

Eye Injury

Montana Utilization and Treatment Guidelines

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**Presented by:
State of Montana**

**Department of Labor and Industry
EMPLOYMENT RELATIONS DIVISION**



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B. General Guideline Principles

The principles summarized in this section are key to the intended implementation of these guidelines and critical to the reader's application of the guidelines in this document.

1. APPLICATION OF GUIDELINES The Department provides procedures to implement medical treatment guidelines and to foster communication to resolve disputes among the providers, payers, and patients through the Administrative Rules of Montana. In lieu of more costly litigation, parties may wish to request an independent medical review from the Department's Medical Director prior to submitting a Petition for a Workers' Compensation Mediation Conference.

2. EDUCATION of the patient and family, as well as the employer, insurer, policy makers and the community should be the primary emphasis in the treatment of upper eye pain and disability. An education-based paradigm should start with communication providing reassuring information to the patient. A more in-depth education within a treatment regime employing functional restorative and innovative programs of prevention and rehabilitation is optimal. A treatment plan should address issues of individual and/or group patient education as a means of facilitating self-management of symptoms and prevention.

3. TREATMENT PARAMETER DURATION Time frames for specific interventions commence once treatments have been initiated, not on the date of injury. Obviously, duration will be impacted by patient compliance, as well as availability of services. Clinical judgment may substantiate the need to accelerate or decelerate the time frames discussed in this document.

4. ACTIVE INTERVENTIONS emphasizing patient responsibility, such as therapeutic exercise and/or functional treatment, are generally emphasized over passive modalities, especially as treatment progresses. Generally, passive interventions are viewed as a means to facilitate progress in an active rehabilitation program with concomitant attainment of objective functional gains.

5. ACTIVE THERAPEUTIC EXERCISE PROGRAM goals should incorporate patient strength, endurance, flexibility, coordination, and education. This includes functional application in vocational or community settings.

6. FUNCTIONAL IMPROVEMENT GOALS should be consistently addressed. Positive patient response results are defined primarily as functional gains that can be objectively measured. Objective functional gains include, but are not limited to, positional tolerances, range of motion (ROM), strength, endurance, activities of daily living, cognition, psychological behavior, and efficiency/velocity measures that can be quantified. Subjective reports of pain and function should be considered and given relative weight when the pain has anatomic and physiologic correlation. Anatomic correlation must be based on objective findings.

7. RE-EVALUATE TREATMENT EVERY 3 TO 4 WEEKS If a given treatment or modality is not producing positive results within 3 to 4 weeks, the treatment should be either modified or discontinued. Reconsideration of diagnosis should also occur in the event of poor response to a

seemingly rational intervention.

8. SURGICAL INTERVENTIONS should be contemplated within the context of expected functional outcome and not purely for the purpose of pain relief. The concept of “cure” with respect to surgical treatment by itself is generally a misnomer. All operative interventions must be based upon positive correlation of clinical findings, clinical course, and diagnostic tests. A comprehensive assimilation of these factors must lead to a specific diagnosis with positive identification of pathologic condition(s).

9. SIX-MONTH TIME FRAME The prognosis drops precipitously for returning an injured worker to work once he/she has been temporarily totally disabled for more than six months. The emphasis within these guidelines is to move patients along a continuum of care and return to work within a six-month time frame, whenever possible. It is important to note that time frames may not be pertinent to injuries that do not involve work-time loss or are not occupationally related.

10. RETURN-TO-WORK is therapeutic, assuming the work is not likely to aggravate the basic problem or increase long-term pain. The practitioner must provide specific physical limitations and the patient should be released to return to work with specific physical activity limitations clearly spelled out per the specific job requirement. Release to “sedentary” or “light duty” is not a specific physical limitation. The following physical limitations should be considered and modified as recommended: lifting, pushing, pulling, crouching, walking, using stairs, overhead work, bending at the waist, awkward and/or sustained postures, tolerance for sitting or standing, hot and cold environments, data entry and other repetitive motion tasks, sustained grip, tool usage and vibration factors. Even if there is residual chronic pain, return-to-work is not necessarily contraindicated.

The practitioner should understand all of the physical demands of the patient’s job position before returning the patient to full duty and should request clarification of the patient’s job duties. Clarification should be obtained from the employer or, if necessary, including, but not limited to, a health care professional with experience in ergonomics, an occupational health nurse, a physical therapist, an occupational therapist, a vocational rehabilitation specialist, or an industrial hygienist.

11. DELAYED RECOVERY Strongly consider a psychological evaluation, if not previously provided, as well as initiating interdisciplinary rehabilitation treatment and vocational goal setting, for those patients who are failing to make expected progress 6 to 12 weeks after an injury. The Department recognizes that 3 to 10% of all industrially injured patients will not recover within the timelines outlined in this document despite optimal care. Such individuals may require treatments beyond the limits discussed within this document, but such treatment will require clear documentation by the authorized treating practitioner focusing on objective functional gains afforded by further treatment and impact upon prognosis.

12. GUIDELINE RECOMMENDATIONS AND INCLUSION OF MEDICAL EVIDENCE are recommendations based on available evidence and/or consensus recommendations. When possible, guideline recommendations will note the level of evidence supporting the treatment

recommendation. When interpreting medical evidence statements in the guideline, the following apply:

Consensus means the opinion of experienced professionals based on general medical principles. Consensus recommendations are designated in the guideline as “*generally well accepted*,” “*generally accepted*,” “*acceptable/accepted*,” or “*well-established*.”

“*Some*” means the recommendation considered at least one adequate scientific study, which reported that a treatment was effective.

“*Good*” means the recommendation considered the availability of multiple adequate scientific studies or at least one relevant high-quality scientific study, which reported that a treatment was effective.

“*Strong*” means the recommendation considered the availability of multiple relevant and high quality scientific studies, which arrived at similar conclusions about the effectiveness of a treatment.

All recommendations in these guidelines are considered to represent reasonable care in appropriately selected cases, regardless of the level of evidence or consensus statement attached to it. Those procedures considered inappropriate, unreasonable, or unnecessary are designated in the guideline as being “not recommended.”

13. CARE BEYOND MAXIMUM MEDICAL IMPROVEMENT (MMI) should be declared when a patient’s condition has plateaued to the point where the authorized treating physician no longer believes further medical intervention is likely to result in improved function. However, some patients may require treatment after MMI has been declared in order to maintain their functional state. The recommendations in this guideline are for pre-MMI care and are not intended to limit post-MMI treatment.

C. Definitions

C.1 General Approach and Basic Principles

Unfortunately, occupational eye injuries are common and carry the potential for severe visual impairment and subsequent visual disability. The first responder's evaluation on whether the problem is a red flag or non-red-flag condition and the action taken can make the difference between a subsequently healed normal eye and blindness. Some cannot wait for referral to an ophthalmologist or optometrist and require immediate action.

Whenever this chapter suggests referral to an ophthalmologist, the primary care physician may refer the patient to an optometrist for care, if in the clinical judgment of the primary care physician, the injury can be treated by an optometrist. A primary care physician may refer the patient to an optometrist to determine whether appropriate care can be rendered by an optometrist.

This chapter provides comprehensive guidelines and practical recommendations for treating the following three major eye complaints seen most frequently in workers:

- Red eye
- Blurred vision (central or peripheral)
- Visual fatigue

Patients with work-related eye complaints are seen commonly by occupational and primary care providers. Eye disorders account for approximately 4% of workers' compensation claims and 1% of total payments. An estimated 2.5 million people suffer eye injuries each year. Between 40,000 and 60,000 of these injuries are associated with severe vision loss, making careful monitoring, proper documentation, and timely referral paramount. In addition to trauma cases, millions of patients visit emergency rooms each year for non-traumatic acute eye conditions such as conjunctivitis. Recommendations for assessing and treating adults with potentially work-related acute eye complaints (i.e., those of 48 hours duration or less) are presented in this clinical practice guideline. Topics include the initial assessment and diagnosis of patients with potentially work-related eye complaints, identification of red flags that may indicate the presence of a serious underlying medical condition, initial management, diagnostic considerations, and special studies for identifying clinical pathology, work-relatedness, return to work in a full- or modified duty capacity, and further management considerations, including the management of delayed recovery.

D. Initial Diagnostic Procedures

D.1 Introduction

The principal recommendations for assessing and treating patients with eye complaints are as follows:

- Initial assessment should focus on detecting indications of potentially serious ocular pathology, termed red flags, and determining an accurate diagnosis. For these purposes, red flags are defined as a sign or symptom of a potentially serious condition indicating that further consultation, support, or specialized treatment may be necessary.
- In the absence of red flags, occupational or primary care physicians can safely and effectively handle work-related eye disorders. Conservative treatment can proceed for 48 to 72 hours for superficial foreign bodies, corneal abrasions, conjunctivitis, and ultraviolet radiation damage. Normally, eye tissues heal rapidly. If eye damage is not well on the way to resolution within 48 to 72 hours, referral to a specialist is indicated. Nonspecific eye complaints may be monitored for a longer period of time while ergonomic and other adjustments are made. The focus is on monitoring for complications, facilitating the healing process, and determining fitness for return to work in a modified- or full-duty capacity.
- Corneal discomfort can be relieved safely with a topically applied ophthalmic nonsteroidal anti-inflammatory drug (NSAID), a systemic nonprescription analgesic, or an intramuscular or intravenous narcotic in severe ocular/face injuries when symptoms or physical findings mandate. Patients requiring narcotic analgesics generally should be referred for ophthalmologic or optometric care. Avoid using topical anesthetics for purposes other than diagnosis or treatment because they may obscure worsening pathology and thus inadvertently cause further injury.
- Visual acuity should be assessed and documented carefully at each examination prior to other examinations or treatment, except for cases of chemical burns.
- Patients recovering from acute eye injury or infection should be encouraged to return to modified work as their condition permits.
- Nonphysical factors, such as psychosocial, workplace, or socioeconomic problems, should be addressed in an effort to resolve delayed recovery.

D.2 History

A. PRESENTING SYMPTOMS The patient may present with symptoms of red eye, blurred vision (central or peripheral), or visual fatigue.

I. Red eye refers to hyperemia of the superficially visible vessels of the conjunctiva, episclera, or sclera. Hyperemia, or engorgement of the conjunctival blood vessels, also known as

inflammation, can be caused by disorders of these structures or of adjoining structures, including the cornea, iris, ciliary body, or ocular adnexa. Red eye can be characterized in three categories:

1. Infections
2. Sterile inflammation
3. Trauma to the eyeball and/or periorbita

II. Blurred vision is a symptom of decreased visual acuity (central and peripheral). The central visual acuity is measured with an Early Treatment Diabetic Retinopathy Study (ETDRS) or Snellen chart at 20 feet (6 meters), at the working intermediate (i.e., computer operators 20 to 30 inches), and near (16 inches) distance. Peripheral vision (visual acuity) is measured by visual fields.

III. Visual fatigue describes a phenomenon related to intensive use of the eyes. It includes complaints of eye or periocular pain, itching, burning, tearing, oculomotor changes, focusing problems, performance degradation, and/or after-colors.

D.3 Physical Examination

Most patients with eye problems improve quickly once any red flag issues are ruled out. The clinical history and physical findings generally are adequate to diagnose the problem and provide treatment. If the patient's limitations due to eye symptoms, other than nonspecific complaints, do not improve in 3 to 5 days, reassessment is recommended. After again reviewing the patient's limitations, history, and physical findings, the clinician may consider referral for further diagnostic studies and discuss these options with the patient. For patients with limitations after 3 to 5 days and unexplained physical findings, such as localized pain or visual disturbance, referral may be indicated to clarify the diagnosis and assist recovery.

Asking the patient open-ended questions such as those listed below allows the clinician to judge the need for further discussion or specific inquiries to obtain more detailed information.

- What are your symptoms?
 - Are you experiencing pain, sensitivity to light, blurring or loss of vision, or headache?
 - Is your problem located primarily in the eye or near the eye? Do you have pain or other symptoms elsewhere?
 - Are your symptoms constant or intermittent? What makes the problem worse or better?
- How do these symptoms limit you?
 - How long can you look at something?

- Can you see clearly?
- When did your current limitations begin?
 - How long has your vision been limited? More than a day or two?
 - Have your symptoms changed? How?
- Have you had similar episodes previously?
- Have you had any previous testing or treatment? With whom?
- What do you think caused the problem?
- What are your specific job duties? How long do you spend performing each duty?
- Do you have other medical problems? Diabetes? High blood pressure? Glaucoma?
- What do you hope we can accomplish during this visit?

