The Red Eye, The Swollen Eye, And Acute Vision Loss: Handling Non-Traumatic Eye Disorders In The ED

1:15 a.m.: A 55-year-old woman arrives in the ED complaining of headache and vomiting. She felt fine when she went to bed but woke with a horrible frontal headache and retching. She has never had a headache like this before and has no significant past medical history. Although a quick neurologic exam is normal, you think, “I’ll get a head CT, and then I suppose I’ll have to tap her.” Before she leaves the department, the nurse cheerily asks, “Why is her eye so red?”

1:18 a.m.: Cancel CT.

Eye problems are common in every ED. While the exact number of emergency visits for eye complaints remains unknown, in the year 2000, nearly 4 million patients presented to U.S. EDs with a complaint referable to the ear or eye.1 The American Academy of Ophthalmology (AAO) estimates that one-third of all Americans have some ocular abnormality. Of these, one-quarter need corrective refraction to achieve normal vision. In all, 3 million Americans have impaired vision despite correction, and 890,000 are legally blind.2

This article addresses three basic complaints: the red eye, visual problems, and periorbital swelling. It addresses both common and unusual causes of eye disorders as well as the approach to specific populations (neonates, children, and the immunocompromised).

Clinical Guidelines

In the past several years, the AAO has published “Preferred Practice Patterns” on a variety of subjects. Each subcommittee of the AAO reviews the medical literature of the previous five years on a particular subject (e.g., conjunctivitis, blepharitis, acute angle-closure glaucoma, etc.). The Committee recommendations are rated A to C with respect to clinical importance, “A” being most important; “B,” moderately important; and “C,” relevant but not critical. The importance; “B,” moderately important; and “C,” relevant but not critical. The

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CME Objectives

Upon completing this article, you should be able to:
1. explain both common and rare causes of the red eye, swollen eye, and acute vision loss in children and adults;
2. determine which causes of non-traumatic eye disorders require ophthalmologic consultation;
3. list eye disorders that can threaten vision; and
4. describe treatment strategies for common eye disorders.

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See “Physician CME Information” on back page.
clinical evidence is also evaluated on a scale of I to III, with Level I reflecting at least one well-designed randomized clinical trial; Level II, non-randomized, case-controlled, or multiple-time trials; and Level III, case reports, descriptive studies, or expert opinion. These guidelines are discussed in further detail in the subsequent sections of this article.

“The eye is the jewel of the body.”—Henry David Thoreau (1817-1862), U.S. essayist, poet, naturalist

The Red Eye

While the red eye is a very frequent complaint in the ED, there are no definite data on its overall prevalence. The most common causes of the red eye include viral, bacterial, and allergic conjunctivitis. While emergency physicians are capable of treating the majority of patients who complain of a red eye, it is important to differentiate benign and self-limited conditions from more serious processes. (See Table 1.) Such vision-threatening conditions include acute angle-closure glaucoma, scleritis, uveitis, and keratitis. (See Table 2 on page 3.)

History

The AAO categorizes the following elements of the history as “A,” or most important; however, the evidence for these is rated as Level III:

- Symptoms and signs: itching, discharge, pain, photophobia, blurred vision, colored halos around lights, headache, or brow pain
- Duration of symptoms
- Unilateral vs. bilateral
- Character of the discharge: purulent vs. clear
- Recent exposure to an infected individual
- Trauma: mechanical (as in rubbing an irritated eye) or foreign body, chemical, ultraviolet (UV) light (welder’s flash, excessive sunlight, skiing without sunglasses, tanning booth, etc.)
- Contact lens wear: the type of lens, duration of wear, hygiene, etc.
- Associated symptoms that may be related to systemic disease: genital discharge, dysuria, upper respiratory infection (URI), skin and mucosal lesions, joint swelling
- Allergy: any systemic complaints
- Use of topical (especially ophthalmic) and/or systemic medications
- Previous episodes of conjunctivitis
- Pregnancy status
- Family history of acute angle-closure glaucoma (this is given a rating of A-II)

The following are considered moderately important, again with Level III evidence:

- Use of personal care items (including eyeliners and other cosmetics)
- Previous ophthalmic surgery
- Presence of immune dysfunction (e.g., HIV, chemotherapy, immunosuppression)
- Prior allergic phenomena, such as atopy or Stevens-Johnson syndrome

Table 1. Diagnostic Characteristics of Selected Disorders That Cause A Red Eye.

<table>
<thead>
<tr>
<th>Characteristic or Site</th>
<th>Conjunctivitis</th>
<th>Episcleritis</th>
<th>Scleritis</th>
<th>Angle-Closure Glaucoma</th>
<th>Acute Anterior Uveitis</th>
<th>Superficial Keratitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperemia</td>
<td>Diffuse, more prominent toward fornices</td>
<td>Focal</td>
<td>Focal or diffuse</td>
<td>Diffuse; most prominent adjacent to limbus</td>
<td>Diffuse; most prominent adjacent to limbus</td>
<td>Diffuse</td>
</tr>
<tr>
<td>Discharge</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Minimal, if present</td>
<td>Yes (if infectious cause)</td>
</tr>
<tr>
<td>Pupil</td>
<td>Not affected</td>
<td>Not affected</td>
<td>Constricted if secondary uveitis present; otherwise not affected</td>
<td>Moderately dilated; unreactive to light</td>
<td>Constricted; poor response to light</td>
<td>Constricted if secondary uveitis present; otherwise not affected</td>
</tr>
<tr>
<td>Ocular pain</td>
<td>Essentially none</td>
<td>Mild to moderate</td>
<td>Moderate to severe</td>
<td>Moderate to severe (often with headache and vomiting)</td>
<td>Moderate</td>
<td>Moderate to severe</td>
</tr>
<tr>
<td>Vision</td>
<td>Generally not affected</td>
<td>Not affected</td>
<td>May be reduced</td>
<td>Severely reduced</td>
<td>Mildly to moderately reduced</td>
<td>Moderately to severely reduced</td>
</tr>
<tr>
<td>Cornea</td>
<td>Clear</td>
<td>Clear</td>
<td>Occasional peripheral opacity; otherwise clear</td>
<td>Hazy</td>
<td>May be hazy (not as prominently as in angle-closure glaucoma)</td>
<td>Hazy</td>
</tr>
</tbody>
</table>

Reproduced with permission: Leibowitz HM. The red eye. N Engl J Med 2000 Aug 3;343(5):345-351. Table 1. Copyright ©2000 Massachusetts Medical Society. All rights reserved.
The social history is considered relevant but not critical, again with Level III evidence:

- Alcohol and tobacco use
- Occupation and hobbies (e.g., welding, skiing, gardening)
- Travel
- Sexual activity

Other relevant questions may include exposure to metal hitting metal (hammering) or other foreign body exposure (drilling, grinding, etc.).

The Physical Examination

Visual acuity and inspection of the external structures of the eye and pupil must be part of any examination in patients with eye-related complaints. Other aspects of the physical examination will vary depending on the clinical presentation. Slit lamp, ophthalmoscopy, measurement of intraocular pressure (IOP), and fluorescein staining should be considered if directed by the history or other facets of the physical examination. The remainder of the HEENT examination may also be revealing.

Visual Acuity

Visual acuity is the “vital sign” of the eye. It is traditionally quantified using the Snellen chart, with the patient standing at a preset distance. The distance of the chart to the eye should be recorded. If the patient is illiterate or pre-verbal, use a picture chart or “tumbling E” chart (in which the patient may indicate which way the “E” is facing with his or her fingers). (See also the section on pediatric patients.) If the patient is unable to stand, use a “near chart” consisting of calibrated letters held at a preset distance from the eye.

It is important to test each eye separately to accurately determine vision in the affected eye. If the patient is unable to see either chart, walk the patient one half the distance to the chart and attempt again. If the patient can read the chart, the numerator of the visual acuity documented will be 10 instead of 20.

Another option is to ask the patient to look through a pinhole. (Construct one simply by poking an 18-gauge needle through a piece of thick paper.) The pinhole will correct most refractive errors and thus compensate for missing or forgotten glasses. An alternative to the pinhole is to have the patient use a hand-held ophthalmoscope when reading the chart. The patient can then “dial” up or down with the lens until the image is clear. The correction can then be recorded as, for example, 20/30 with a -8 lens.

Table 2. Glossary Of Terms.

- **Blepharitis**: inflammation of the eyelids
- **Conjunctivitis**: inflammation of the conjunctiva
- **Keratitis**: inflammation of the cornea
- **Episcleritis**: inflammation of the subconjunctival connective tissue and the blood vessels that course between the sclera and conjunctiva
- **Scleritis**: inflammation of the sclera
- **Anterior uveitis**: inflammation of the iris and ciliary body (also called iritis or iridocyclitis)
- **Posterior uveitis**: inflammation of the choroid

If none of these methods are successful, grossly estimate vision by asking the patient to count fingers at two feet. If they cannot count fingers, try hand motion or, failing that, light perception.

