

Appendix L Chronic conditions that may present with acute symptoms

Condition	Symptoms	Comment
Cataract	Decreased vision	Usually a slowly progressive decrease in vision. Occasionally a rapid deterioration in vision especially with posterior subcapsular cataracts that encroach on the visual axis. Cataract types named after anatomical location: cortical, nuclear, anterior and posterior subcapsular. Cataract surgery for visual improvement.
Corneal edema	Decreased vision Photophobia Tearing	May develop following cataract surgery (pseudophakic bullous keratopathy or aphakic bullous keratopathy), or with a corneal dystrophy. Corneal transplant surgery can restore vision.
Macular degeneration	Decreased vision Metamorphopsia	Usually a slowly progressive condition. If rapid decrease in vision or metamorphopsia (wavy vision), possible hemorrhage or exudation in macula. Fluorescein angiogram to determine leakage points. Laser photocoagulation may be of benefit. If there is new blood formation (i.e., choroidal neovascularization) then injections of anti-VEGA agents into the vitreous cavity may cause blood vessel regression.

Subject Index

About the Authors

Dr. Raymond Stein is the Medical Director of the Bochner Eye Institute and Professor of Ophthalmology and Vision Sciences at the University of Toronto. As a leader in refractive surgery, he has been honored with numerous awards from a variety of prestigious international medical organizations, including the American Academy of Ophthalmology, the International Intraocular Implant Club, and the Contact Lens Association of Ophthalmologists. Dr. Raymond Stein was elected to serve as President of the Canadian Society of Cataract and Refractive Surgery. Dr. Raymond Stein has been invited to lecture at numerous international medical conferences, hospitals, universities, and societies. He has written many peer-reviewed articles, book chapters, and books. He co-authored the first clinical textbook on laser vision correction titled The Excimer Laser. He has also served as editor of the scientific journals Clinical and Surgical Ophthalmology and Clinical Signs in Ophthalmology. He has established himself as an expert in refractive and implant surgery. His patients have travelled from over 65 countries for surgery at the Bochner Eye Institute. As a leader in the field, he was chosen to serve as the chief eye surgeon for the W Network's Style by Jury TV show. Dr. Raymond Stein was a Benjamin Franklin Scholar at the University of Pennsylvania and then completed a medical degree at the University of Toronto. After his residency at the Mayo Clinic in Rochester, Minnesota he completed a Cornea and External disease fellowship at the Wills Eye Hospital in Philadelphia. In addition to serving as the Medical Director at the Bochner Eye Institute, he is the Cornea Consultant at the Mount Sinai Hospital. Raymond Stein served as Chief of Ophthalmology at the Scarborough Hospital for 21 years. Dr. Raymond Stein is committed to providing high quality care to patients with a variety of ophthalmological needs. His surgical expertise is in performing cataract and implant surgery, laser vision correction, phakic implants, and keratoconus treatments including corneal collagen cross-linking and topography guided PRK

About the Authors

Dr. Rebecca Stein is a board-certified Ophthalmologist with advanced training in Canada, United States, and the United Kingdom. She completed her Ophthalmology residency at the University of Toronto. After graduation, she was chosen for a prestigious fellowship in Cornea and External disease, Cataract and Refractive Surgery in New York with world renowned Dr. Eric Donnenfeld. Dr. Stein is the great granddaughter of Dr Maxwell Bochner, the granddaughter of Dr. Harold Stein, and the daughter of Dr. Raymond Stein. She is the fourth generation ophthalmologist at the Bochner Eye Institute, a first in North America. Dr. Rebecca Stein has a strong interest in ophthalmic education and enjoys teaching medical students, residents, and eyecare professionals, alongside her clinical duties. She has been an invited speaker to the American Academy of Ophthalmology, American Society of Cataract Refractive Surgery, and Family Day in Eye Care. She is actively involved in clinical research and has had multiple peer reviewed papers published in the areas of cornea and refractive surgery, corneal cross-linking, and blepharoconjunctivitis. She is also a co-author of the 11th edition of The Ophthalmic Assistant text book. Dr. Rebecca Stein is committed to providing high-quality care, including cataract surgery, refractive lens exchange, laser vision correction, keratoconus treatment, implantable contact lenses, pterygium, and corneal transplantation. She offers numerous advanced refractive surgery procedures at Bochner Eye Institute utilizing the latest technologies and techniques