A. OCULAR EXAMINATION FOR EYE INJURY

The examination of the injured eye should include:

- Visual acuity (each eye separately) with best correction or pinhole
- Inspection of the ocular structure. (If an open globe is suspected, no pressure should be exerted on the globe.)
- Position of the eyes and eye movements (six cardinal positions) if globe is intact
- Examination of the pupils for size and reaction to light
- Gross visual fields by confrontation
- Ophthalmoscopy
- Intraocular pressure (IOP) determination if acute glaucoma is suspected and the globe is intact

It is important for primary care physicians to make immediate referrals to the closest ophthalmologist, optometrist, or eye institute when eye injuries exceed their capability. Make the patient comfortable (with intravenous analgesics, if necessary), and protect the injured eye from further injury by applying a rigid Fox shield or equivalent. Depending on the type of injury, transport the patient on a stretcher.

Yardsticks that can be used to evaluate standard emergency care include:

1. The detail and accuracy of the history obtained at the time of or after admission
2. The thoroughness of the admission examination
3. The correlation of critical results with medication and/or other treatment provided to the patient

B. SPECIFIC EYE INJURY DIAGNOSIS

If the patient does not have red flags for serious conditions, the clinician can then determine which other eye disorder is present. The criteria presented follow the clinical thought process from the mechanism of illness or injury to unique symptoms and signs of a particular disorder and finally to test results, if any tests were needed to guide treatment at this stage.

The clinician must be aware that several symptoms and signs are common to a number of eye injuries or disorders. Therefore, accurate diagnosis depends on linking the mechanism of injury or pathogenesis, symptoms, signs, and findings of the eye examination with findings on magnification and, if necessary, with fluorescein staining of the eye. In the following lists, an asterisk (*) after a symptom or sign indicates a danger signal.

Symptoms of Red Eye							
Symptom	Referral Advisable if Present	Acute Glaucoma	Acute Iridocyclitis	Keratitis	Bacterial Conjunctivitis	Viral Conjunctivitis	Allergic Conjunctivitis
Blurred vision	Yes	3	1-2	3	0	0	0
Pain	Yes	2-3	2	2	0	0	0
Photophobia	Yes	1	3	3	0	0	0
Colored halos	Yes	2	0	0	0	0	0
Exudation	No	0	0	0-3	3	2	1
Itching	No	0	0	0	0	0	2-3

Note: The range of severity of the symptom is indicated by 0 (absent) to 3 (severe).

Source: Table modified from Bradford CA, ed. Basic Ophthalmology. 7th ed. San Francisco, Calif: American Academy of Ophthalmology; 1999.

Blurred Vision. Blurred vision often indicates serious ocular disease. Blurred vision that

improves with blinking suggests a discharge or mucus on the ocular surface.

Severe pain. * Pain may indicate keratitis, ulcer, iridocyclitis, or acute glaucoma. Patients with conjunctivitis may complain of a scratchiness or mild irritation, but do not have severe pain.

Photophobia. * Photophobia is an abnormal sensitivity to light that accompanies iritis. It may occur either alone or secondary to corneal inflammation. Patients with conjunctivitis have normal light sensitivity.

Colored halos. * Rainbow-like fringes or colored halos seen around a point of light are usually a symptom of corneal edema, often resulting from an abrupt rise in intraocular pressure. Therefore, colored halos are a danger symptom suggesting acute glaucoma as the cause of a red eye.

Exudation. Exudation, also called mattering, is a typical result of conjunctival or eyelid inflammation and does not occur with iridocyclitis or glaucoma. Patients often complain that their lids are “stuck together” on awakening. Corneal ulcer is a serious condition that may or may not be accompanied by exudate. Mucoïd discharge generally is related to allergic conditions. Watery discharge may occur with viral conditions, and a purulent discharge is related to bacterial conditions.

Itching. Although a nonspecific symptom, itching usually indicates an allergic conjunctivitis.

Signs of Red Eye							
Symptom	Referral Advisable if Present	Acute Glaucoma	Acute Iridocyclitis	Keratitis	Bacterial Conjunctivitis	Viral Conjunctivitis	Allergic Conjunctivitis
Ciliary Flush	Yes	1	2	3	0	0	0
Conjunctival Hyperemia	No	2	2	2	3	2	1
Corneal Opacification	Yes	3	0	1-3	0	0-1	0
Corneal Epithelial Disruption	Yes	0	0	1-3	0	0-1	0
Pupillary Abnormalities	Yes	Mid-dilated, non-reactive	Small; may be irregular	Normal or small	0	0	0

Shallow Anterior Chamber Depth	Yes	3	0	0	0	0	0
Elevated Intra-Ocular Pressure	Yes	3	-2 to +1	0	0	0	0
Proptosis	Yes	0	0	0	0		0
Discharge	No	0	0	Some times	2-3	2	1
Preauricular Lymph node Enlargement	No	0	0	0	0	1	0

Note: The range of severity of the symptom is indicated by 0 (absent) to 3 (severe).

Source: Table modified from Bradford CA, ed. Basic Ophthalmology. 7th ed. San Francisco, Calif: American Academy of Ophthalmology; 1999.

Reduced visual acuity.* Reduced visual acuity suggests a serious ocular disease, such as an inflamed cornea, iridocyclitis, glaucoma, or vitreous hemorrhage. It never occurs in simple conjunctivitis unless the associated cornea is involved.

Ciliary flush.* Ciliary flush is an injection of the deep conjunctival and episcleral vessels surrounding the cornea. It is seen most easily in daylight and appears as a faint violaceous ring in which individual vessels cannot be seen by the unaided eye. These engorged vessels, whose origin is the ciliary body, are a manifestation of inflammation of the ciliary body and the anterior segment of the eyeball. Ciliary flush is a danger sign often seen in eyes with corneal inflammations, iridocyclitis, or acute glaucoma. Usually ciliary flush is not present in conjunctivitis.

Conjunctival hyperemia. Conjunctival hyperemia is an engorgement of the larger and more superficial bulbar conjunctival vessels. A nonspecific sign, it may be seen in almost any of the conditions causing a red eye.

Corneal opacification.* In a patient with a red eye, corneal opacities always denote disease. These opacities may be detected by direct illumination with a penlight, or they may be seen with a direct ophthalmoscope (with a plus lens in the viewing aperture) outlined against the red fundus reflex. Several types of corneal opacities may occur, including:

- Keratic precipitates, or cellular deposits on the corneal endothelium, usually too small to be visible. Occasionally forming large clumps, these precipitates can result from iritis or chronic iridocyclitis.
- A diffuse haze obscuring the pupil and iris markings. This may be characteristic of corneal edema. It is frequently seen in acute glaucoma.
- Localized opacities. These may be due to keratitis or ulcer.

Corneal epithelial disruption.* Disruption of the corneal epithelium, which occurs in corneal inflammations and trauma, can be detected in two ways. The first method uses fluorescein vital stain, which detects disruption of the epithelium.

- The examiner should be positioned in such a way as to observe the reflection from the cornea of a single light source (e.g., window or penlight) as the patient moves his or her eye into various positions. Epithelial disruptions cause distortion and irregularity of the light reflected by the cornea.
- Apply fluorescein to the eye. Areas denuded of all layers of the epithelium will stain a bright green with a blue filter. The second method uses rose bengal vital stain, which detects degeneration or absence of one or more layers of the epithelium.
- Examiner positioned in the same manner as described above.
- Apply rose bengal vital stain. Diseased epithelium will stain a reddish purple color.

Pupillary abnormalities.* The pupil in an eye with iridocyclitis typically is somewhat smaller than that of the other eye due to reflex spasm of the iris sphincter muscle. The pupil is also distorted occasionally by posterior synechiae, which are inflammatory adhesions between the lens and the iris. In acute glaucoma, the pupil is usually fixed, mid-dilated (about 5 to 6 mm), and slightly irregular. Conjunctivitis does not affect the pupil.

Shallow anterior chamber depth.* In a red eye, a shallow anterior chamber (especially related to acute ocular pain, nausea, and sometimes vomiting) always suggests the possibility of acute angle-closure glaucoma. Anterior chamber depth can be grossly estimated through side illumination with a penlight. The most exact technique and practice standard involves using a slit lamp with or without a diagnostic anterior segment contact lens. Intraocular pressure (IOP) is then measured.

Elevated IOP.* IOP is unaffected by common causes of red eye other than iridocyclitis and glaucoma. In any red eye without obvious infection, IOP can be measured to rule out glaucoma as clinically indicated (routinely at the time of all eye screening examinations generally after age 40); however, under some circumstances, routine screening for IOP should be part of the examination.

Proptosis.* Proptosis is a forward displacement of the globe. Proptosis of sudden onset suggests serious trauma, orbital infection, or tumor. The most common cause of chronic proptosis is

thyroid disease. Orbital mass lesions also result in proptosis and should be considered. Proptosis may be accompanied by conjunctival hyperemia or limitation of eye movement. Small amounts of proptosis are detected most easily by standing behind a seated patient and looking downward to compare the positions of the two corneas. Acute orbital proptosis secondary to trauma is an ophthalmologic emergency because it may cause severe pressure on the eyeball, which can lead to central retinal artery occlusion.

Preauricular nodes. The type of discharge may be an important clue to the cause of conjunctivitis. Preauricular node enlargement can be a prominent feature of common viral as well as some unusual varieties of chronic granulomatous conjunctivitis, known collectively as Parinaud's oculoglandular syndrome. Usually, such enlargement does not occur in acute bacterial conjunctivitis. The adenovirus is found most commonly, especially in epidemic keratoconjunctivitis, which generally is spread by direct contact with secretions and often results from failure to wash hands after direct contact with infected patients.

D.4 Red-Flag Conditions and Preferred Specific Treatment

Red Flags for Potentially Serious Eye Conditions Requiring Immediate Ophthalmologic/Optometric Examination		
Disorder	Medical History	Physical Examination
Ocular injury, open globe	Trauma due to high-velocity foreign-body injury Visual loss Bleeding Local pain	Visible foreign body in globe; deformity of globe Loss of globe pressure Distorted pupil and/or iris Subconjunctival hemorrhage
Ocular injury, closed globe	Direct blow Visual loss Diplopia	Eyelid ecchymosis Subconjunctival hemorrhage Vitreous hemorrhage Lens dislocation Retinal edema and/or tear Decreased visual acuity Hyphema Retrobulbar hemorrhage Extraocular motion deviation
Thermal burns	Exposure of eyes to hot material/extreme heat Superficial eye pain Photophobia	Burns of lids and/or surrounding structures Damage to cornea, conjunctiva, and/or sclera Decreased visual acuity
Radiation injury	Exposure of eyes to ultraviolet, laser, or bright light Delayed severe superficial eye pain (4-6 hours) Tearing	Blepharospasm Tearing Corneal punctate staining and/or sloughing of epithelium

	Photophobia	Retinal damage
Chemical burns	Alkali, acid, solvent splash Painless visual loss	Corneal erosion Conjunctival chemosis Necrosis of anterior segment of tissues and vessels Decreased visual acuity Circumcorneal vascular ischemia Necrosis of cornea and/or conjunctiva Glaucoma
Hydrofluoric (HF) acid burns	HF acid splash Delayed damage	Necrosis of cornea and/or conjunctiva Decreased visual acuity
Corneal ulcer	Abrasion or infection Superficial pain Foreign-body sensation Photophobia Visual loss	Corneal infiltrates and ulcers Decreased visual acuity Ulceration on slit-lamp exam and fluorescein staining

A. Assessing Red Flags and Indications for Immediate Referral

Physical examination evidence of severe ocular compromise that correlates with the medical history and test results may indicate a need for immediate consultation. The examination may further reinforce or reduce suspicions of infection or major trauma (e.g., open globe, chemical exposure, or radiation damage). A medical history suggestive of pathology in an area other than the eye may warrant examination of the head, neck, or other areas.

TIMING OF REFERRALS OR SPECIAL STUDIES

Referrals for work-related eye complaints generally fall into two categories—immediate and following conservative treatment. Immediate referral to an ophthalmologist or optometrist is necessary for many cases of eyelid disorders or injuries, open globe wounds or penetrating foreign bodies in the eye, thermal and chemical injuries (e.g., alkali, acid, solvent, or hydrofluoric acid burns), central retinal artery and/or vein occlusions, acute glaucoma, corneal ulcers, and retrobulbar hemorrhages.

Once these red flags have been ruled out, conservative treatment by the primary care physician can proceed for 48 to 72 hours for superficial foreign bodies, corneal abrasions, non-alkali chemical splashes, conjunctivitis, and nonionizing radiation damage.

Normally, tissues of the eye heal rapidly. If eye damage is not well on the way to resolution within 48 to 72 hours, referral to an ophthalmologist or optometrist is indicated. Nonspecific eye complaints may be monitored for a longer period of time while making ergonomic and other adjustments.

As indicated below, some special studies, such as radiography and ultrasonography of the globe or orbit, may be indicated in the acute period to rule out fracture of the orbit or the presence of a foreign body, either intraocularly or in the orbit. Diagnose and begin treatment for conditions

that need referral while stabilizing the patient and preparing him or her for transfer. A series of general and diagnostic modalities for the red flag and non-red-flag conditions are provided in Tables 6 and 7.