The External Eye Examination

Inspect the skin around the eyes as well as the lids and lid margins for rashes, swelling, vesicles, discoloration, and malposition of the lids. The character of the conjunctival discharge should be noted: A purulent discharge almost always means a bacterial infection (A-1). Also examine for preauricular adenopathy (A-II), which often accompanies viral conjunctivitis and chlamydial infections. One unusual (and unsettling) finding is a lice infection of the lids.

The Pupil

The size of the pupil and its response to light and accommodation may help narrow the differential diagnosis. In one study of more than 300 consecutive patients presenting to an eye clinic with a unilateral red eye, the presence of miosis (small pupil) in the affected eye was a fairly reliable indicator of iritis.

Miosis is often secondary to spasm of the ciliary muscle. While miosis is a nonspecific response to inflammation or injury, it generally indicates a disease process deep to the cornea. Two simple maneuvers also suggest ciliary muscle spasm. The first involves shining a bright light in the unaffected eye. If the opposite eye has iritis, the consensual response will elicit pain in the eye hidden from the light, a phenomenon known as consensual photophobia. Pain in response to accommodation may also indicate iritis. To perform this test, have the patient look across the room and then quickly look at your finger held a few inches in front of their face. If they complain of pain with this test, iritis is likely.

A detailed discussion of the pupillary exam as well as visual fields can be found in the section on acute vision loss.

The Slit Lamp

The slit lamp provides magnification of structures anterior to the iris, including the anterior chamber, cornea, and conjunctiva. Subtle abnormalities or small foreign bodies involving the cornea and palpebral conjunctiva are often best visualized with the slit lamp. The device provides a better assessment of corneal defects or infiltrates than ophthalmoscopy or visual inspection.

Some physicians routinely have trouble using the slit lamp. “Slit-lamp-aphobia” can easily be overcome using the following interventions. These include maintaining the patient’s forehead against the restraining strap, using a narrow, bright light at a very oblique angle, and moving the joystick of the slit lamp forward and backward to focus on various points between the cornea and iris. The presence of cells (sparkles) and flare (searchlight in the fog) suggests inflammation or trauma to the eye. (See the subsequent section on uveitis for specific findings.) While gross hypopyon (a meniscus of white cells in the anterior chamber) and hyphema (red cells) can often be visualized with an ophthalmoscope, smaller collections are seen more clearly.
Topical Anesthetics

Patients with acute eye irritation may require topical anesthesia to cooperate with the examination. Topical anesthetics such as proparacaine 0.5% (Ophthane, Ophtetic) or tetracaine 0.5% are used to facilitate examination of the eye and provide diagnostic information as well. These agents facilitate lid eversion, tonometry, fluorescein staining, and examination of the patient with severe eye pain. In addition, topical anesthesia can help differentiate pain due to simple corneal injury from pain due to iritis, glaucoma, or other serious eye problems. In one study, eye pain relieved in 93% of patients (sensitivity, 80%; specificity, 86%).

As to which agent to use, in one trial proparacaine was less painful than tetracaine. Dilution of proparacaine with a balanced salt solution (1:15 dilution) further decreases the burning sensation that many patients experience with 0.5% proparacaine.

Whichever anesthetic is used, it should be supplied in unit doses in order to prevent the spread of infectious agents. A 1987 study of an ophthalmology outpatient clinic revealed some disturbing findings. The usual practice in this clinic was to use one bottle of anesthetic drops for each slit lamp for an entire day, after which the opened bottles were discarded. Normally, the anesthetic is instilled prior to the fluorescein, but for this study, it was instilled afterward. At the end of the day, every bottle of anesthetic drops was touched to the surface of the eye, thus facilitating bacterial transmission. During the past 10 years, a small, highly portable electronic device called the Tono-pen has appeared on the market. It uses a strain gauge that converts the voltage changes to a digital read-out. A disposable “mini-condom” covers the tip to prevent cross-contamination. While it is relatively easy to use, it remains quite expensive. The Tono-pen is as accurate as the Goldmann tonometer in the usual physiologic ranges (10-30 mmHg). However, it slightly overestimates pressures in the low ranges and, more importantly, underestimates pressures in the high ranges. It is appropriate for general screening, but probably not in glaucoma clinics, where fine-tuning of pressures at the high end is needed.

The Schiotz tonometer is still in use in many EDs and outpatient clinics. It is a metallic tonometer that mechanically indents the cornea. The resultant deflection on the scale must then be converted using a supplied chart to arrive at the IOP reading. It is important to remember that the lower the measurement on the Schiotz, the higher the IOP. The Schiotz is somewhat cumbersome to use, can only be used in cooperative patients in the supine position, and must be cleaned thoroughly after each use. To use the Schiotz to its best advantage, it is helpful to ask the patient to look at a spot on the ceiling directly in front of their eye, taking care not to apply pressure to the globe when the eyelids are retracted.

Whichever tonometer is used, be sure to disinfect the contact surface against viruses and bacteria properly if disposable tips are not used. An effective solution is sodium hypochlorite (bleach) at 500 ppm. While tonometry is the most objective means to determine IOP, digital ballottement of the eye can provide important information. Gentle palpation of the globe in the setting of acute angle-closure glaucoma can confirm that the involved eye is much harder than the unaffected side. (Do not perform this maneuver in the setting of ocular trauma or suspected corneal perforation, as this may herniate intraocular contents.)
**Fluorescein Staining**

The final step in the evaluation of the red eye, when indicated, is fluorescein staining of the cornea. It can be performed with or without topical anesthesia. The lower lid is gently retracted, and the tip of a moistened fluorescein strip is touched to the lower conjunctival sulcus. This maneuver prevents accidental injury to the cornea by the fluorescein strip. (Touching the strip to the cornea ensures a corneal abrasion, providing every patient with a false diagnosis). Alternatively, hold the strip above the eye and apply a drop of sterile irrigant to it, allowing the fluorescent drop to plop into the conjunctival sac. Shine a cobalt blue or Wood’s light on the cornea in a darkened room. Fluorescein is readily picked up by damaged superficial cells or exposed deeper cells of the cornea. Absence of staining implies an intact or undamaged cornea. The pattern of staining can be helpful in pinpointing the diagnosis:

- A superficial criss-cross of mostly vertical lines (ice-rink sign) suggests a foreign body under the upper lid (either still present or already dislodged)
- A focal area of staining may indicate a corneal abrasion
- A deep area of staining with raised or blurred margins may indicate a corneal ulcer
- Herpes virus infections (see Figure 1) produce a characteristic pattern of dendrites, reminiscent of a bunch of grapes, tree roots, or a jagged flash of lightning (if you have a vivid imagination)

**Funduscropy**

Funduscropy rarely provides useful information in the red eye, as most etiologies involve processes in the anterior rather than posterior portion of the globe. In addition, the fundus may be difficult to visualize because of photophobia or defects in the cornea.

**Common, Benign Causes Of A Red Eye**

**Bacterial Conjunctivitis**

Emergency physicians recognize bacterial conjunctivitis as a common affliction. The main pathogens in adults include *Staphylococcus species, Streptococcus pneumoniae, and Haemophilus influenzae*—a microbiologic spectrum that varies little worldwide. Patients report a rapid onset of irritation as well as redness and a purulent discharge that usually starts in one eye and then spreads to the other within 48 hours. Patients with bacterial conjunctivitis often give a history of morning crusting and difficulty opening within 48 hours. Patients with bacterial conjunctivitis often give a history of morning crusting and difficulty opening.

**Gonococcal Conjunctivitis**

Gonococcal conjunctivitis is particularly aggressive, with the potential to spread quickly to deeper eye structures and result in permanent visual impairment. The incubation period is thought to be from a few hours to three days. Rarely, the onset can be 1-2 weeks. The spread is probably oculogenital, and patients should be questioned about urethral or vaginal discharge or other symptoms of STDs. An important clue to the diagnosis is a discharge so copious that it re-accumulates immediately, as soon as it is wiped away. (Some have made the imaginative comparison to a “waterfall of pus.”) The eye is “angry,” demonstrating bloody scleral injection, and the lids are often swollen and red. In one series, keratitis, anterior chamber inflammation, periorcular edema and tenderness, gaze restriction, and preauricular lymphadenopathy were common. The initial evaluation should look for evidence of corneal infiltrates or ulcerations. A Gram’s stain showing gram-negative diplococci is diagnostic, but a culture is generally obtained to confirm the diagnosis.