Management or Referral

The conditions that a primary care physician may appropriately treat include blepharitis, stye and chalazion, and conjunctivitis. Patients requiring prolonged treatment or those in whom the expected response to treatment does not occur promptly may be referred to an ophthalmologist or optometrist.

B. Blunt Trauma

Ocular contusions are caused by blunt trauma to the eye or periorbital structures that may cause contusion of the globe and/or periorbita. There may be no symptoms; however, some patients complain of local pain, visual loss, diplopia, or a red eye. The clinician may observe any of the following—ecchymosis of the eyelid; corneal edema; subconjunctival hemorrhage; microscopic or gross hyphema; reduced visual acuity or abnormal visual fields; dislocation or subluxation of the lens; retinal tears, edema, or detachment; or restriction of ocular motion if extraocular muscles are trapped in a blowout fracture.

C. Retrobulbar Hemorrhage

A retrobulbar hemorrhage may increase the pressure on the globe such that the IOP may become greater than the perfusion pressure of the eye, leading to total ischemia of the retina. A relaxing incision at the lateral canthus must be completed within 10 minutes of the rise in IOP or the eye may be irreversibly damaged secondary to the high IOP.

D. Orbital Floor Fractures

Much discussion and controversy surround managing blowout fractures of the orbit. At various times, recommendations have included operating on all orbital floor fractures and operating on none of them. As the understanding of blowout fractures and their sequelae has evolved over time, so too has an understanding of when surgery is appropriate, and who benefits from such surgery. In the past, the focus often was on early versus late repair. At present, the focus is on understanding the mechanisms of diplopia and enophthalmos in orbital floor fractures, the best way to evaluate a patient, and the best way to restore maximal function and appearance (Harstein and Roper-Hall, 2000).

Diplopia caused by orbital floor blowout fractures is one of the major complications of orbital injuries. When vertical movement of the eye is impaired, surgery is indicated and is performed after complete resolution of orbital hemorrhage and edema. The maximal time before the first surgical procedure is 14 days (Taher, 1993). Treatment indications for orbital floor fractures are evolving. Nonresolving oculocardiac reflex, the “white-eyed” blowout fracture, and early enophthalmos or hypoglobus are indications for immediate surgical repair. Surgery within 2 weeks is recommended in cases of symptomatic diplopia with positive forced ductions and evidence of orbital soft tissue entrapment on computed tomographic (CT) scan or large orbital floor fractures that may cause latent enophthalmos or hypo-ophthalmos (Burnstine, 2002).

E. Hyphema

Complications of traumatic hyphema include increased IOP, peripheral anterior synechiae, optic atrophy, corneal blood staining, secondary hemorrhage, and accommodative impairment. The reported incidence of secondary anterior chamber hemorrhage, i.e., rebleeding, in the setting of traumatic hyphema ranges from 0 to 38%. The risk of secondary hemorrhage may be higher among African Americans than among whites. Secondary hemorrhage is generally thought to convey a worse visual prognosis, although the outcome may depend more directly on the size of the hyphema and the severity of associated ocular injuries. Some issues involved in managing a patient with hyphema are using various medications (e.g., cycloplegics, systemic or topical steroids, antifibrinolytic agents analgesics, and antiglaucoma medications), the patient's activity level, use of a patch and shield, outpatient versus inpatient management, and medical versus surgical management. Special considerations are widely accepted in managing children, patients with hemoglobin S, and patients with hemophilia. It is important to identify and treat ocular injuries that often accompany traumatic hyphema. Consider each of these management issues and refer to the pertinent literature in formulating the following recommendations:

- Advise routine use of topical cycloplegics and corticosteroids, consider systemic antifibrinolytic agents or corticosteroids, and always use a rigid shield.
- Recommend activity restriction (quiet ambulation). If compliance (with medication use or activity restrictions), follow-up, or increased risk for complications (e.g., history of sickle cell disease or hemophilia) is a concern, inpatient management can be offered.
- Indications for surgical intervention include the presence of corneal blood staining or dangerously increased IOP despite maximum tolerated medical therapy, among others.

A study was performed to evaluate the clinical course of patients treated for traumatic microhyphema and the occurrence of elevated IOP and secondary hemorrhage. A total of 162 patients met the study criteria. All patients were treated initially as outpatients according to the protocol for traumatic microhyphema (i.e., atropinization, bed rest, shield, and restriction of antiplatelet medications). Three patients were subsequently hospitalized. The occurrence of IOP elevation (>21 mmHg) and rebleeding was recorded. Of 150 patients with normal IOP at presentation, only 1 (0.7%) developed an elevated IOP at any point to warrant treatment (28 mmHg). Rebleeding was documented in three patients, one of whom developed a layered hyphema. Few complications result from traumatic microhyphema treated with standard measures. Closeness of follow-up may be determined by IOP on presentation. Secondary hemorrhage seems to be unaffected by the use of topical corticosteroids (Recchia, et al., 2002).

F. Burns

I. THERMAL BURNS OF THE EYE

Thermal burns of the eye are caused by exposure to hot gases, liquids, or solids. Unless there is local contact only with the eye, the periocular structures are typically damaged as well. Damage may range from superficial burns of the lids and surrounding structures to superficial destruction of the cornea, conjunctiva, or sclera, to greater destruction including exposure of the globe. If damage exceeds superficial burns of the lids and surrounding structures, prompt intervention by a specialist is imperative.

II. ELECTROMAGNETIC RADIATION INJURY TO THE EYE

Patients with electromagnetic radiation injury to the eye may have no initial symptoms. Severe cases may show a marked decrease in central visual acuity, but there may be severe delayed consequences. Depending on the exact electromagnetic spectrum, the symptoms or signs may be localized to the external segment, lens, retina, and choroid. This type of injury can cause scarring of the cornea or retina or cataracts. Visual field disorders also may result from damage to the retina or choroid. Burns from the blue end of the visible spectrum and ultraviolet A are discussed under nonionizing radiation exposure.

III. CHEMICAL BURNS

When they make contact with ultrasensitive eye tissues, toxic substances immediately begin to cause damage. Studies show that after the first 10 seconds of chemical contact, chances of full recovery become fleeting. Aside from general tissue damage, acids and alkalis can change the pH in the eye itself. From this detrimental change, severe eye damage, including blindness, may result. A history of chemical exposure is an emergency, and examination should be delayed until after the eye is flushed to dilute the chemical. It is imperative that emergency flushing begin immediately. To ensure the best chances for a minimal amount of eye damage, correct emergency equipment, proper placement, and knowledge of its use are necessary in the workplace. The requirements governing medical services and first aid is covered in OSHA 1910.151(a)(b), whereas ANSI Z-358.1, Emergency Eyewash and Shower Equipment, provides guidance. At the site, water is the initial dilution agent to flush the eye or body. Subsequently, an isotonic saline or balanced Ringer's solution is preferred and should be used, if available (otherwise, use sterile intravenous fluids), until a tear pH of about 7 is obtained after ceasing irrigation for 10 minutes. Proper flushing usually takes at least 15 minutes, but can take as long as 24 hours.

Irrigation technique. ANSI Z-358.1, Emergency Eyewash and Shower Equipment, sets forth the requirements for having the facilities to dilute a chemical within 10 seconds of undergoing an industrial eye chemical hazard. Once at the site of an industrial injury, emergency medical personnel or first responders should resolve pain and blepharospasm by applying a topical ophthalmic anesthetic (proparacaine hydrochloride). The interpalpebral fissure should be widened by means of a lid retractor (e.g., Demarres), the eye should be irrigated directly with isotonic saline, Ringer's lactate or other ocular solutions and a contact lens should be removed to facilitate irrigation of the eyeball. The irrigation is not completed until the upper lid is double everted so that all cul-de-sacs (recesses) of the conjunctiva are thoroughly irrigated and visualized. Irrigation should continue until the conjunctival secretions show a consistent pH of approximately 7 after ceasing irrigation for 10 minutes. In the event of a chemical exposure, begin eye irrigation immediately, and remove contact lenses as soon as practical. Do not delay irrigation while waiting for contact lens removal because the lens may come out with the irrigation or can be removed when irrigation is complete. Contact lenses adhere to the cornea and sometimes the paralimbal conjunctiva, depending on the type, and they have been shown to protect the cornea and/or conjunctiva beneath the lens. However, they do not fulfill the requirements of PPE. If a contact lens has not been washed out during the irrigation, emergency medical personnel may remove it following completion of irrigation.

Alkali burns. Alkali burns of the eye typically cause pain initially and may have disastrous

consequences if not treated immediately. Alkali exposure can cause corneal ulceration or conjunctival, scleral, and/or anterior segment degeneration that is manifested as a blanched or “marbleized” appearance. The cornea may become opacified. The diagnosis is usually based on a history of exposure to alkaline chemicals, but occasionally testing the pH of tears or residual liquid is required. Immediate referral to an ophthalmologist or optometrist is recommended. Irrigation in most cases should be continued until the patient is seen by the ophthalmologist or optometrist. A casual examination of the eye may reveal that the globe is white because there is severe ischemia of the conjunctiva or episcleral vessels, a finding that would be noted during a slit-lamp examination.

Acid burns. Acid burns of the eye, caused by acid splashes or vapors, can have the immediate effects of corneal erosion, corneal necrosis, and decreased visual acuity unless irrigation is accomplished immediately. In patients with acid burns, the eye looks inflamed immediately, unlike alkali burns, where the eye is white due to necrosis of the superficial ocular vessels. Delayed effects are unusual in patients with acid burns; hydrofluoric (HF) acid burns are the exception.

Hydrofluoric acid burns. Hydrofluoric acid causes delayed tissue destruction out of proportion to the apparent exposure. With an HF acid concentration of less than 20%, the onset of symptoms may be delayed up to 24 hours. With high concentrations, symptoms may begin relatively quickly. The patient’s main complaint is severe eye pain out of proportion to the apparent exposure. HF acid penetrates tissue remarkably well and causes deep as well as superficial necrosis. HF acid exposure must be treated immediately with copious irrigation with water or isotonic saline solution for 5 minutes and then by calcium gluconate 1% solution or Ringer’s lactate solution providing Ca^{2+} and Mg^{+} atoms to the cell replacing the Ca^{2+} and Mg^{+} atoms that were incorporated into insoluble calcium and magnesium fluoride molecules. Immediate referral to an ophthalmologist or optometrist after emergency care is recommended while calcium gluconate is irrigated into the eye.

G. Corneal Ulceration

Corneal ulcers, which can permanently damage vision, are an ophthalmologic emergency. They may be bacterial, viral, fungal, or parasitic in origin and may occur following corneal lacerations, abrasions, and intrusion of foreign bodies. They may result from poorly fitted or inadequately cleaned contact lenses. Patients with corneal ulcers present with complaints of changes in visual acuity, photophobia and/or eye pain, tearing, and a sensation that a foreign body is in the eye. The presence of corneal ulcers can be determined by direct visualization, but magnified viewing with fluorescein staining is needed to completely rule out their presence.

H. Open Globe Eye Injury

Direct trauma to the eye from high-velocity objects can cause laceration or perforation of the globe. The trauma can be perforating or penetrating. Patients with damage to the integrity of the globe can present with decreased visual acuity, local pain, and bleeding. The cardinal sign is distortion of the globe with loss of tension or IOP; the pupil is not round, but rather is distorted and/or nonreactive. In addition, ecchymosis or other signs of damage to periorbital structures are evident. The clinician may observe subconjunctival hemorrhage, distortion of the iris or pupil, or

herniation of the iris through the cornea. There also may be retinal damage. The injured eye should be protected with a metallic or plastic shield. Transfer by stretcher is recommended.

Nonionizing Radiation Burns	
Technique	Ultraviolet Burns (welders, mercury vapor lamps, sun) Clinical Findings
Healing time	Onset symptoms of photophobia, pain, lacrimation, blepharospasm, 5-12 hours after exposure; healing < 24 hours
Fluorescein staining	Yes
<i>Complications:</i> Recurrent erosion Keratitis/corneal ulcer	No No, unless the patient is using topical anesthetics routinely
Patching	No generally; some patients may be more comfortable with patching
NSAID ophthalmic solution/topical	Yes
Ophthalmic antibiotics/topical ointments: Erythromycin, polymyxin B Gentamycin, tobramycin (gram-positive and gram-negative bacteria, especially <i>P. aeruginosa</i>)	No No
<i>Cycloplegics:</i> Short-acting Mydracyl 1%, Cyclogyl 1% solutions Longer-acting scopolamine .25%, homatropine 5% (large abrasions/iritis)	Yes, if ciliary spasm is present No
Topical steroids	No
Tetanus booster	No
Referral—generally	None, unless healing is not complete in 24 hours

I. Foreign Bodies of the Cornea or Conjunctiva

These may be superficial and may be removed less than 6 hours after the injury. Superficial foreign bodies generally may be removed with a moist swab soon after injury and should be handled like a simple abrasion. Foreign bodies can be divided into two types presents details for treating various types of foreign bodies):

- Simple. A superficial foreign body removed within hours generally will heal within 24 hours and have a low rate of inflammation of the cornea or conjunctiva and no iritis.
- Complex. A foreign body of the cornea and conjunctiva in which the trauma has taken place generally will heal in 24 to 48 hours. A metallic foreign body may be surrounded by swollen necrotic tissue and metallic pigmentation.

Types of Foreign Bodies of the Cornea or Conjunctiva		
Technique	Simple (Foreign Body < 6 hours)	Complex (Foreign Body with Metallic Pigmentation, Swelling of Tissues)
Healing time	Within 24 hours	24-72 hours or longer depending on amount of metallic residue and length of time foreign body has been in contact with the tissues
Fluorescein staining	Yes	Yes
<i>Complications:</i> Recurrent erosion Keratitis/corneal ulcer	No No	No Higher incidence of keratitis/corneal ulcer
Patching	No	No
NSAID ophthalmic solution/topical	No, usually only mild superficial corneal pain	Yes, based on corneal pain present
<i>Ophthalmic antibiotics/topical ointments:</i> Fluoroquinolones (e.g., ciprofloxacin, ofloxacin, norfloxacin)	No	Yes; routine use prophylactically and therapeutically for keratitis/corneal ulcer and endophthalmitis
<i>Cycloplegics:</i> Short-acting mydriacyl 1%	No, only when incidence of ciliary spasm	Yes, if iritis/iridocyclitis present.
Intermediate cyclogyl 1% solutions	No	The type and concentration depends on the degree of ititis/iridocyclitis and how long you desire cycloplegia.
Longer-acting scopolamine.25%, homatropine 5% (large abrasions/iritis)	No	
Topical steroids	No	No; only if prescribed by an ophthalmologist or optometrist

Tetanus booster	No	Based on type of foreign body; verify immunization state; tetanus toxoid as per prophylaxis protocols
Referral—generally	None	Ophthalmologist or optometrist

J. Chemical Splashes

Chemical splashes from a solvent, acid, or alkali agent generally are red flag conditions, but preliminary treatment by a primary care physician can be provided before referring the patient to an ophthalmologist or optometrist. Primary treatment is irrigation of the eye with water and/or isotonic saline until the pH returns to approximately 7, measured by pH paper, 10 minutes after stopping irrigation.

K. Subconjunctival Hemorrhage

In the absence of blunt trauma, hemorrhage beneath the subconjunctiva (the potential space between the conjunctiva and the sclera) requires no treatment and, unless recurrent, no evaluation. Causes may include a sudden increase in ocular venous pressure, such as occurs with coughing, sneezing, vomiting, or vigorous rubbing of the eye. Many subconjunctival hemorrhages occur during sleep when no prodromal conditions exist. If recurrent, an underlying bleeding disorder should be ruled out.

L. Blepharitis

Response to the treatment of blepharitis, or inflammation of the eyelid, is often frustratingly slow, and relapses are common. The mainstays of treatment are as follows:

- Apply a warm compress over the closed eyelids for 10 to 15 minutes with the cloth rewarmed (by running through hot water) as it cools. The compress helps to increase the fluidity of the meibomian glands and loosen the debris from the bases of the lashes.
- To remove the secretions, follow compresses with lid scrubs, such as baby shampoo diluted with water (one drop of shampoo in cup of water) on a cotton ball or clean cloth, which is preferable to a cotton-tipped applicator. These measures may be performed two to four times daily.
- Apply a topical antibiotic (erythromycin or bacitracin) following lid scrubs twice daily. Tetracycline (orally, daily) or doxycycline (orally, daily) is added for patients with rosacea or chronic blepharitis who are not responding to conservative measures. For pregnant or breastfeeding women and children younger than 12 years of age, substitute erythromycin.
- Give patients with punctate epithelial keratitis (PEK) artificial tears five to six times daily.

M. Chalazion

Chalazion is a chronic granulomatous inflammation of a meibomian gland that may develop spontaneously or may follow a hordeolum, acute meibomitis, or stye. Chalazia are chronic

granulomata of fat bodies and they may require excision. Because most chalazia are sterile, antibiotic therapy is of no value, but hot compresses may be useful for early lesions. Incision with curettage is indicated when lesions do not resolve spontaneously or with other medical therapy. A persistent or recurring lid mass should undergo biopsy because it may be a rare meibomian gland carcinoma or a squamous cell carcinoma of the conjunctiva rather than a benign chalazion.

N. Bacterial Conjunctivitis

Bacterial conjunctivitis is treated with frequent antibiotic eye drops as well as antibiotic ointment applied at bedtime. Cool compresses may give some relief. There is no specific medical treatment for viral conjunctivitis, but patients should be instructed in proper precautions to prevent contagion. Corticosteroids have no place in treating infectious conjunctivitis. Eye drops containing a combination of antibiotics and corticosteroids are not indicated for the treatment of ocular inflammation by the primary care or occupational medicine physician.

O. Stye or Hordeolum

A stye or hordeolum is an acute inflammation of the eyelid that may be characterized as an external swelling (involving the hair follicle or associated glands of Zeis or Moll) or an internal swelling (involving the meibomian glands). An external hordeolum occurs on the surface of the skin at the edge of the lid. An internal hordeolum presents on the conjunctival surface of the lid. Styes, generally localized abscess, are treated initially with hot compresses and topical antibiotics. An internal or external hordeolum (stye) may be a sequela of acute blepharitis (meibomitis) and require incision and drainage of the abscess. Incision with curettage is indicated when lesions do not resolve spontaneously or with medical therapy.

E. Follow-up Diagnostic Imaging and Testing Procedures

E.1 Special Studies and Diagnostic and Treatment Considerations

Special studies are not indicated during the first 2 to 3 days of treatment except for red flag conditions.

Radiography of the globe may be indicated if the patient's history indicates the possibility of injury by a penetrating high-speed radiopaque foreign body. Ultrasonography can be used to locate non- and radiopaque foreign bodies. Computed tomographic (CT) scan of the orbit may be indicated in cases of significant blunt trauma and associated fractures at the time of initial evaluation and treatment. Magnetic resonance imaging (MRI) is never indicated when there may be a possibility of a metallic foreign body.

Ability of Various Techniques to Identify and Define Ocular Pathology		
Technique	Identify Physiologic Insult	Identify Anatomic Defect
History	+++	+
Physical examination, including visual acuity testing and funduscopy	++++	++++
Fluorescein staining	0	++++
Slit-lamp examination	0	++++
Tonometry	+++	0
Imaging studies		
Plain-film radiography	0	+ ^a
Ultrasonography	0	++++ ^b
CT scan	0	++++ ^a
MRI	0	++++ ^c

Note: Specificity and repetitiveness from 0 (absent) to (maximum).

^a For evaluating suspected periorbital and other depressed fractures.

^b For evaluating suspected retinal detachment, chamber dimensions, and intraocular foreign bodies.

^c For evaluating foreign body and intracranial pathology.

F. Specific Eye Injury Diagnosis, Testing and Treatment

F.1 Management of Red Eye

A. MANAGEMENT OF RED EYE

Primary care physicians commonly see patients who complain of a red eye. This condition may result from a simple disorder such as a subconjunctival hemorrhage that will resolve spontaneously. The general physician may treat numerous other causes. Vision threatening disorders that cause a red eye require early recognition and prompt referral to an ophthalmologist or optometrist for optimal management based on the results of the initial examination.

B. HISTORY

Information obtained from a careful history and examination directs the approach to management. The onset of a red eye, duration of the redness, and clinical course should be noted to help to distinguish the causative agents. The patient's complaint may reveal the cause of the red eye. For example, itching may signify allergies. A scratchy or burning sensation suggests lid, conjunctival, or corneal disorders, including foreign bodies, in-turning eyelashes, and dry eyes. Localized lid pain or tenderness is a common presenting complaint of a sty or an acute chalazion of the lid.

Deep, intense, aching pain is not localized, but may reflect corneal laceration, iritis, or acute glaucoma, as well as sinusitis or tension headaches. Photophobia suggests problems arising from the anterior segment of the eye, such as corneal abrasions, iritis, and acute glaucoma. A halo effect around lights is a sign of corneal edema commonly seen in acute glaucoma. Individuals who have corneal edema associated with contact lens wear also may experience halo vision.

C. OBSERVATION

When the patient enters the examination area, the physician can observe his or her ability to see the way in and gauge depth. Photophobia or pain can be inferred if the affected eye is held shut. Tearing or discharge can be observed as well. A history of chemical splash is an emergency and examination may be delayed until the eye is flushed to dilute the chemical.

D. EXAMINATION

The primary care physician should evaluate the red eye with a visual acuity chart, a penlight (slit lamp preferred), a tonometer, a sterile fluorescein dye strip, topical anesthetic drops, and an ophthalmoscope. Most clinics today have a Titmus or Stereo Optical visual screener or an Armed Forces Tester, a noncontact "puff" tonometer (Reichert Optical Company), and a slit lamp. A systematic approach to the examination should then be conducted, beginning by examining the face, orbital area, and lids and ending with a close view of the eyeball. The preferred method for examining the eyeball includes using the slit-lamp biomicroscope and the ophthalmoscope.

Any patient who complains of a red or painful eye should be examined to detect any of the conditions described below:

CONJUNCTIVA/SCLERA

- Conjunctivitis is manifested by hyperemia of the conjunctival blood vessels; the cause may be bacterial, viral, allergic, or irritative; the condition is common and often not serious.
- Episcleritis is an inflammation (often sectorial) of the episclera, the vascular layer between the conjunctiva and the sclera. It is neither common nor serious nor does it produce a discharge. It may be allergic and is occasionally painful.
- Scleritis is an inflammation (localized or diffuse) of the sclera. Although it is potentially serious to the eye, it is uncommon, often protracted, usually accompanied by pain, and may indicate serious systemic disease such as a collagen-vascular disorder.
- Subconjunctival hemorrhage is an accumulation of blood in the potential space between the conjunctiva and the sclera. It is rarely serious except as related to orbital trauma.
- Pterygium is an abnormal growth consisting of a triangular fold of tissue that advances progressively over the cornea, usually from the nasal side. It is usually not serious. Localized conjunctival inflammation may be associated with pterygiae. Most cases occur in tropical climates. Surgical excision is indicated if the pterygium encroaches on the visual axis.

CORNEA

- Herpes simplex keratitis is an inflammation of the cornea caused by the herpes simplex virus. It is common, potentially serious, and can lead to corneal ulceration.
- Abrasions and foreign bodies may be associated with hyperemia ciliary flush (circumcorneal hyperemia).

ANTERIOR CHAMBER

- Acute angle-closure glaucoma is an uncommon form of glaucoma due to sudden and complete occlusion of the anterior chamber angle by its tissue. It is serious. The more common chronic open-angle glaucoma causes no redness of the eye.
- Iritis or iridocyclitis is a serious inflammation of the iris, alone or with the ciliary body; it often is manifested by ciliary flush (circumcorneal hyperemia).

ADNEXA

- Adnexal disease affects the eyelids, lacrimal apparatus, and orbit. It includes dacryocystitis, styes, and blepharitis. Red eye also can occur secondary to lid lesions (such as basal cell carcinoma or squamous cell carcinoma), thyroid disease, and vascular lesions in the orbit.
- Abnormal lid function can result in a red eye. Potentially serious lesions such as Bell's palsy, thyroid ophthalmopathy, and others allow ocular exposure.

The American Academy of Ophthalmology specifies nine diagnostic steps to use when evaluating a patient with a red eye (Bradford):

1. Determine whether the visual acuity is normal or decreased using a Snellen chart or (preferred) ETDRS chart at 20 feet or 6 meters, or the 1 meter ETDRS chart if required.
2. Decide by inspection what pattern of redness is present and whether it is due to subconjunctival hemorrhage, conjunctival hyperemia, ciliary flush, or a combination of these.
3. Detect the presence of conjunctival discharge and categorize it as to amount – profuse or scant – and character – purulent, mucopurulent, serous, or hemorrhagic.
4. Detect opacities of the cornea, including large keratic precipitates, or irregularities of the corneal surface, such as corneal edema, corneal leukoma (a white opacity caused by scar tissue), and irregular corneal reflection. Conduct the examination using a biomicroscope, or penlight and transilluminator, at least. Biomicroscopy is the practice standard.
5. Search for disruption of the full thickness of the corneal epithelium by staining the cornea with fluorescein² and lack of corneal epithelium vitality by staining with rose bengal.
6. Use of a slit lamp (biomicroscope) allows one to estimate the depth of the anterior chamber as normal or shallow and to detect any microscopic blood or white blood cells, which would indicate either hyphema or hypopyon, respectively. (A hypopyon is indicated by the presence of protein and white blood cells in the anterior chamber, e.g., when a corneal ulcer is present, and a hyphema is indicated by protein and red blood cells in the anterior chamber.)
7. Detect irregularity of the pupils and determine whether one pupil is larger than the other. Observe the reactivity of the pupils to light to determine whether one pupil is more sluggish than the other or is nonreactive.
8. Determine whether the intraocular pressure is high, normal, or low by performing tonometry if indicated clinically, e.g., if acute angle closure glaucoma is suspected. (Tonometry is contraindicated when external infection or lack of globe integrity is obvious.)
9. Detect the presence of proptosis, lid malfunction, or any limitations of eye movement.