Such patients need parenteral antibiotics, saline
Chlamydial Conjunctivitis

Although exceedingly rare in the U.S., trachoma (a type of chlamydia ocular infection) is the number-one cause of blindness throughout the world. Chlamydial conjunctivitis, a less serious disease, is caused by different serovars than trachoma. It appears to be on the rise, paralleling the increase in genital chlamydial infections. Nearly 1% of adults with genital chlamydia may develop ocular involvement. In adults, the infection is contracted through ocular genital spread. The average incubation period is five days, with a range of 2-19 days. Untreated chlamydial conjunctivitis may persist for months.

Patients may appear to have a viral conjunctivitis, with diffuse conjunctival injection, superficial punctate lesions of the cornea, and preauricular lymphadenopathy. However, the discharge is mucoid, not watery, as in the case with viral infections. A distinctive finding in chlamydial conjunctivitis is a pathognomonic enlargement of bulbar conjunctival follicles. (Because these follicles develop between the second and third week of infection, they may not be evident at the time of the ED visit.) The diagnosis can be made in the laboratory with either a direct immunofluorescent antibody test or by chlamydia culture. Treatment is systemic: either azithromycin 1 g (Zithromax) PO or doxycycline 100 mg BID for seven days. Patients should be screened for syphilis, and sexual contacts should be treated for chlamydia.

Viral Conjunctivitis

Viral conjunctivitis is the leading cause of red eye, although exact numbers remain obscure. There are many viruses that produce conjunctivitis. Among the most important are the adenoviruses, often associated with epidemic keratoconjunctivitis. Transmission is most commonly from hand to eye, but other vectors include contaminated tonometers, improper hand-washing technique, and contaminated swimming pools. Epidemics may occur in densely populated habitats, such as military outposts or school dormitories.

Viral conjunctivitis usually presents with diffuse conjunctival redness, lid edema, and a copious, watery discharge. Patients may find a puddle of clear ocular discharge on their pillow each morning. A preauricular lymph node, while characteristic (and also seen with chlamydial infections), is not always present. Other distinctive findings may include subconjunctival hemorrhage, corneal erosions, or punctate keratitis. The symptoms may last a week, often spreading to the other eye.

There is no role for routine viral cultures. Spontaneous resolution occurs in the vast majority of cases; however, occasional complications of superficial keratitis, membranous conjunctivitis, and conjunctival scarring occur.

In most cases of viral conjunctivitis, no specific treatment is necessary. Topical steroids are used only when severe keratitis is present (see the subsequent section on keratitis). In patients with a conjunctival membrane (membranous conjunctivitis), debridement of this casing followed by topical steroids may hasten recovery. While some authorities recommend topical antibiotics, ostensibly to prevent secondary bacterial infection, there is little evidence and no trials within the past 15 years to support this practice. Some authors cite patient expectation to justify prescribing topical antibiotics. (“What? We always get drops for pinkeye! The school nurse said you would give us some.”) Studies comparing topical ketorolac 0.5%, trifluridine 1.0% (Viroptic), dexamethasone 0.5%, and artificial tears (the placebo) found no statistical benefit in any of the treatment arms.

All cases are highly contagious for about seven days, and families should be educated on proper hygiene (separate towels, frequent hand-washing, avoiding touching the nose or lips, and avoiding close contact). This needs to be taken into account when advising healthcare or daycare workers on when to return to work. Most adults who can maintain careful hygienic measures can return to work before the conjunctivitis has resolved. The emergency physician should be acutely aware of potential patient-to-physician (and then to another patient) transmission. Use disposable gloves during the examination and wash hands vigorously after touching a patient with conjunctivitis.

Allergic Conjunctivitis

Allergic conjunctivitis is often a seasonal phenomenon in sensitive individuals. Vernal and atopic conjunctivitis are chronic conditions that begin in childhood and wax and wane for decades before recurrences finally cease. Some patients develop ocular symptoms during a generalized allergic flare, in response to a specific allergen such as cat dander, or in response to certain ophthalmic preparations (Neomycin being the classic). Up to 15% of soft contact lens users may develop an allergic response to the lenses. Mast cells, present in many eye structures, are involved in the allergic response, with histamine being the culprit in the actual symptom complex.

During the acute flare, the major complaint is itching, accompanied by burning and tearing. The discharge is mucoid or stringy. On physical exam, both eyes are usually injected, and the bulbar conjunctiva is often strikingly edematous (chemosis). Some patients may even have bulging tissue protruding from their closed lids. In atopic conjunctivitis, the eyelids may become thickened or scarred, with loss of eyelashes. In atopic and vernal types, the tarsal conjunctiva is often hypertrophied. In contact-lens-associated giant papillary conjunctivitis, large, square papillae (often described as cobblestones) are present on the palpebral conjunctiva, best seen by inverting the lower lid.

The treatment of allergic conjunctivitis depends on the etiology. In mild cases, removing the allergen (such as the
eyedrops), if possible, using warm compresses, and avoiding eye-rubbing may be all that is needed. In cat-sensitive individuals, eye-rubbing in the presence of cat dander causes a prolonged allergic response.30 Topical over-the-counter vasoactive drops that contain a combination of vasoconstrictor and antihistamine (such as Vasoco-A or Naphcon-A) may provide temporary relief of itching, swelling, and tearing.41 The topical nonsteroidal medications ketorolac 0.5% and diclofenac 0.1%, one drop QID, have both been shown to be effective in reducing symptoms at seven days.41-43 Ketorolac is the only topical NSAID to have an actual FDA indication for allergic conjunctivitis.44 Mast cell stabilizers (sodium chromolyn [Crolom], lodoxamide [Alomide], or nedocromil [Alacril]) have shown statistically significant improvement in several trials.45-47

“Your a parasite for sore eyes.” —actor Gregory Ratoff

Blepharitis

Blepharitis is an acute or chronic inflammation of the eyelids, often accompanied by conjunctival irritation. It may be caused by a variety of bacterial, viral, or parasitic infections as well as allergic, systemic, or dermatological diseases. Blepharitis is divided into three main categories: staphylococcal or seborrheic (both of which involve the anterior eyelid) and meibomian gland dysfunction (which involves the posterior eyelid). These conditions may be difficult to differentiate on initial examination.

Keratoconjunctivitis sicca, a condition of low tear volume, is associated with up to 50% of staphylococcal blepharitis cases. Patients with dry eyes often complain of eye irritation, fluctuating vision, red eyes, and sometimes photophobia, symptoms suggestive of an unstable tear film.49 Keratoconjunctivitis sicca may be diagnosed by the Shirmer test (a qualitative measurement of tear production using filter paper placed in the lower fornix) or observing the rate of dilution of fluorescein on the surface of the cornea.50

Inspection of the eyelids in blepharitis may show chronic scaling, erythema or edema of the lid margins, abnormal direction of eyelashes, or peculiar apposition of the lid margins (entropion or extropion). The staphylococcal variety tends to have hard, crusty deposits at the lid margins, whereas those of the seborrheic type are often oily or greasy.51 The magnitude of the findings reflects the severity and the chronicity of the disease. There are no specific diagnostic tests for blepharitis. Cultures of the lid margins may be done for recurrent, severe inflammation or cases resistant to standard therapy.

Treatment of blepharitis involves a daily regimen of lid hygiene: warm compresses to soften the encrustations and warm the meibomian secretions, followed by brief, gentle massage of the eyelids, then cleaning the lids with a commercial preparation or baby shampoo diluted 1:10. Erythromycin ophthalmic ointment is useful when staphylococcal blepharitis is suspected. Instruct the patient to apply the ointment to the lid margins several times daily for one or more weeks. For resistant and severe inflammation, a one- to three-week course of topical steroids is often used. Most patients with blepharitis need to be counseled about the chronicity of the problem, the likelihood of recurrent flare-ups, and the need for a regular regimen of lid hygiene.52 Patients with keratoconjunctivitis sicca are managed initially with artificial tear supplements.

Pinguecula And Pterygium

Pinguecula, a benign degeneration of the conjunctiva related to UV light exposure and aging, appears as a fatty-looking yellow spot, usually on the nasal aspect of the conjunctiva. A pterygium develops over years in individuals who spend a lot of time exposed to UV light (e.g., farmers and fishermen). It appears as a raised, yellowish, fleshy lesion, usually on the nasal aspect of the bulbar conjunctiva. It usually extends only to the peripheral cornea, seldom interfering with vision.4 Occasionally these lesions become inflamed, leading to complaints of eye irritation, foreign body sensation, pain, or tearing. The erythema is usually confined to the areas surrounding the lesions.

Topical indomethacin (not available in the U.S.) has been shown to reduce the inflammatory symptoms of both pinguecula and pterygium when used over a 14-day period.52 It performed as well as topical dexamethasone in a small prospective, randomized trial.53 Several nonsteroidal ophthalmic preparations are available in the U.S.: ketorolac 0.5% (Acular), diclofenac 0.1% (Voltaren), flurbiprofen 0.03% (Ocufern), and suprofen 1% (Profenal). Most have an FDA indication for ophthalmologic surgery and have been used extensively and with good results in controlling postoperative inflammation.53 Such drugs may be useful in treating an inflamed pinguecula or pterygium, but hard data are slim.