A comprehensive examination is preferred in patients with ocular diseases or injuries. At a minimum, perform a visual acuity assessment prior to any treatment, except in chemical injuries, where immediate irrigation is mandated. Ocular (visual) screening is extremely useful and can fulfill the minimal examination requirements.

E. METHODS OF TESTING

Visual Acuity: Quantitative Bilateral Tests. Acuity is measured at infinity (as a minimum) and near and intermediate distances (based on job description) and is performed with and without

corrective devices (e.g., glasses or contact lenses) and without removing other corrective devices (e.g., intraocular lenses).

Slit-Lamp Biomicroscopy. Slit-lamp examination is the standard method of examining the eye. The slit lamp uses intense illumination and magnification. Use of the slit-lamp biomicroscope has been established as a competency by the American College of Occupational and Environmental Medicine for occupational health physicians. The general findings noted in a slit-lamp examination (biomicroscope) and their clinicopathologic correlations appear at the end of this chapter under “Additional Resources.”

F. HOW TO INTERPRET THE FINDINGS OF RED EYE

The associated signs and symptoms of various disorders overlap to some extent. Although many conditions can cause a red eye, several signs and symptoms signal danger. The presence of one or more of these danger signals (i.e., a red flag) alerts the physician that the patient has a disorder requiring an ophthalmologist’s or optometrist’s attention.

G. DIAGNOSTIC CRITERIA

Red Eye Differential Diagnosis

Red eye can be categorized generally in four classes—conjunctivitis, iritis, keratitis (corneal inflammation or foreign body), and acute glaucoma. The changes in vision, type of discharge, presence or absence of pain, papillary size, presence of conjunctival injection, pupillary response to light, IOP, appearance of the cornea, and anterior chamber depth assist in making the diagnosis.

Differential Diagnosis – Red Eye

- *Acute angle-closure glaucoma.* An uncommon form of glaucoma due to sudden and complete occlusion of the anterior chamber angle by iris tissue – serious. The more common chronic open-angle glaucoma causes no redness of the eye.
- *Iritis or iridocyclitis.* An inflammation of the iris alone or of the iris and ciliary body; often manifested by ciliary flush – serious.
- *Herpes simplex keratitis.* An inflammation of the cornea caused by the herpes simplex virus; common – potentially serious; can lead to corneal ulceration.
- *Conjunctivitis.* Hyperemia of the conjunctival blood vessels; cause may be bacterial, viral, allergic, or irritative; common – often not serious.
- *Episcleritis.* An inflammation (often sectorial) of the episclera, the vascular layer between the conjunctiva and the sclera; uncommon, without discharge – not serious; possibly allergic, occasionally painful.

F.2 Occupational Eye Infection

Occupational hazards cause very few eye infections either directly or primarily. Rather, most occupational eye infections are attributable to workers who transfer the disease process. The

significant eye infections include epidemic keratoconjunctivitis (EKC), infections caused by bloodborne pathogens, and tropical disease.

A. Laboratory Diagnosis. Most mild cases of conjunctivitis are managed without laboratory assistance. While representing a compromise with ideal management, it is justified by the economic waste of obtaining routine smears and cultures in such a common and benign disease. Most clinicians prescribe broad-spectrum topical ophthalmic antibiotic treatment. Cases of presumed bacterial conjunctivitis that do not improve after 2 days of antibiotic treatment should be referred to an ophthalmologist or optometrist to confirm the diagnosis and to conduct appropriate laboratory studies. In cases of hyperpurulent conjunctivitis, when copious purulent discharge is produced, conjunctival cultures and ophthalmologic consultation are necessary because of a possible gonococcal cause. Gonococcal hyperpurulent conjunctivitis is a serious, potentially blinding disease. In doubtful cases, smears of exudate or conjunctival scrapings can confirm clinical impressions regarding the type of conjunctivitis. Generally, the amount of exudate in an ocular infection is very small, especially in corneal ulcers, and this task should be left to the ophthalmologist with his or her microsurgical techniques. Typical findings include polymorphonuclear cells and bacteria in bacterial conjunctivitis. Cultures for bacteria and determinations of antibiotic sensitivity also are useful in cases that are resistant to therapy.

B. Specific Infections

- ***Epidemic keratoconjunctivitis (EKC).*** This is a classic condition originally described as shipyard conjunctivitis in 1934. In Western countries, EKC occurs mostly in industrial plants, where the disease periodically affects a considerable number of workers. Outbreaks appear from time to time in hospitals (Leopold), families (Dawson et al.), children (Dawson; Darwell, et al.), and ophthalmologic clinics, perhaps due to the use of unsterilized tonometers, eyedroppers, or finger-to-eye transmission (Pillat; Jawetz et al.; Dawson and Darwell; and others). Males are affected more frequently than females.
- ***Bloodborne pathogens.*** Infections also may be acquired by exposure to bloodborne pathogens, as in the acquired immune deficiency syndrome (AIDS), human immunodeficiency virus (HIV) infection, and hepatitis B virus (HBV) infection. Bloodborne pathogen exposure may be acquired by the spread of infectious products from afflicted individuals. Cross contamination may occur through the use of contaminated instruments, hands, etc. Bloodborne pathogen regulations (CFR 1910.1030) apply to occupational exposures from blood and other potentially infectious materials (e.g., semen, vaginal secretions, cerebrospinal fluid, synovial fluid, pleural fluid, pericardial fluid, peritoneal fluid, amniotic fluid, saliva in dental procedures, tears, body fluid visibly contaminated with blood, and all bodily fluids in situations where it is difficult or impossible to differentiate between bodily fluids, as well as any unfixed tissue or organ other than intact skin from a living or dead human). An exposure is defined as a specific eye, mouth, or other mucous membrane, non-intact skin, or parenteral contact with blood or other potentially infectious materials resulting from the performance of an employee's duties. The term includes any pathogenic microorganisms present in human blood that can cause disease in persons who are exposed. These examples include hepatitis C, malaria, and syphilis. The intent of the regulation is to prevent the development of an exposure incident through appropriate administrative controls and

personal protective equipment. An applanation tonometer, for example, must be thoroughly cleansed and sterilized after being used on an individual with HIV in the tears before using it on another individual because it may transmit the virus. Personal protective equipment appropriate to a procedure must be used.

- **Topical disease.** Some infectious diseases of the eye are not unique to the duties of individuals but rather are acquired from tropical conditions found in the area of employment. Examples include onchocerciasis, leishmaniasis, and trachoma.

C. PREVENTION AND CONTROL OF OCCUPATIONAL EYE INFECTIONS.

Occupational eye infections spread by medical personnel through the use of inappropriate procedures are not uncommon. Various controls may be employed to help eliminate these infections:

- **Administrative controls.** These are designed to prevent dissemination of infectious agents to the eye. Members of medical departments are at risk for conditions such as EKC and AIDS. Tropical conditions can be readily referenced by geographic areas of the world and specific administrative controls should be implemented as recommended by the Centers for Disease Control and Prevention (CDC). Hands should always be washed between patient contacts.
- **Safe work practices.** The medical staff should help to prevent transmitting diseases person to person (i.e., EKC and AIDS). On some occasions, bodily secretions or blood products containing the AIDS virus may be cleaned up by nonmedical staff, and similar safe practices must be used.
- **Personal protective equipment for examiners.** In accordance with dictates of the Occupational Safety and Health Administration (OSHA, 29 CFR 1910.132), personal protective equipment must be worn by individuals who may be exposed to hazardous conditions. Using disposable gloves and eye and face protection is mandated in situations where medical personnel may be exposed to or may transmit infectious products from themselves to others. Such equipment includes type C spectacles with a full side shield, type D spectacles with a detachable side shield, type E spectacles with a non-removable lens, and type H cover goggles with indirect ventilations, as dictated by the American National Standards Institute (ANSI Z87.1-2003). In cases involving potential exposure to AIDS, personal protective equipment is mandated and directed in 29 CFR 1910.1030. Exposure from splattering or droplet generation would necessitate using the eye protection noted above and a mask or an ANSI Z87.1 type N face shield.
- **Employee education and training.** OSHA regulations (29 CFR 1910.132) mandate training about potential hazards and their prevention by using appropriate personal protective equipment. No individual who may be exposed to hazards should be allowed to work in such an environment without appropriate education and training.

D. Fundamental Clinical Signs of Ocular Inflammation

Inflammation of the conjunctiva and cornea produces only a few clinical signs. Some of these, such as hyperemia of conjunctival vessels, edema, and conjunctival papillae, are nonspecific and

may not be helpful in determining the etiology of inflammation. Others, such as conjunctival follicles, giant papillae, membranes, phlyctenules, and marginal infiltrates, are more specific and can be helpful in determining etiology.

E. CONJUNCTIVA

Morphologically, the conjunctiva consists of two layers, the epithelium and the underlying stroma (substantia propria). In a few areas, the overlying conjunctival epithelium is attached to an underlying structure, such as the tarsus or bulbar limbus, by fine fibrous strands or anchoring septa.

I. Papillary Response of the Conjunctiva. A conjunctival papillary response is a nonspecific clinical sign that can result from any type of inflammation. Papillae in the palpebral conjunctiva or at the limbus are the equivalent of simple hyperemia elsewhere in the conjunctiva. Only where fine fibrous strands are present and are attached to subjacent tissues can conjunctiva fully develop. A papillary response presents with a fine, mosaic-like pattern of elevated, polygonal, hyperemic areas separated by pale channels. A central fibrovascular core is present within each papilla. This gives rise to a central vessel that, on reaching the surface of the structure, erupts into a spoke-like pattern that is readily evident on biomicroscopic examination. The papillae result from leakage of fluid and acute inflammatory cells (polymorphonuclear leukocytes, etc.) from the vascular core, resulting in swelling of the tissue. The connective tissue septa that anchor the overlying epithelium to the deeper collagenous tissue are responsible for forming the papillae. The connective tissue septa restrict the size of papillae to less than 1 millimeter. Various types of papillae can develop in essentially three conjunctival areas: 1) upper palpebral conjunctiva; 2) lower palpebral conjunctiva; and 3) bulbar limbus. Each area will have a somewhat different clinical appearance of papillae owing to variations in anatomy.

Giant papillae are a unique form. They are greater than 1 millimeter in size and have several different clinical appearances and etiologies. There is a different clinical spectrum of giant papillae from various etiologies, including palpebral vernal conjunctivitis, limbal vernal conjunctivitis, atopic keratoconjunctivitis, giant papillary conjunctivitis (GPC) of contact lenses, and giant papillae from prostheses and ends of nylon sutures. The giant papillae seen with the atopic diseases of palpebral vernal and atopic keratoconjunctivitis are polygonal in shape with a flat surface. These are usually much larger than follicles and vary in size and shape. The giant papillae produced by contact lenses have a wide spectrum of clinical appearance.

The common, mild form of GPC has giant papillae larger than 1 millimeter, but the papillae do not have the polygonal shapes or flat surfaces that are typical of giant papillae of vernal conjunctivitis. In other areas, inflammation causes simple hyperemia rather than papillae. Normally, small blood vessels exist that extend into the conjunctival stroma among the fibrous strands. Conjunctival lymphoid follicles also are present within the stroma and can be commonly seen in the inferior conjunctival sac in young individuals and occasionally in older people.

II. Follicular Response of the Conjunctiva. A follicular response of the conjunctiva is a much more specific clinical sign and narrows the differential diagnosis as to the etiology of the inflammation. The conjunctival follicle is a smooth elevation of the conjunctiva that represents a lymphocytic response with an active germinal center. Vessels may encroach on the surface of the

follicles but are not seen within the follicle. A papillary response is nonspecific and can occur along with any follicular response. These pathologic etiologies produce a more severe follicular response in the inferior conjunctival cul de sac than in the upper tarsal conjunctiva, with the exception of trachoma. Unfortunately, the clinical sign of pathologic follicles can be obscured by an overlying papillary response or an overlying inflammatory membrane or pseudomembrane.

III. Conjunctival Pseudomembrane or Membrane. The conjunctival pseudomembrane or membrane is another condition that has some specificity. A transudation of fluid, rich in protein and fibrin, is extruded through the walls of the altered conjunctival blood vessels, coagulating on the surface of the conjunctiva and producing a pseudomembrane or membrane. The difference in the two is one of severity; because pseudomembranes are less firmly adherent, they do not produce bleeding when stripped from the conjunctival surface.