Corneal Abrasions

Corneal abrasions are defects of the normal corneal epithelium caused by trauma from small objects (often a fingernail, twig, hairbrush, or comb). They also occur after removal of a foreign body. Corneal abrasions from contact lenses represent a separate category with a unique set of clinical problems. In a one-year survey of admissions to a British emergency eye clinic, corneal abrasions accounted for 10% of the visits.54

Corneal abrasions are quite painful, and most people do not return to full functioning until the abrasion is healed. Patients describe immediate, sharp pain followed rapidly by tearing, photophobia, a decrease in visual acuity, and a persistent foreign body sensation. The eye will appear injected. Topical anesthetic drops will often significantly improve the pain, reduce blepharospasm, and allow a full examination. Fluorescein staining reveals the corneal defect. The magnification provided by a slit lamp allows a detailed
quantification of the size as well as the depth of the lesion. The natural history of most abrasions is full healing in 2-3 days. Except in the cases of abrasions associated with contact lens use, infection occurs in fewer than 1% of cases.

Until the mid-1990s, accepted therapy involved occlusive eye patches, antibiotic ointments (felt to be more soothing than drops), oral analgesics, and optional cycloplegics. The theory behind the occlusive patches was to provide a stable corneal environment to promote rapid re-epithelialization. Patches were also thought to reduce pain. A meta-analysis by Flynn et al in 1998 that combined five randomized clinical trials showed no statistical difference in healing between patched and un-patched eyes, and no reduction in pain in patients whose eyes were patched. These trials, however, enrolled only patients with small- to moderate-sized abrasions (< 10 mm²). Large abrasions seem to enjoy improved healing if patched.

Unlike eye patching, topical NSAID drops may improve patient comfort. In one randomized, double-blind, placebo-controlled trial of 100 patients with corneal abrasions, topical ketorolac 0.5% (Acular) was shown to reduce pain and photophobia significantly at the one-day mark. The ketorolac group was also able to return to function one day sooner, on average, than the placebo group. There was no difference in rates of healing or complications. A smaller study using diclofenac 0.3% (Voltaren) showed a small but statistically significant improvement in pain scale at two hours. The exact mechanism of action of these topical NSAIDs has not yet been delineated. It is probably some combination of reduction in pain sensation and anti-inflammatory effect. For traumatic, non-contact lens abrasions with significant pain, Kaiser et al recommend ketorolac 0.3% QID for three days or until the patient is comfortable, a broad-spectrum antibiotic ointment TID for three days or until the abrasion is healed, an optional short-acting cycloplegic such as cyclopentolate, and no patch (unless the abrasion is > 10 mm²). Many emergency physicians prescribe narcotic pain medicines for patients with corneal abrasions; these drugs are especially appreciated when the patient tries to go to sleep.

Corneal abrasions in contact lens users represent a distinct problem. There are approximately 25 million contact lens wearers in the U.S. They are all at increased risk of developing infected abrasions—referred to as ulcerative keratitis. Overnight, extended-wear soft lenses carry a 10- to 15-fold risk of infection. The causative organism is most often *Pseudomonas* species. The course can be fulminant, leading to permanent vision loss from corneal scarring.

**Do not patch corneal abrasions secondary to contact lens use.** In 1987, Clemons et al reported six cases of *Pseudomonas* keratitis following pressure patching for contact-lens-associated corneal abrasions. The occlusive patch favors bacterial replication by raising corneal temperature and interfering in the normal protective effects of routine eye blinking, tear exchange, and tear movement. The treatment of contact-lens-associated abrasions should begin with an antibiotic ointment that covers *Pseudomonas* (such as gentamicin [Genoptic] or combination polymyxin/bacitraclin). Steroid combinations should be avoided, as they may favor bacterial replication. Follow-up within 24 hours should be arranged, because suppuration of the abrasions can occur rapidly. Contact lens use should not resume until the abrasion is fully healed. The offending lenses should be replaced or inspected carefully for evidence of damage.

**Superficial Keratitis**

Superficial keratitis may be caused by UV light exposure (Welder’s flash or snow blindness), use (or overuse) of contact lenses, topical medications, dry eyes (keratoconjunctivitis sicca), blepharitis, as well as viral infections. Patients present with eye pain, redness, tearing, and decreased vision. In the case of UV keratitis, symptoms may not begin until 8-12 hours after exposure, prompting a nighttime visit to the ED. Both eyes are usually affected. Correlating the history with the characteristic ocular findings provides the diagnosis.

Fluorescein staining reveals multiple punctate lesions of the cornea, some of which stain intensely, others of which appear as tiny gray spots. Broad-spectrum topical antibiotics are prescribed to prevent infection, while cycloplegics provide significant pain relief if there is associated iritis (a full discussion of cycloplegics appears later). Oral pain medications are often needed as adjunctive therapy. The erosions of superficial keratitis generally resolve in 2-3 days. Patients who complain of continued symptoms should be referred to an ophthalmologist for follow-up.

**Less Common (But More Serious) Causes Of The Red Eye**

The following conditions can threaten vision and should be referred to an ophthalmologist. The majority of these disorders present with eye pain and redness.

**Corneal Ulcers**

Approximately 30,000 cases of corneal ulcers or microbial keratitis occur yearly in the U.S. The microbiology of bacterial infections differs somewhat depending on the geographic location, with staphylococci, streptococci, and gram-negative organisms being the most common. *Pseudomonas* species are the predominant organisms in contact-lens-associated cases. Acanthamoeba, fungi, herpes simplex virus (HSV), and herpes zoster (HZ) have also been implicated. The normal cornea is quite resistant to infection, due to the tight junctions between the top two layers of the cornea. Some preceding injury or insult is usually necessary for an infection to develop. The most common pre-existing condition is prior corneal surgery, such as corneal transplant, radial keratotomy, or cataract surgery.

**Ocular surface disease** (e.g., keratoconjunctivitis sicca or blepharitis), systemic diseases with eye involvement (e.g., rheumatoid disease or sarcoidosis), trauma, cranial nerve VII palsy, immunosuppression (including topical and systemic steroids), and contact lens wear are also risk factors for ulcerative keratitis.

Most patients with bacterial corneal ulcers complain of rapidly progressive eye pain, blurred vision (particularly if the ulcer is in the central field of vision), and photophobia. On occasion, a purulent discharge occurs. Parasitic, viral, or fungal infections may present in a more indolent fashion.

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**Emergency Medicine Practice**

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The presence of corneal abnormalities on physical examination differentiates ulcers from uncomplicated conjunctivitis. The cornea will appear hazy to the naked eye. The raised margins and crater of the ulcer can clearly be seen with the slit lamp, and the ulcer will stain intensely with fluorescein.

The use of Gram's stain, culture, and scrapings from the ulcer margins remains controversial. A survey of ophthalmologists in Southern California revealed that fewer than half used culture prior to initiating antibiotic therapy. The need for cultures is controversial because most ulcers respond to empiric therapy and because bacterial sensitivity results do not seem to correlate with clinical response. The AAO recommends that cultures be done in the following settings: a large or deep infiltration, an ulcer that is chronic or unresponsive to broad-spectrum antibiotics, or when there is a suspicion of fungal, amoebic, or mycobacterial infection. Cultures are best done with both a specialized platinum (Kimura) spatula and a dacron, calcium alginate, or cotton swab that is then inoculated directly onto the appropriate medium and transferred immediately to the laboratory. The type of medium is dependent on the type of infection suspected; consult with your microbiology lab. Cultures of the patient's contact lenses, lens case, or cleaning solution may be useful when acanthamoeba infection is suspected.

Four to six percent of ulcers do not respond to initial therapy. Because of the high potential for complications, such as corneal scarring, decreased vision, endophthalmitis, and ultimate enucleation, an ophthalmologist should be involved early in the care of such patients. Two large trials show that monotherapy with ofloxacin (Ocufox) or ciprofloxacin (Ciloxin) is as effective as combination therapy. Some academic ophthalmologists are concerned about emerging quinolone resistance to gram-positive organisms and choose instead to use combinations of fortified antibiotic solutions reconstituted from powdered parenteral preparations and artificial tears. They often have pharmacies willing and able to reconstitute these solutions. The most common first-line choice is cefazolin (Ancef) plus tobramycin or gentamicin. Specific directions for reconstituting these preparations can be found in the AAO Preferred Practice Pattern issue on bacterial keratitis. Loading doses of any of these medications are usually begun on a 5- to 15-minute cycle followed by every hour instillation for the first 24 hours. At this point, the eye is re-examined with a slit lamp. The regimen may be modified depending on clinical response. If topical steroids were being used, they should be discontinued, or at least reduced to the minimum amount needed to control the underlying condition. The initiation of topical steroids for corneal ulcers is controversial and without conclusive scientific evidence. Nevertheless, many ophthalmologists continue to use low-dose topical steroids for corneal ulcerations in the belief that they will reduce inflammation.

Many corneal ulcers may be managed on an outpatient basis with daily re-examinations. Ensure that the patient is able to comply with treatment. For instance, can an older or arthritic patient actually administer the eye drops? Will the patient actually return for next-day follow-up?

Acute Anterior Uveitis

Acute anterior uveitis, also called iritis or iridocyclitis, is an inflammation of the anterior portions of the uvea. The majority of cases are idiopathic; however, uveitis may develop in association with viral infections, HSV, HZ, or AIDS. Other implicated etiologies include Lyme disease, syphilis, toxoplasmosis, brucellosis, intraocular foreign bodies, and anterior segment ischemia. Acute iritis may also develop several days after blunt trauma to the eye. Unless specifically questioned, patients may not make the connection between the trauma and their current eye pain (although a “black eye” is a powerful clue). Patients complain of significant eye pain, tearing, and photophobia. The eye is injected, with the most marked hyperemia adjacent to the iris (the limbus); this is called limbal injection or flush. The pupil may appear constricted or irregular and may react poorly to light. Some patients demonstrate a moderate-to-severe reduction in visual acuity. As described in the section on the physical examination, testing for a consensual light reflex or accommodation will cause significant pain in the affected eye. The hallmark of anterior uveitis is the presence of inflammatory cells and “flare” in the anterior chamber on slit lamp exam. The cells (“sparkle”) are quantified in a 1x1 mm slit on a scale of 0-4, such that +1 = 5-15 cells; +4 > 60 cells. The flare (“smoke”), which represents protein in the anterior chamber, is also quantified on a scale of 0-4, such that +1 = very slight, +4 = intense. The flare may persist even after the cells are gone. If the inflammation is severe, a layer of pus (hypopyon) will be evident on direct examination of the anterior chamber.

Topical steroids, prescribed after consultation with an ophthalmologist, are the cornerstones of therapy. Prednisolone (Predforte) is commonly prescribed, given every 1-2 hours initially, then slowly tapered over 3-4 weeks. A response to steroids is usually noted by 3-4 days. Intermediate-acting cycloplegics, such as homatropine or scopolamine, are used to control the pain from ciliary spasm. Sympathetic stimulants such as tropicamide (Mydriasil) are inappropriate, as they have no affect on the ciliary constrictor muscle. Cyclopentolate (Cyclogyl) is not recommended because it may aggravate the inflammation.

Because of the significant potential for long-term sequelae, such as glaucoma, pupillary abnormalities, cataract formation, and macular dysfunction, an ophthalmologist should be involved early in the care of anterior uveitis. Why do you hasten to remove anything which hurts your eye, while if something affects your soul you postpone the cure until next year? —Horace

HSV Keratitis

As many as 500,000 cases of ocular herpes infections are diagnosed in the U.S. each year. Primary HSV infections are associated with ocular lesions in 2%-6% of cases, whereas ocular lesions are present in 10%-30% of secondary disease. Secondary infection represents reactivation of the virus that has lain dormant, sometimes for decades, in the trigeminal ganglion. Many factors have been implicated in
reactivation: UV light, cold, wind, systemic illness, emotional stress, surgery, menstruation, minor local trauma, and immunosuppression, either from disease or medication. Recently, there have been case reports of HSV following laser refractive surgery as well as laser glaucoma surgery. 

Primary ocular disease rarely results in vision loss; however, each recurrence raises the risk of stromal immune response and permanent morbidity. The cumulative risk of recurrence is estimated at 30% per year.

The most common presenting complaints are irritation, tearing, photophobia, and blurred vision. Past medical history should include questions regarding facial lesions, such as cold sores or blisters, genital lesions, previous episodes of corneal ulcers or iritis, recent topical or systemic steroids, immunosuppressive diseases, or medications.

Corneal sensation, which may be tested using thin wisps of cotton from a sterile cotton-tipped applicator, is decreased in 80% of cases. When the herpes-infected eye is touched, the corneal reflex may be depressed or the patient may notice decreased sensation in the involved eye.

The best diagnostic test in the ED involves slit lamp examination in combination with fluorescein staining. Using the “blue light” on the slit lamp will illuminate the pathognomonic dendrites. Dendrites are single or multiple masses that have linear branches that end in terminal bulbs. However, depending on the time of presentation, these dendrites may not be present. HSV begins as a punctate keratitis, which then progresses to dendritic lesions that then coalesce into geographic ulcerations. Iritis with a cell or flare reaction on slit lamp exam occurs in 40% of cases.

Disease confined to the epithelium responds to topical or oral antiviral agents in 90%-95% of cases. Forty percent of cases resolve spontaneously without sequelae (but the ED physician should still treat them). Three topical antivirals ( trifluridine [Viroptic], vidarabine [Vira A], and idoxuridine [Stoxil]) and three oral preparations (acyclovir [Zovirax], famcyclovir [Famvir], and valacyclovir [Valtrex]) are available in the U.S. For disease limited to the epithelium, any of the topical or oral preparations are used for 10-14 days. A topical antibiotic preparation is added when ulcers are present. Acyclovir is the only oral agent to have been studied in ocular clinical trials. The other agents are used off-label based on genital infection trials.

Corticosteroids are sometimes used for HSV immune keratitis and keratouveitis. Their beneficial effects include inhibition of white cell infiltration, scar formation, and neovascularization. However, they may promote the spread of a superficial viral infection, increase the incidence of bacterial or fungal infections, and result in steroid-induced glaucoma or cataracts. They may also require a prolonged taper to prevent inflammatory rebound. Consult an ophthalmologist before instituting steroids. When steroid drops are used, a prophylactic antiviral preparation is added. A series of clinical trials by the Herpetic Eye Disease Study (HEDS) multicenter group did not show an improvement in resolution with the addition of oral acyclovir to a topical regimen. However, a subsequent HEDS trial showed that prophylactic oral acyclovir reduces the rate of recurrent HSV eye infections.

### Herpes Zoster
Like HSV, HZ is a disease of recrudescence, the virus emerging in specific nerve root distributions years after the original infection. HZ of the ophthalmic division of the trigeminal nerve (cranial nerve V) represents 10% of total HZ infections. In the vast majority of cases, the characteristic vesicular skin lesions precede ocular involvement.

A vesicular lesion on the tip of the nose (Hutchinson’s sign) is thought to be associated with a higher prevalence of eye infection. There are case reports of ocular involvement occurring prior to skin involvement and ocular lesions without skin involvement. Although HZ may affect many different areas of the eye, one-half of untreated opthalmic HZ involves the cornea. The corneal manifestations vary from punctate keratitis to ulcers to pseudodendrites to deeper stromal keratitis. The dendrites of HZ may be difficult to differentiate from those of HSV. About 40%-60% of immunocompetent patients develop an associated iritis. These patients are at risk for ocular hypertension. The characteristic skin lesions accompanied by corneal anesthesia are usually enough to make the diagnosis. A Tzanck smear of vesicular lesions or epithelial scrapings may show multinucleated giant cells but will not differentiate HZ from HSV. Corneal cultures, while diagnostic, take 1-2 weeks. PCR of tear film or corneal scrapings shows promise as a rapid test in hard-to-diagnose cases.

The skin lesions may be treated with a seven- to 10-day course of acyclovir (Zovirax, 800 mg 5 times a day), famcyclovir (Famvir, 500 mg TID), or valacyclovir (Valtrex, 1 g TID). All of these preparations promote healing and reduce pain if given within 72 hours of onset—best if within 48 hours. However, the incidence of post-herpetic neuralgia, a particular problem in the elderly, is unaffected. Topical antivirals have not been shown to be effective in HZ. Topical steroids should be reserved for cases of active stromal keratitis and anterior uveitis, under the direction of an ophthalmologist.

### Fungal Conjunctivitis
Fungal conjunctivitis is initially clinically indistinguishable from bacterial infection. The diagnosis may be suggested by a history of trauma from vegetation or work on a farm or vegetable garden, but sometimes the initial trauma is so mild as to have been forgotten. Contact lens users and immunocompromised patients are also at risk. Corneal scrapings and culture provide the diagnosis. Treatment, requiring topical antifungals and sometimes subconjunctival injection, is best left to the ophthalmologist.