F. CORNEA

I. Edema. There are two types of corneal epithelial edema:

- *Intracellular epithelial edema*, a swelling within the epithelial cells as a result of local epithelial inflammation or nutritional compromise of the corneal epithelium, can be localized or generalized. Examples of intracellular epithelial edema are Sandler's veil, seen with scleralcorneal contact lenses and rarely with soft contact lenses, and circumscribed epithelial edema seen with polymethylmethacrylate (PMMA) contact lenses. This intracellular epithelial edema is due to hypoxia.
- *Intercellular epithelial edema* manifests as fluid between the epithelial cells, maintained within the epithelial layer by the zonula occludens and macular adherens, the cellular adhesions among the corneal epithelial cells. Intercellular epithelial edema results from fluid that passes from the corneal stroma into the epithelial layer whenever the IOP exceeds the corneal stromal swelling pressure. Intercellular epithelial edema is seen clinically as microcystic epithelial edema or in the more severe form as epithelial bullae.

II. Epithelial Filaments. Epithelial filaments of the cornea may occur in various types of keratitis. Epithelial filaments are coils of epithelial cells attached to the cornea at their base. Mucus and other debris adhere to these epithelial filaments. The corneal epithelial filament is a clinical sign that can occur from a variety of etiologies.

III. Active Corneal Stromal Inflammation. Active corneal stromal inflammation is most readily identified clinically by infiltrates of leukocytes and edema within the corneal stroma. The infiltrates appear as focal opacities on biomicroscopic examination and can lie at any level of the stroma. In an avascular cornea, a stromal infiltrate is usually composed predominantly of polymorphonuclear leukocytes. These cellular elements can originate from the limbal vascular arcades and migrate to the site of corneal injury. Alternatively, they can enter the stroma from the tear film or the aqueous humor when defects occur in the layers of the cornea that act as barriers. In a vascularized cornea, inflammatory cells make their way into stroma by way of the new vascular channels, and the infiltrates are comprised of mixed cellular components.

IV. Stromal Edema. Stromal edema from an inflammatory etiology almost invariably coexists

with inflammatory infiltrates. This is evident clinically by increased thickness of the corneal stroma, which is roughly proportional to its water content. Stromal edema may be localized or generalized.

V. Corneal Scarring. Corneal scarring results wherever the inflammatory process is severe enough to cause tissue destruction. This process interrupts the regular lamellar arrangement of corneal collagen, resulting in loss of transparency. The scarring process involves manufacture of new collagen from active stromal keratocytes. Pigment, particularly melanin, is sometimes included in the structure of the scar. A number of deposits—calcium, lipid, proteinaceous material, or iron are encountered most often—in the corneal stroma can appear in cases of long-standing inflammation.

VI. Neovascularization. Neovascularization is an additional indicator of active corneal inflammation. The new vessels can lie in the superficial or deep cornea depending on the nature of the inflammatory stimulus. It is important to recognize that there is a normal superficial vascular arcade at the corneal limbus. The distance that the vascular arcade extends onto the corneal limbus varies from person to person, but once the vessels leave the normal arcade and extend onto the cornea:

- A superficial micropannus can develop, extending 1 to 2 millimeters beyond the normal vascular arcade or as a gross pannus that extends more than 2 millimeters beyond the normal vascular arcade. It is important to distinguish between micropannus and gross pannus because the differential diagnosis of each varies.
- Deep stromal (interstitial) vascularization, which has a less specific etiology, can be caused by any chronic inflammation associated with stromal edema.

VII. Chronic Inflammation. This can result from several etiologies and can produce various superficial opacities in a horizontal band across the interpalpebral area of the cornea; this is known clinically as band-shaped keratopathy.

VIII. Epithelial Keratitis. Epithelial keratitis is a common clinical sign with several possible etiologies. The term superficial punctate keratitis (SPK) should be reserved for the specific clinical entity described by Braley and Thygeson. Other etiologies of epithelial keratitis are not specific entities but are secondary to a number of other causes. Morphologic and distribution differences may help to differentiate the many causes of epithelial keratitis.

IX. Corneal Endothelium. The corneal endothelium can be involved secondarily by inflammatory processes in the corneal stroma (keratitis) or in the anterior uveal tract (anterior uveitis). In the latter instance, inflammatory cells are present in the anterior chamber and appear on the biomicroscope as white or gray specks circulating in the aqueous humor. Aggregates of these cells can accumulate on the endothelial surface of the cornea, where they are referred to as keratic precipitates (KPs). Several clinical forms of KPs are recognized. In the punctate or granular form, the cells are primarily polymorphonuclear leukocytes and lymphocytes. Larger cellular aggregates are known as “mutton fat” KPs; macrophages predominate in this type of deposit. Finally, the fibrinous KP, in which the endothelial deposits are composed largely of fibrin with few inflammatory cells, also is recognized. KPs usually resolve completely, but

residual hyalinized deposits can remain on the posterior cornea.

X. Retrocorneal Membranes. Retrocorneal membranes (thin, filmy membranes of connective tissue that cover the posterior corneal surface) can develop following inflammation and occur especially following hyphema, penetrating keratoplasty, or corneal perforation due to trauma or infection. These membranes can, at times, be vascularized or pigmented. It is thought that endothelial cell damage or death precedes proliferation of a retrocorneal membrane, and in the vast majority of cases, its occurrence is accompanied by edema of the corneal stroma.

G. ANTERIOR CHAMBER

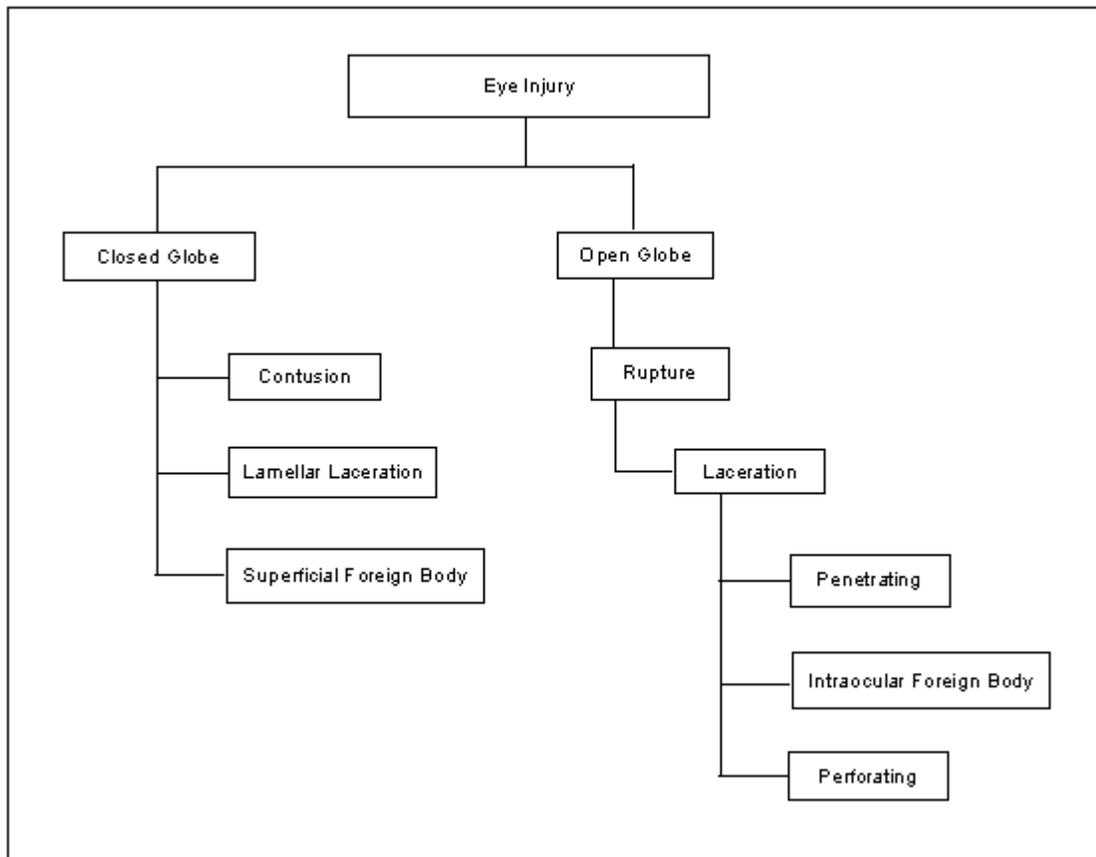
Examining the depth and contents of the anterior chamber is extremely important and correlates well with the symptoms and signs previously obtained.

- *Depth.* In the case of narrow-angle glaucoma, the edema of the cornea is associated with a narrow-angle *iris bombe'* where the anterior surface of the iris almost touches the peripheral cornea and where the anterior chamber is extremely shallow. An ancillary finding in these cases is an increase in IOP averaging 40 to 60 mmHg.
- *Content.* The content of the anterior chamber is significant because it correlates with the other findings of the conjunctiva and cornea.
- In cases of a uveitis (purulent cyclitis), the presence of protein and cells in the anterior chamber is classic.
- When the number of cells develop sufficiently that they precipitate, a hypopyon will be present. A hypopyon is associated with layering of cells in up to 50% of the inferior anterior chamber.
- A hyphema is an accumulation of red blood cells secondary to trauma in the anterior chamber generally in the inferior half, but it can occupy the entire anterior chamber. When there is an associated IOP increase, the blood will be forced into the cornea, causing blood staining of the cornea.

F.3 Ocular Trauma

A new standardized classification of ocular trauma has been proposed and is known as the Birmingham Eye Trauma Terminology (BETT). Definitions in medical dictionaries are tailored to general medical use and cannot be applied effectively to ocular trauma. The new system always uses the entire globe as the tissue of reference; therefore, the type of the injury is described unambiguously without having to indicate the tissue involved. When a tissue is specified, it refers to wound location, not to injury type. A corneal penetrating injury thus involves an open globe injury with the wound being in the cornea. The system provides unambiguous definitions for each term and a complete classification of injury types.⁵ **This new system should be used in all cases of ocular trauma.** When the BETT system was published in 1996, it was reasonably expected that it eventually would become the standardized international language of ocular trauma. Ophthalmologists were urged to use this terminology in clinical practice and research. It is mandated by Graefes' Archives, Klinische Monatsblätter, and Ophthalmology.

The New Standardized Classification of Eye Trauma



Source: Figure modified from Kuhn F, Morris R, Witherspoon CD, Heimann J, Jeffers JB, Treister G. A standardized classification of ocular trauma. *Graefes Arch Clin Ophthalmol.* 1966;234(6):399-403.

A. Elements of the History of Ocular Injury

While a detailed, accurate history is essential in all injuries, it is especially important to obtain a detailed history of an ocular injury because incorrect or misleading information may lead to blindness. Such information may be obtained from a variety of sources, including the patient, the first responder(s), and others involved in or associated with the accident.

Information for acute trauma should include the four Ws:

- *Where:* Location of the accident
- *When:* Time and date
- *Who:* Other individuals involved
- *What:* A detailed description of the accident circumstances, including force and load. If chemical exposure was involved, seek available Material Safety Data Sheets (MSDSs) information. Critical data include:

- What chemical? MSDS information:
 - *Type of chemical* (alkali, acid, solvent)
 - *Type of exposure* (liquids, solids, fumes)
 - *Dose of exposure*
 - *pH* of the material
 - *Concentration* of the material
 - *Solubility* of the material
 - *Contact time*
- Emergency medical care provided by first responder(s):
 - Product manufacturer
 - Availability of chemical data:
 - Material Safety Data Sheets (MSDSs)
 - Regional poison control center
 - Internet

Under federal law, 29 CFR 1910.1200, Hazard Communication, MSDS are to be furnished by the employer to the worker or his or her health care provider on request. Unfortunately, the quality of the information on MSDSs varies greatly because the information is supplied by the manufacturer of the product and there is no effective mechanism in place to ensure that it is accurate.

Table 5. BETT Glossary of Terms (Definitions and Explanations)

Eyewall Sclera and cornea

Although the eyewall technically has three coats posterior to the limbus, for clinical and practical purposes, violation of only the most external structure is taken into consideration.

Closed globe injury No full-thickness wound of eyewall

Open globe injury Full-thickness wound of eyewall

Contusion No (full-thickness) wound

The injury is either due to direct energy delivery by the object (e.g., choroidal rupture) or to the changes in the shape of the globe (e.g., angle recession).

Lamellar laceration Partial-thickness wound to the eyewall

Rupture Full-thickness wound of the eyewall caused by blunt object

Because the eye is filled with incompressible liquid, the impact results in a momentary increase

of the IOP. The eyewall yields at its weakest point (at the impact site or elsewhere; e.g., an old cataract wound dehisces even though the impact occurred elsewhere); the actual wound is produced by an inside-out mechanism.

Laceration Full-thickness wound of the eyewall caused by a sharp object
The wound occurs at the impact site by an outside-in mechanism.