### Acute Angle-Closure Glaucoma
One in 50 Americans over 35 is at risk for glaucoma; primary angle-closure glaucoma (PACG) represents about 10% of total cases. Risk factors for PACG include older age, female sex, a history of hyperopia (farsightedness), a family history of acute angle-closure glaucoma, Eskimo or Asian extraction, and a prior history of anterior uveitis. The pathophysiology of PACG involves obstruction of the trabecular meshwork. When the entire circumference of the anterior chamber angle is occluded, IOP increases and
corneal edema occurs. Attacks may be precipitated in a suspected individual by anything that causes excessive pupillary dilatation: low light, stress, fatigue, or medication with sympathetic or parasympathetic actions.7,8

Acute PACG classically presents as severe unilateral eye or brow pain, decreased vision, and colored halos around lights (due to corneal edema). Vagal stimulation from severe pain may precipitate significant nausea and vomiting. There are case reports of elderly patients presenting only with nausea and vomiting and generalized headache or with abdominal pain, which results in an extensive GI work-up or even laparotomy.7,9 While PACG can affect both eyes, an acute attack usually presents unilaterally with a red eye, hazy cornea, and a dilated, minimally reactive pupil. IOPs are often in the 50-70 mmHg range. The diagnosis is usually obvious by the combination of history and tonometry. Using a slit lamp and goniometer, an ophthalmologist can demonstrate that the peripheral iris blocks the trabecular meshwork. If a hazy cornea impedes a flashlight is held off to the side and parallel to the iris with the beam shining across the anterior chamber. If the whole iris is illuminated, the angle is open. If a shadow appears on the nasal aspect of the iris, the angle is narrow or closed. This test has a sensitivity of 80% and specificity of 69%.7,9

If untreated, acute angle-closure glaucoma will result in blindness within a few days. The initial treatment targets lowering IOP in preparation for definitive surgery.6 Call an ophthalmologist as soon as the diagnosis is made or strongly suspected. Accepted components of acute medical therapy include topical beta-blockers, a topical alpha-adrenergic agent (such as apraclonidine), oral or intravenous acetazolamide, topical steroids, and low-dose pilocarpine.7,8 Intravenous osmotic agents, such as mannitol, are effective in lowering IOP, but they need to be used with great caution in the setting of congestive heart failure or renal insufficiency. The miotic effect of pilocarpine is blocked at IOPs over 60 mmHg; however, the action on the ciliary muscle and the anterior movement of the lens continue, so that IOP may be paradoxically increased with aggressive use of pilocarpine. Many ophthalmologists have their own established routine, and even the AAO does not endorse one particular medical algorithm.4 (See Table 3.)

Scleritis
Scleritis, or inflammation of the anterior or posterior sclera, is an idiopathic disease that may represent the first sign of a connective tissue or systemic disease. There are three types of scleritis: diffuse, nodular, and necrotizing. The latter carries a severe prognosis not only for the eye, but also for the underlying disease. Scleritis has been associated with rheumatoid arthritis (the most common), relapsing polychondritis, systemic lupus erythematosus, Wegener granulomatosis, Cogan syndrome, polyarteritis nodosa, Takayasu disease, sarcoidosis, porphyria, syphilis, tuberculosis, brucellosis, Lyme disease, and HZ.7,9

Scleritis presents with a red eye, decreased vision, and eye pain without discharge.4 The eye is usually tender to palpation. In some cases the sclera may be so thin that the bluish uvea is seen shining through. Therapy with a nonsteroidal anti-inflammatory agent can be started; however, steroid preparations are usually needed to control the disease. If the disease is non-necrotizing, topical steroids are often prescribed initially. There is a high failure rate (47% in a small descriptive Canadian study),60 so that approximately 60% of patients end up on oral steroids or other immunosuppressive drugs.61 Referral to an ophthalmologist should be done promptly, because of the often chronic nature of the disease, the potential to permanently impair vision, and to rule out necrotizing scleritis.4,79

Episcleritis
Episcleritis is an inflammation of the blood vessels that course between the conjunctiva and sclera. It is almost always a benign condition that resolves spontaneously in 1-2 weeks, never to return again. It may recur in about 20% of cases and can occasionally evolve into scleritis.79,82

Episcleritis usually presents as localized conjunctival erythema associated with mild ocular pain. The blood vessels are engorged in the affected region, and a nodule may be present as well. Vision, cornea, and pupil are all normal. Often, no therapy is required, but episcleritis often responds to oral4 or topical82 anti-inflammatory medications.

Corneal Perforation
The most common cause of corneal perforation is infectious breakdown of the corneal stroma. Perforations may also occur as a result of blunt or penetrating trauma to the eye, inflammatory conditions, environmental exposure (as in cranial nerve VII nerve palsies), and degenerative diseases. Presenting symptoms include pain, decreased visual acuity, and increased tearing. There are three clinical findings classic for perforation: a flat or shallow anterior chamber, uveal prolapse, and a positive Seidel test. The Seidel test is done as follows: After topical anesthesia, the area suspicious for perforation is painted with a sterile fluorescein strip dipped in some sterile saline. Using a slit lamp with a cobalt
blue light, the painted area is observed for clearing or
dilution of the fluorescein (sometimes an active swirled of
the dye is visible, representing the leaking aqueous).
Clearing or dilution represents a positive Seidel test and
confirms the perforation. Treatment is surgical. In patients
who are unable to undergo surgery, tissue adhesives are
sometimes used to seal the perforation.83 All perforations
should be rapidly referred to an ophthalmologist.

Endophthalmitis
Endophthalmitis is the most dreaded ocular infection. As
the word implies, it is a deep infection of the eye and carries
a poor prognosis. Patients who have had recent ocular
surgery are at risk for developing endophthalmitis.106 It
occurs in 0.1%-0.77% of cases post penetrating keratoplasty
(e.g., cornea, cataract, lens surgeries). At other times, it is a
complication of severe bacterial keratitis or ulceration.

Intravenous as well as subconjunctival or intraorbital
antibiotics are used in an attempt to save the eye.83

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Pediatric Considerations
The history for children presenting with a red eye requires
some additional elements:

- The age of the child
- The presence or absence of fever
- Ill contacts
- Pre- or postnatal STD infections in the mother

The physical examination can be challenging and often
needs to be adapted to the age of the child. For a pre-reader,
the eye chart should have pictures or “directional E’s.”
The extraocular muscle (EOM) examination can be accom-
plished using brightly colored objects to encourage the
infant or child to track the object. The retinal exam may
require that an assistant (or parent) distract the child over
the shoulder of the examining physician. Be sure to examine
the ears in young children with purulent eye discharge; they
may be suffering from the conjunctivitis-otitis syndrome and could
require oral, rather than topical, therapy.

Bacterial Conjunctivitis
Bacterial conjunctivitis presents as it does in adults with

Ten Pitfalls To Avoid

1. “An eye patch never hurt anyone.”
   Au contraire! The patient with a corneal ulcer who gets an
   eye patch is at risk for perforation. Do not patch corneal
   abrasions secondary to contact lens use.

2. “He just had some eye pain. That’s not an indication for
   visual acuities.”
   Having an eye complaint (and some say having an eye) is
   an indication for visual acuities. They are the “vital sign” of
   the eye.

3. “I know he had a lot of pus leaking from the eye, but I
   thought he would do fine with some Sulamyd.”
   This gentleman had gonococcal ophthalmia and was
   admitted the next day. Clues to this diagnosis include
   copious pus; an angry, often-hemorrhagic sclera;
   preauricular adenopathy; and anterior chamber
   inflammation. Gram’s stain of the discharge will reveal the
   gram-negative diplococci within the leukocytes. Such
   patients require admission and parenteral antibiotics.

4. “Since the H. flu vaccine, I've been sending all kids with
   periorbital cellulitis home on oral antibiotics.”
   This child did not do well, even after decompressive
   surgery to the orbit. While it is true that the fierceness of
   this disease has relented in the past decade, the decision to
   treat as an outpatient must be made on an individual basis.
   Children who appear toxic, those with proptosis or
   impairment of extraocular motions, and patients with
   decreased vision need admission and parenteral antibiotics.

5. “It looked like he had fire ant bites to his eyelids. Heck,
   they even bit him on the tip of his nose.”
   Hel-looo... fire ant bites? Patients with herpes zoster need
   acyclovir, not Benadryl.

6. “We don’t do fluorescein exams in our ED.”
   Start. Fluorescein exams are essential in diagnosing
   keratitis and corneal abrasions. They are also helpful in
detecting corneal ulcers and corneal perforations.

7. “He was complaining of eye pain. I told him to take a few
days off his job as a machinist and it would get better.”
   Intraocular foreign bodies rarely get better without surgery.
   A history of metal-on-metal exposure is key. Look for an
   irregular pupil. A Seidel test (see text) may be positive for
   leakage of aqueous humor.

8. “She was just another elderly woman with conjunctivitis
   who came to the ED in the middle of the night.”
   By the time the ophthalmologist saw her three days later,
   her IOP was over 80. Conjunctivitis usually does not present
   with severe eye pain, hazy cornea, and unreactive pupil.
   Check IOPs in suspicious cases; at the very least, compare
   the tension in each eye by fingertip assessment.