Penetrating injury Entrance wound
Both wounds caused by the same agent.

Note: Some injuries remain difficult to classify. For instance, an intravitreal BB pellet is technically an intraocular foreign body (IOFB) injury. However, because this is a blunt object that requires a huge impact force if it enters, not just contuses, the eye, there is an element of rupture involved. In such situations, an ophthalmologist or optometrist should either describe the injury as “mixed” (i.e., rupture with an IOFB) or select the most serious type of the mechanisms involved.

F.4 Management of Blurred Vision

Blurred vision is a symptom of a decrease in visual acuity that may be central or peripheral. The patient presenting with symptoms or signs of blurred vision may be referred to an ophthalmologist or, based on results of visual (ocular) screening, an optometrist.

A. Central (Modified from Rhee DJ, Pyfer MF, Rhee DM, eds. *The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Diseases*. 3rd ed. Philadelphia, Pa:Lippincott, Williams & Wilkins; 1999. Chapter 1, p. 1-2)

1. A central decrease in visual acuity may be transient or last longer than 24 hours.
 - Transient visual loss (vision returns to normal within 24 hours, usually within 1 hour).
 1. Few seconds (usually bilateral): Papilledema
 2. Few minutes: Amaurosis fugax [transient ischemic attack (TIA), unilateral], vertebrobasilar artery insufficiency (bilateral)
 3. Between 10 and 60 minutes: Migraine (with or without subsequent headache)
2. Visual loss lasting longer than 24 hours.
 1. Sudden painless loss: Retinal artery or vein occlusion, ischemic optic neuropathy, vitreous hemorrhage, retinal detachment, optic neuritis (usually pain with eye movements)
 2. Gradual, painless loss (over weeks, months, or years): Cataract, open-angle glaucoma, chronic retinal disease [e.g., age-related macular degeneration (ARMD), diabetic retinopathy]

3. Painful loss: Acute angle-closure glaucoma, optic neuritis (pain with eye movements), uveitis, corneal hydrops (keratoconus)
3. Gradual change in refractive error (over months, years).
 1. Myopia—nearsightedness
 2. Hyperopia—farsightedness
 3. Presbyopia—lack of accommodation for reading or performing tasks at near (approximately 9 inches or 22 centimeters)

B. Peripheral Modified from Rhee DJ, Pyfer MF, Rhee DM, eds. *The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Diseases*. 3rd ed. Philadelphia, Pa: Lippincott, Williams & Wilkins; 1999. Chapter 2, p. 16)

The peripheral vision (visual acuity) can be measured by means of a visual field examination. Visual field types of defects (identified below) will help to determine the anatomic defects and the most likely diagnosis.

Altitudinal defect. Ischemic optic neuropathy.

Arcuate scotoma. Glaucoma.

Binasal field defect. Glaucoma, bitemporal retinal disease (e.g., retinitis pigmentosa).

Bitemporal hemianopia. Chiasmal lesion (e.g., pituitary adenoma, meningioma, craniopharyngioma, aneurysm, glioma)

Blind spot enlargement. Papilledema, glaucoma, optic nerve drusen, optic nerve coloboma, medullated nerve fibers off the disc, drugs, myopic disc with a crescent, others.

Central scotoma. Macular disease, optic neuritis, ischemic optic neuropathy (more typically produces an altitudinal field defect), optic atrophy (e.g., from tumor compressing the nerve, toxic/metabolic disease).

Homonymous hemianopsia. Optic tract or lateral geniculate body lesion; temporal, parietal, or occipital lobe lesion of the brain (stroke and tumor more common; aneurysm and trauma less common).

Migraine. May cause a transient homonymous hemianopsia.

Constriction of the peripheral fields leaving only a small residual central field. Glaucoma, retinitis pigmentosa, or some other peripheral retinal disorder, chronic papilledema, after panretinal photocoagulation, central retinal artery occlusion with cilioretinal artery sparing, bilateral occipital lobe infarction with macular sparing, nonphysiologic visual loss, carcinoma-associated retinopathy.

F.5 Management of Visual Fatigue

Visual fatigue is a term used to describe phenomena related to intensive use of the eyes. It can include complaints of eye or periocular pain, itching or burning, tearing, oculomotor changes, focal problems, performance degradation, after-colors, and other phenomena. Patients presenting with signs or symptoms of visual fatigue may be referred to an ophthalmologist or optometrist.

The ability to perform most tasks depends on many visual and nonvisual variables, and the factors that influence the visual performance include:

- The patient's visual capability
- The visibility of the task
- Psychological and general physiologic factors

Studies indicate that the visual complaints occur in 50 to 90% of video display terminal (VDT) workers. The vision problems result from visual inefficiencies and eye-related symptoms. They are caused by a combination of individual visual system problems and poor visual ergonomics. The problems occur whenever the task's visual demands exceed the patient's visual abilities.

The visual symptoms can be resolved, for the most part, with good visual ergonomics, by properly managing the environment, and by providing proper visual care. Ergonomics is the science of designing machines and work tasks with the capabilities and limitations of the human being in mind.

Visibility of Tasks

The ability to perform a task safely, efficiently, and comfortably depends on its visibility, as well as on the worker's visual capabilities. Naturally, the better the visibility, the easier it is to perform the task, and the factors influencing a task's visibility include:

- Size
- Distance
- Illumination
- Glare
- Contrast
- Color
- Time available to view task
- Movement of the task
- Atmospheric conditions

Ergonomic research supports the following regarding VDTs:

- Place frequently used displays in the primary visual display area. The top of the display should be opposite the operator's eyes, which face forward, extending down to a point at which the operator is looking down at a 30-degree angle. Devices viewed as they are

operated, such as buttons, keyboards, and controls, should be seen in this area, at the work surface, and in the plane of the operator's eyes.

- The optimal viewing distance for visual displays is about 50 centimeters (20 inches). Workers with refractive error or presbyopia can wear corrective lenses designed specifically for the job. Lenses of this type also can be incorporated into multifocal eyeglasses (progressive add lenses) with overviews (add on segment at the top of the lens).
- Proper illumination is important and may be evaluated for each task.

Visual performance can be impaired by whole-body vibration in the range of 10 to 25 cycles per second (hertz). Such vibration, which may be generated by power saws, cranes, conveyors, and other machinery should be damped or separated from the worker.

Vision Screening for the Worker

In order to determine the exact loss of function in patients with blurred vision and visual fatigue, a visual (ocular) function screening should be completed.

Elements of Visual (Ocular) Function

Visual (ocular) function requirements are important to the safety, health, and efficiency of industrial workers in nearly all occupations. Vision is most important for identifying distant objects and for detailed perception of shape and color. Visual senses allow workers to judge distance and gauge movements in the visual field.

Visual screening was defined by a joint proposal from the American Academy of Pediatrics, the American Academy of Ophthalmology, the American College of Occupational and Environmental Medicine, and the American Academy of Pediatric Ophthalmology and Strabismology. The definition is based on the following:

- The key element is determination of screening visual acuity, both quantitative and bilateral.
- Graduated visual acuity stimuli should be employed to allow quantitative determination of visual acuity (e.g., Snellen chart).
- Screening may include determination of contrast sensitivity, ocular alignment, color vision, and visual fields.

Current Testing Methods

Most required visual tests may be provided by using visual screeners (see "Additional Resources").

F.6 Work Activities

THE PROSPECTIVE WORKER

In order to apply the post-offer examination findings, detailed knowledge of the job is required.

Such information is derived from a visual analysis of the occupation. These data, or visual skills demanded of the worker, are written into the job requirements and meet with eventual tabulation.

This visual survey must be accomplished so that the occupational health professional can enter the shops, learn the jobs and shop language, and be completely familiar with the workers' daily environment. From this point on, the occupational health professional can be of great help to the medical director and the personnel director, who are trying to place new workers in jobs where they can attain their full work capacity. From material gained at the time of the visual analysis, the worker receives eye protection for the job requirements and is offered protection against impact through the use of case-hardened glass or plastic. Using such a device provides a twofold result—good working vision and eye protection. Knowing the job's requirements is necessary to prescribe proper lenses because occupational glasses offer visual potential based on the working distance and a safety defense determined by the job's characteristic hazards.

Each individual applying for a position should undergo a complete physical appraisal, which should include a vision (ocular) screening procedure. In more progressive plants, the visual screening could include a battery of tests supplied by a single ocular screening or rating instrument. In a well-integrated program, the results from these procedures can then be matched against the job's visual requirements. Failure to meet the guidelines established for that particular job places the worker and company management at risk both from a safety and production standpoint. The occupational health practitioner can play a key role by using these tests to interpret the job applicant's visual skills. In large plants, the practitioner interprets the findings of testing done by nontechnical employees (e.g., ophthalmic personnel or occupational health nurse). Small organizations will conduct the examination themselves or have it done by an off-site primary care physician. Medical and personnel directors can then use the test and examination data to place the prospective worker in a job best suited to his or her visual function.

G. Therapeutic Procedures

G.1.a Patient Comfort

Comfort is often a patient's first concern. Nonprescription analgesics provide sufficient pain relief for most patients with acute eye symptoms. Persistence of eye pain is a red flag. If treatment response is inadequate (i.e., symptoms and limitations continue), prescription pharmaceuticals can be tried, but only briefly, before referring the patient to an ophthalmologist or optometrist. Comorbid conditions, side effects, cost, and provider and patient preferences guide the clinician's choice of recommended agents. Table 11 summarizes comfort options.

Generally, three sources of pain are secondary to a red eye:

- Periorbital pain
- Cornea, conjunctival, or eyelid pain
- Ciliary and iris spasm

Conditions that require referral must be diagnosed and treated initially, and the patient must be stabilized while making preparations for transfer. A series of general and diagnostic treatment modalities for red flag and non-red flag conditions are provided.

G.1.b Anesthetic Agents

Topical anesthetics of short onset and duration with a low potential for causing hypersensitivity (e.g., proparacaine hydrochloride 0.5%) are used commonly during the eye examination and treatment only to facilitate removal of superficial foreign bodies or rust rings or to facilitate the examination when blepharospasm or severe local pain prevents adequate visualization of the eye (e.g., in patients with flash burns or severe corneal abrasions). The agents listed allow the clinician to perform ocular procedures such as tonometry, removing foreign bodies from the surface of the eye, and lacrimal canalicular manipulation and irrigation. Cocaine, the prototypical topical anesthetic, is a natural compound; the others are synthetic. Cocaine is now used rarely as an anesthetic agent. Topical anesthetics should not be used on an open globe. The practitioner should inquire about allergy to local anesthetics before using them. Proparacaine hydrochloride has the shortest onset, duration, and hypersensitivity. Chronic use by welders, for example, may lead to keratitis. Take adequate precautions to prevent pilfering of the clinic's bottles of anesthetic. A new delivery system for topical ophthalmic anesthetics in the form of a strip is now in the final pre-distribution stage.

Topical Anesthetic Agents		
USP or National Formulary Name	Trade Name	Concentration (%)
Cocaine hydrochloride	—	1-4

Proparacaine hydrochloride	AK-Taine	0.5
	Alcaine	0.5
	Ophthetic	0.5
Tetracaine hydrochloride	AK-T-Caine	0.5
	Pontocaine hydrochloride	0.5

Source: From *Physicians Desk Reference for Ophthalmic Medicine*. 3rd ed. 2002; Table 11.

G.1.c Analgesics

SYSTEMIC

The safest and most effective analgesic medication for acute eye problems appears to be acetaminophen. Opioids may be no more effective than acetaminophen but should be avoided if possible or used only until an emergent referral to an ophthalmologist or optometrist is made.

OPHTHALMIC TOPICAL

Four topical nonsteroidal anti-inflammatory drugs (NSAIDs) are available for ophthalmic application that function as local anesthetics and analgesics. They are diclofenac, flurbiprofen, ketorolac, and suprofen. Flurbiprofen and suprofen, which are indicated only to inhibit intraoperative miosis, are very similar in activity, and some hospitals use them interchangeably. Diclofenac has an official indication for the postoperative prophylaxis and treatment of ocular inflammation. Ketorolac is indicated for treating postoperative inflammation and relieving ocular itching due to seasonal allergic conjunctivitis. It also has shown some success in alleviating the pain associated with keratotomy. Both diclofenac and ketorolac also have been used successfully to prevent and treat cystoid macular edema. NSAIDs cause little, if any, rise in IOP. The Ocular PF ophthalmic solution of ketorolac without preservative does not cause such transient stinging and burning on instillation (20% of patients in a clinical trial) whereas the solution with preservative does (40%). Diclofenac (Voltaren ophthalmic) caused stinging and burning in 15% of patients, but keratitis was reported in 28% of patients undergoing cataract surgery. Ocular PF ophthalmic would appear to be best tolerated by patients with fewer side effects. Studies that evaluated the effectiveness of an ophthalmic NSAID in treating noninfected, non-contact lens-related, traumatic corneal abrasions without a pressure patch have been completed (Kaiser, 1995). After randomization, patients receiving ketorolac tromethamine 0.5% ophthalmic solution noted significantly decreased levels of pain, photophobia, and foreign-body sensation compared with the control vehicle group. In addition, the time before resuming normal activities was shorter in the group that received ketorolac tromethamine 0.5% ophthalmic solution. There was no statistical difference in the amount of tearing, healing time, acuity changes, or complication rates between the two groups. Ketorolac tromethamine 0.5% ophthalmic solution provides increased patient comfort without clinically adverse effects when used as adjunctive therapy in treating noninfected, non-contact lens-related traumatic corneal abrasions (Kaiser, 1995).