9. “I looked in his eye and didn't see a foreign body. All he
   had were a lot of vertical scratches to his cornea.”
   And a foreign body under the lid. Evert the lids when a
   patient complains of a foreign body sensation—especially
   when they demonstrate an “ice rink” sign.

10. “I thought she was malingering. She said she was almost
    blind in her right eye, but she blinked when I pretended to
    poke her in the eye. Besides, her pupil reacted to light.”
    She had optic neuritis, not hysteria. Patients with ON may
    still have a light reflex; the swinging flashlight test would
    have been abnormal (if it had been done). Blind patients
    still blink when a threat is made to their eye secondary to
    the corneal reflex in response to a rush of air. (Plus, there
    was nothing wrong with this lady's other eye.) ▲
injected conjunctivae and a purulent discharge. In preschoolers, it is the major cause of a red eye. The predominant organisms are nontypeable H. influenzae and S. pneumoniae, with Moraxella catarrhalis, Neisseria gonorrhoeae, and Neisseria meningitides occurring less frequently. N. gonorrhoeae may occur outside the neonatal population in a sexually abused child.

The syndrome of conjunctivitis/otitis begins as a low-grade fever and mild URI, but then progresses to a painful red eye with purulent discharge. Otitis media, either symptomatic or asymptomatic, may be present initially or develop later. It is usually due to nontypeable H. influenzae, less commonly to S. pneumoniae. Children who are less than 3 years old or who attend daycare are more likely to develop this syndrome.

Several prospective studies have demonstrated the efficacy of topical antimicrobials in hastening a clinical and bacteriologic cure. Trimethoprim-polymyxin B eradicated H. influenzae better than gentamicin sulfate or sodium sulfacetamide. One study demonstrated the efficacy and safety of topically applied ciprofloxacin ophthalmic solution when it was compared to tobramycin ophthalmic drops in 257 children with bacterial conjunctivitis.

There is some evidence to suggest the utility of systemic antibiotics effective against H. influenzae to treat bacterial conjunctivitis in children less than 6 years of age (whether or not they have an associated otitis media). Topical therapy does not eradicate nasopharyngeal carriage of H. influenzae or N. meningitides, nor does it prevent or treat an associated otitis media. Topical therapy may also be difficult for parents to apply successfully as directed. In the young child, the use of an oral agent effective against H. influenzae might be considered even in the absence of a documented otitis media. A three- to five-day treatment course of an agent such as amoxicillin/clavulanate results in a clinical and bacteriologic cure of the conjunctivitis and may be effective prophylaxis for the otitis. However, there is currently only one small, randomized study to support this approach. There is more supporting evidence for a 10-day course of oral antibiotics for bacterial conjunctivitis.

Systemic antibiotics successfully treat the conjunctivitis and prevent (or cure) the associated otitis media.

### Viral Conjunctivitis

Viral conjunctivitis is the most common cause of conjunctivitis in school-aged children. Viral conjunctivitis ranks second after bacteria as the cause of acute conjunctivitis in preschoolers. Adenovirus predominates in the fall. Viral conjunctivitis is more commonly associated with pharyngitis than bacterial conjunctivitis. Conjunctivitis caused by adenovirus may be hemorrhagic, and some children can demonstrate significant periorbital swelling. Presentation and treatment (or lack thereof) are similar to adult viral conjunctivitis. Unlike adults, who can be expected to follow recommendations on hygiene and minimizing viral transmission, children should probably be kept home from school or daycare for approximately one week.

“You can’t depend on your eyes when your imagination is out of focus.” —Mark Twain (1835-1910)

### Neonatal Conjunctivitis

Neonatal conjunctivitis is caused by N. gonorrhoeae, Chlamydia trachomatis, and, less commonly, HSV. Infection is acquired during passage through an infected birth canal. While neonatal ophthalmic prophylaxis is universal in the United States, it is not 100% effective. The medications used are 1% silver nitrate solution, 0.5% erythromycin ophthalmic ointment, or 1% tetracycline ophthalmic ointment. Only silver nitrate is effective against penicillinase-producing gonococcus.

Ophthalmia neonatorum, or gonococcal conjunctivitis, presents in the first week after birth, with fever and a profuse and purulent discharge that rapidly re-accumulates.

Continued on page 17
Clinical Pathway: Management Of The Red Eye

- Obtain visual acuities (Class I)
- Perform eye examination (Class I)
- Evaluate for foreign body (lid eversion as indicated) (Class I)
- Fluorescein exam (Class I)

- Significant pain?
  - Unreactive pupil?
  - Steamy cornea?  
  - Photophobia?
  - Pain on accommodation?
  - Pain with consensual light response?  
  - Photophobia?
  - Pain?
  - Corneal ulcer?  

Yes → • Measure intraocular pressure (Class I)
  • Treat for acute angle-closure glaucoma if present (see Table 3) (Class I)

No  

Yes → • Evaluate for iritis (Class I)
  • Use slit lamp to look for cell or flare reaction in anterior chamber. If positive:
  - Consult with ophthalmologist (Class I)
  - Pred-Forte if approved by consultant (Class I)
  - Homatropine drops (Class II)

No  

Yes  

No → • Consult with ophthalmologist (Class I)
  • Determine need for corneal scraping (Class II)
  • Aggressive antibiotic therapy (see text) (Class I)

Go to top of next page

The evidence for recommendations is graded using the following scale. For complete definitions, see back page. **Class I:** Definitely recommended. Definitive, excellent evidence provides support. **Class II:** Acceptable and useful. Good evidence provides support. **Class III:** May be acceptable, possibly useful. Fair-to-good evidence provides support. **Indeterminate:** Continuing area of research.

This clinical pathway is intended to supplement, rather than substitute for, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

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Clinical Pathway: Management Of The Red Eye

- Photophobia?
- Decreased corneal sensation?
- Dendrites on fluorescein staining?

Yes → • Consult with ophthalmologist (Class I)
       • Oral acyclovir or topical antivirals (Class I)

No → • Photophobia?
       • Exposure to UV light?
       • Fluorescein exam demonstrating keratitis?

Yes → • Topical mydriatic (cyclopentolate or homatropine) (Class II)
       • PO narcotics (Class II)
       • Counseling regarding proper eye protection (sunglasses, welder's mask) (Class I)

No → • Exuberant, purulent discharge?
       • Angry or hemorrhagic conjunctivitis?
       • Preauricular adenopathy?
       • Keratitis?

Yes → • Evaluate and treat for gonococcal conjunctivitis (Class I)
       • Consider Gram's stain and culture of discharge (Class II)

No → • Purulent discharge?
       • Normal vision?
       • No keratitis?
       • No iritis?

Yes → • Age greater than 6: topical antibiotic drops (Class I)
       • Age less than 6: consider oral antibiotics (Class II)

No → • Clear discharge?
       • Preauricular adenopathy?
       • Normal vision?
       • No keratitis?
       • No iritis?

Yes → • No treatment necessary (Class I)
       • Give precautions regarding transmission of virus (Class I)

No → No treatment necessary (Class I)

The evidence for recommendations is graded using the following scale. For complete definitions, see back page. **Class I:** Definitely recommended. Definitive, excellent evidence provides support. **Class II:** Acceptable and useful. Good evidence provides support. **Class III:** May be acceptable, possibly useful. Fair-to-good evidence provides support. **Indeterminate:** Continuing area of research.

This clinical pathway is intended to supplement, rather than substitute for, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

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Clinical Pathway: Diagnosing The Swollen Eye In Adults

Onset
Acute
Probable cause: infectious
- Normal host
Probable cause: bacterial
- Extraocular eye muscle movement?
  - Normal
    - Probable diagnosis: periorbital cellulitis
  - Painful or abnormal
    - Probable diagnosis: orbital cellulitis
- Immunocompromised host
Probable cause: fungal
Probable diagnoses:
- Tumor
- Thyroid-related ophthalmopathy
- Idiopathic orbital inflammatory syndrome

Gradual

Clinical Pathway: Diagnosing Acute Vision Loss In Adults

Age of patient
< 50
Probable diagnosis: optic neuritis
- Any age
Possble causes:
- Retinal detachment
- Infection
- Tumor
- > 60
Probable cause: vascular
- Systemic symptoms?
  - No
    - Retinal exam
      - Pale
        - Probable diagnosis: central retinal artery occlusion
      - Congested
        - Probable diagnosis: central retinal vein occlusion
  - Yes
    - Probable diagnosis: temporal arteritis

The evidence for recommendations is graded using the following scale. For complete definitions, see back page. Class I: Definitely recommended. Definitive, excellent evidence provides support. Class II: Acceptable and useful. Good evidence provides support. Class III: May be acceptable, possibly useful. Fair-to-good evidence provides support. Indeterminate: Continuing area of research.