G.1.d Pressure Patching

The cornea is richly supplied by sensory nerves whose endings ramify in the epithelium. These nerves are among the most sensitive in the body. A corneal epithelial defect produces immediate pain, tearing, photophobia, and foreign body sensation that often motivates the patient to seek

medical attention. A corneal abrasion is limited to the superficial corneal epithelium and usually results from trauma secondary to fingers, branches, paper, or metal. The defects generally heal within 2 to 3 days without any long-term complications. Corneal abrasions are very common and account for up to 10% of new admissions to eye emergency units (Kaiser, 1995).

Antibiotic ointment, with or without a topical mydriatic and a pressure patch, has been the traditional treatment of traumatic, non-contact lens-related corneal abrasions. Unfortunately, using a pressure patch is not a benign treatment because it removes binocular vision, can be uncomfortable for the patient, and may retard healing. Several studies have questioned the effectiveness of patching corneal abrasions. To date, no large-scale study has been performed to evaluate the effectiveness of pressure patching to treat traumatic corneal abrasions and after removing corneal foreign bodies.

Kaiser (1995) reported that patients with traumatic corneal abrasions healed significantly faster, had less pain, and had fewer reports of blurred vision when they were not wearing a patch. There was no difference in the amount of photophobia, tearing, foreign-body sensation, or blurred vision. Finally, compliance in the no-patch group was better. In Hulbert's study (1991) of both pain and healing after foreign-body removal, it appears that both parameters were influenced favorably by not patching.

Potential disadvantages of patching can be noted. Pseudomonas ulcers have been documented after eye patching of corneal abrasions caused by contact lenses. Also, patching has been noted to decrease the natural irrigation effect of tears and to decrease corneal oxygen tension while increasing corneal temperature. Adverse effects on depth perception and visual fields are well known. In cases of open globe and/or major injury to the orbit, the injured eye should be covered with a metallic or plastic shield for protection.

G.1.e Mydriatics and Cycloplegics

The autonomic drugs that produce mydriasis (pupillary dilatation) and cycloplegia (paralysis of accommodation and iris constriction muscles) are among the most frequently used topical medications in ophthalmic practice. The most commonly used mydriatic is the direct-acting adrenergic agent phenylephrine hydrochloride, usually in a 2.5% concentration. The other mydriatic, an indirectly acting adrenergic hydroxyamphetamine, is available only in combination with tropicamide. Phenylephrine is used alone or, more commonly, in combination with a cycloplegic agent for refraction or for pupillary dilatation. The 2.5% concentration is favored for these cases. The possibility of severe adverse systemic effects arises from using the 10% solution. Anticholinergic agents have both cycloplegic and mydriatic activity. They usually are used for refraction, pupillary dilatation, relief of inflammation, and relief from iris and ciliary spasm. It is important to remember that the effect of these medications depends on many factors, including age, race, and eye color. For example, the mydriatics and cycloplegics tend to be less effective in dark-eyed than in blue-eyed individuals.

Mydriatics and Cycloplegics			
Generic Name	Trade Name	Concentration (%)	Onset/Duration of Action

Phenylephrine hydrochloride	AK-Dilate Mydrin Neo-Synephrine Available generically	Solution 2.5%, 10% Solution 2.5% Solution 2.5%, 10% Solution 2.5%, 10%	30-60 minutes/3-5 hours
Hydroxyamphetamine hydrobromide*	Paremyd	Solution 1%	15-60 minutes/3-4 hours
Atropine sulfate	Atropisol Atropine-Care Isopto Atropine Available generically	Solution 1% Solution 1% Solution 1% Solution 1% Ointment 1%	45-120 minutes/7-14 days
Cyclopentolate hydrochloride	AK-Pentolate Cyclogyl Pentolair Available generically	Solution 1% Solution 0.5%, 1%, 2% Solution 1% Solution 1%	30-60 minutes/6-24 hours
Homatropine hydrobromide	Isopto Homatropine Available generically	Solution 2%, 5% Solution 2%, 5%	30-60 minutes/3 days
Scopolamine hydrobromide	Isopto Hyoscine	Solution 0.25%	30-60 minutes/4-7 days
Tropicamide	Mydriacyl AK-Tropicacyl Available generically	Solution 0.5%, 1% Solution 0.5%, 1% Solution 0.5%, 1%	20-40 minutes/4-6 hours

*In combination with tropicamide 0.25%.

Note: Dapiprazole hydrochloride (Rev-Eyes) ophthalmic solution 0.5% sterile (Bausch&Lomb Pharmaceutical, Inc.). Source: From Physicians Desk Reference for Ophthalmic Medicine. 3rd ed. 2002; Table 2.

The drug dapiprazole hydrochloride can be used to reverse the effects of phenylephrine and, to a lesser extent, tropicamide. Activity against phenylephrine is excellent: 88% reversal is seen at the end of 1 hour. Against tropicamide, results are significantly lower: 38% at the end of 2 hours. When using both drugs, it therefore remains important to instruct the patient to use sunglasses and to avoid driving or operating dangerous machinery. There is no significant alteration in IOP in normotensive (intraocular tension with normal pressure under glaucoma treatment) glaucomatous eyes.

G.1.f Antimicrobial Therapy

Antibiotics are used routinely in ophthalmology and optometry for both treatment and prophylaxis. They are used prophylactically to manage foreign bodies and corneal abrasions and in pre- and postoperative care, where they are administered as an ophthalmic solution or ointment. Because these antibiotics are prescription drugs, no known over-the-counter antibiotics are available to be used. Corneal abrasions associated with contact lens wear are commonly evaluated and treated in acute care clinics and emergency departments by non-ophthalmologists and non-optometrists.

The risk of progression to suppurative keratitis in this setting requires management distinct from that of other mechanical (e.g., fingernail scratch) corneal abrasions. The antibiotic chosen should reflect the need for prophylaxis against *Pseudomonas*. Conditions favoring bacterial growth, specifically occlusive patching and/or use of steroid-containing compounds, must be avoided, and a 24-hour follow-up examination is recommended (Schein, 1993).

Again, ensure that the patient is not allergic to the proposed antibiotic prior to its use. Patients with more serious conditions, such as bacterial corneal ulcers (red flag), or those whose foreign-body abrasion is not healed in 24 hours or is showing no evidence of healing should be referred to an ophthalmologist or optometrist for further treatment. Patients with potentially contaminated corneal abrasions or foreign bodies may have their tetanus immunization evaluated and may be treated in accordance with the tetanus immunization protocol.

Commercially Available Ophthalmic Antibacterial Agents			
Generic Name	Trade Name	Concentration of Ophthalmic Solution (1%)	Concentration of Ophthalmic Ointment
Individual Agents			
Bacitracin	AK-Tracin	Not available	500 units/g
Chloramphenicol ^a	AK-Chlor	0.5%	Not available
	Chloromycetin	0.16-0.5%	1%
	Chloroptic	0.5%	1%
	Available generically	0.5%	Not available
Ciprofloxacin hydrochloride	Ciloxan	0.3%	0.3%
Erythromycin	Llotycin	Not available	0.5%
	Available generically	Not available	0.5%
Gentamicin sulfate	Garamycin	0.3%	0.3%
	Genoptic	0.3%	0.3%
	Gentacidin	0.3%	0.3%
	Gentak	0.3%	0.3%
	Available		

	generically	0.3%	0.3%
Levofloxacin	Quixin	0.5%	Not available
Norfloxacin	Chibroxin	0.3%	Not available
Ofloxacin	Ocuflox	0.3%	Not available
Sulfacetamide sodium	AK-Sulf	10%	10%
	Bleph-10	10%	10%
	Cetamide	Not available	10%
	Isopto Cetamide	15%	Not available
	Sulamyd Sodium	10%, 30%	10%
	Sulf-10	10%	Not available
	Available generically	10%, 15%, 30%	10%
Tobramycin sulfate	Tobrex	0.3%	0.3%
	Tobralcon	0.3%	0.3%
	Available generically	0.3%	Not available
Mixtures			
Polymyxin B/bacitracin zinc	AK-Poly-Bac	Not available	10,000 units
	Polysporin		500 units/g
	Available generically		
Polymyxin B/neomycin/bacitracin	AK-Spore	Not available	10,000 units
	Neosporin		3.5 mg
	Available generically		400 units/g
Polymyxin B/neomycin/gramicidin	AK-Spore	10,000 units	Not available
	Neosporin	1.75 mg	
	Available generically	0.025 mg/ml	
Polymyxin B/oxytetracycline	Terramycin	Not available	10,000 units
	TERAK		5 mg/g
Polymyxin B/trimethoprim	Polytrim	10,000 units	Not available
	Available generically	1 mg/ml	

^a Although noted, used very rarely.

Source: Table from *Physician's Desk Reference for Ophthalmic Medicine*. 3rd ed. 2002; Table 2.

G.1.g Ocular Anti-inflammatory Agents (Steroids)

The wide variety of medications available to treat ocular inflammation are listed below. Corticosteroids (steroids) are used most commonly, and many are available in combination with antibiotics and/or other medications. Ocular anti-inflammatory drugs (steroids) should not be initiated by the primary care physician but may be followed after initiation by an ophthalmologist or optometrist. Herpes simplex keratitis may be difficult to diagnose by a non-ophthalmologist or non-optometrist and can be extremely progressive when steroids are used without the presence of an appropriate antiviral agent.

Topical corticosteroids can elevate IOP and, in susceptible individuals, can induce glaucoma. Some corticosteroids, such as fluorometholone acetate, medrysone, and loteprednol, cause less elevation of IOP than others. Corticosteroids also may cause cataract formation, a complication more likely with high systemic use.

Topical Anti-inflammatory Agents		
Name and Dosage Form	Trade Name	Concentration
Topical Steroids		
Dexamethasone	Maxidex Ophthalmic Suspension	0.1%
Dexamethasone sodium phosphate ophthalmic ointment	AK-Dex Decadron Available generically	0.05% 0.05% 0.05%
Dexamethasone sodium phosphate ophthalmic solution	AK-Dex Decadron Available generically	0.1% 0.1% 0.1%
Fluorometholone ophthalmic ointment	FML S.O.P.	0.1%
Fluorometholone ophthalmic suspension	Fluor-Op FML FML Forte Available generically	0.1% 0.1% 0.25% 0.1%
Fluorometholone acetate ophthalmic suspension	Flarex Eflone	0.1% 0.1%
Loteprednol etabonate	Lotemax	0.5%
Medrysone ophthalmic suspension	HMS	1%
Prednisolone acetate ophthalmic suspension	Pred Mild Econopred Econopred Plus Pred Forte	0.12% 0.125% 1% 1%

	Available generically	1%
Prednisolone sodium phosphate ophthalmic solution	AK-Pred	0.125%
	Inflamase	0.125%
	Available generically	0.125%
	AK-Pred	1%
	Inflamase Forte	1%
	Available generically	1%
Rimexolone ophthalmic suspension	Vexol	1%
Nonsteroidal Anti-inflammatory Drugs		
Diclofenac ophthalmic solution	Voltaren	0.1%
Ketorolac ophthalmic solution	Acular	0.5%

Source: Table from *Physician's Desk Reference for Ophthalmic Medicine*. 3rd ed. 2002; Table 8.

Corticosteroids once were thought to be contraindicated in infectious disease states. However, it is now appreciated that steroids, when used in conjunction with appropriate antimicrobial, antifungal, or antiviral agents, may help to prevent more serious ocular damage. The correct diagnosis and appropriate agent are critical. Steroids may be administered by four different routes when treating ocular inflammation.

Usual Route of Steroid Administration in Ocular Inflammation	
Condition	Route
Blepharitis	Topical
Conjunctivitis	Topical
Episcleritis	Topical
Scleritis	Topical and/or systemic
Keratitis	Topical
Anterior uveitis	Topical and/or periocular
Posterior uveitis	Systemic and/or periocular
Endophthalmitis	Systemic/periocular, intravitreal
Optic neuritis	Systemic or periocular
Cranial arteritis	Systemic

Sympathetic ophthalmia	Systemic and topical
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Source: Table from *Physician's Desk Reference for Ophthalmic Medicine*. 3rd ed. 2002; Table 9.