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Continued from page 13

There is also lid edema and erythema. Corneal ulceration and perforation can occur.

Neonatal HSV infection occurs in up to 1/3000 live births. A mother with primary infection poses the greatest risk to her infant. HSV of the eye is part of the skin, eye, mouth disease; however, skin and mouth lesions may be absent at presentation. The conjunctivae are erythematous and with characteristic dendrites on fluorescein staining of the cornea.92

The incubation period for chlamydial conjunctivitis is longer, and so infants may present up to several weeks after birth. The conjunctiva is beefy red, with a watery or mucopurulent discharge. The infant may be afebrile. Among infants born to infected mothers, 12%-25% will develop chlamydial conjunctivitis.

It is important to differentiate ophthalmia neonatorum and HSV infection from chlamydia. The first two require a full sepsis evaluation, including complete blood count, chemistries, blood and eye cultures, lumbar puncture (LP), admission, and IV antibiotics. Emergent ophthalmic evaluation is mandatory for the potential for perforation with gonococcus and vision loss with HSV.92 Chlamydia, on the other hand, is largely an outpatient disease. Oral antibiotics are given to these patients because many of these infants will infect their respiratory tracts through infected drainage via the nasolacrimal ducts. There are no randomized clinical trials dealing with these diseases; however, clinical experience and the potential severity of the complications make aggressive treatment the current standard of care. (See Table 4.)

Kawasaki Disease

Kawasaki disease, or mucocutaneous lymph node syndrome, is a multisystem disease, probably of infectious origin, that presents with a bilateral, non-exudative conjunctivitis that spares the perilimbic area. It occurs primarily in children under the age of 8. The development of coronary artery aneurysms is a source of significant morbidity. Recognition of the disease by the emergency physician and resultant early treatment with intravenous immunoglobulin (2 g/kg over 10 hours) and high-dose aspirin (100 mg/kg) reduce both the duration of the disease and the incidence of coronary artery aneurysms.

Conclusion

Bacterial, viral, and allergic conjunctivitis are usually benign causes of the red eye that present with a variable discharge, redness, and, in some cases, itching or mild irritation. The presence of significant pain with or without impairment of visual acuity should alert the emergency physician to the presence of a more serious condition. The character of the pupil and the cornea and the presence or absence of any lesions on fluorescein staining will further help to delineate the etiology. Corneal ulcer, uveitis, and HSV keratitis, among other conditions, need to be referred to an ophthalmologist for definitive care. (See Table 5 on page 18.)

In the pediatric population, ophthalmia neonatorum and HSV infections need a full septic workup with admission for IV antibiotics or acyclovir. With the exception of antibiotic drops for bacterial conjunctivitis (and possibly systemic antibiotics for young children with this disease), there are few extensive clinical trials to support what is considered standard therapy for most of these conditions.

The Swollen Eye

Infections, inflammatory processes, or tumors may all present as a swollen or protuberant eye. As the treatment and outcome are different with each etiology, it is important for the emergency physician to narrow the diagnostic possibilities. Certain conditions are more prevalent in certain age groups. (Intraocular neoplasms, for instance, peak before the age of 10.) Immunosuppressed patients are at risk for certain fungal diseases. In addition to a thorough eye exam, a working knowledge of the anatomy of the orbit, including the extraocular muscles, is essential to arrive at the correct diagnosis. The CT scan is the test of choice for evaluating the swollen eye.

Bacterial Infections

The orbit is separated from the eyelids by a fascial layer that attaches to the periosteum of the orbital bones (the orbital septum; see Figure 2 on page 18). Periorbital cellulitis (POC) (also known as pre-septal cellulitis) is defined as infection anterior to the orbital septum (i.e., involving the tissue of the lids). In contrast, orbital cellulitis (OC or post-septal cellulitis) involves the orbit (and frequently tissues on both sides of the orbital septum). Both POC and OC will present

Table 4. Therapy For Neonatal Conjunctivitis.

<table>
<thead>
<tr>
<th>Disease</th>
<th>Medication</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chlamydia trachomatis</td>
<td>Erythromycin 50 mg/kg/d PO divided QID for 14 days</td>
<td></td>
</tr>
<tr>
<td>Neisseria gonorrhoeae</td>
<td>Ceftriaxone 25-50 mg/kg IV or IM in a single dose, not to exceed 125 mg*</td>
<td>Frequent saline eye irrigation; ophthalmologic consult</td>
</tr>
<tr>
<td>Herpes simplex virus</td>
<td>Acyclovir 30-60 mg/kg/d IV divided q8h for 14-21 days and 1% to 2% trifluridine, 1% iododeoxyuridine, or 3% vidarabine, topically</td>
<td>Ophthalmologic consult</td>
</tr>
</tbody>
</table>

* Other acceptable regimens can be found in the 2000 Red Book: Report of the Committee on Infectious Diseases (see sources).

with significant pain, a tender and erythematous unilateral swelling around the eye, conjunctival injection, and chemosis. In POC, patients demonstrate full and painless extraocular motions. Any impairment of extraocular movements or pain on EOM testing should alert the emergency physician to the possibility of OC.

In adults, the majority of cases of OC result from contiguous spread of sinusitis, primarily the ethmoid sinuses to the orbit. The initial pathogens are *Staphylococcus aureus*, *Streptococcus pneumoniae*, and *Haemophilus influenzae*; however, anaerobic bacteria soon predominate.93 Other sources of OC are maxillary dental abscesses.94

A CT scan is indicated whenever there is suspicion that the infection has spread beyond the septum, if the diagnosis is unclear, or if the differential diagnosis includes non-infectious etiologies. It will delineate the extent of orbital infection, presence of abscess collections, sinusitis, and orbital osteomyelitis.

Treatment requires antibiotics covering *Staphylococcus* and *Streptococcus* species as well as anaerobic organisms. There are no controlled outcome studies. General recommendations include ampicillin/sulbactam or a third-generation cephalosporin plus clindamycin. An orbital abscess, osteomyelitis, or sinusitis usually requires adjunctive surgical drainage.93 Complications of OC include cavernous sinus thrombosis, subdural empyema, and meningitis or cerebritis.

**Fungal Infections**

Diabetics, patients with hematologic malignancies, those treated with immunosuppressive drugs, and generally debilitated patients are at risk for mucormycosis. Fungal sinusitis develops in patients with poor phagocytic function and spreads contiguously to the orbit.

Fungal OC can be clinically indistinguishable from bacterial disease. It also tends to be unilateral. The most common clinical presentation is proptosis, decreased vision, chemosis, ophthalmoplegia, and trigeminal anesthesia. Treatment is with intravenous amphotericin B and surgical debridement.93

Immunocompetent adults with chronic sinusitis can develop a syndrome of allergic fungal sinusitis. Seven percent of patients have some orbital involvement. Treatment is surgical drainage of the sinuses and oral antifungals.93

**Inflammatory Conditions**

Idiopathic orbital inflammatory syndrome (formerly called orbital pseudotumor) is thought to be an autoimmune disorder (10% of cases are associated with diabetes, asthma, rheumatoid arthritis, systemic lupus erythematosus, or Crohn’s disease). Its presentation may be indistinguishable from OC; however, a CT scan may show a diffuse infiltrate that enhances with contrast, as opposed to an abscess with or without sinusitis. Once systemic diseases such as sarcoidosis, Wegener’s granulomatosis, tuberculosis, or syphilis are ruled out, treatment is usually begun with corticosteroids.95 There are no large clinical trials; most evidence is based on case series.

Thyroid-related ophthalmopathy (TRO) is diagnosed in 22% of patients before systemic hyperthyroidism develops.95 White females between 30 and 50 predominate, with a female-to-male ratio of 6:1. TRO is generally bilateral and gradual in onset, and usually it is not painful. The eyes are generally proptotic due to collagen deposition, increased retrobulbar fat, and orbital inflammation. A classic finding

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**Table 5. Key Points In Managing The Red Eye.**

1. **Important historical data**
   - Onset—gradual vs. rapid
   - Type of discharge—clear, mucoid, or purulent
   - Pain—presence or absence
   - Itching
   - Changes in vision
   - Past medical history—diabetes, immunocompromise, sexually transmitted diseases
   - Current medications

2. **Important physical findings**
   - Condition of the eyelids—crusting, discharge, swelling
   - Visual acuities—normal or decreased
   - Location of redness—focal or diffuse
   - Cornea—clear or hazy
   - Anterior chamber—cells and flare, or visible hypopyon
   - Pupil—normal, constricted, or dilated
   - Fluorescein staining—ulcer, punctate, dendrites

3. **Immediate referral to an ophthalmologist**
   - Lack of response to presumed bacterial conjunctivitis
   - Hyperacute (gonococcal) bacterial conjunctivitis
   - Keratitis due to HSV infection
   - Acute angle-closure glaucoma
   - Scleritis
   - Uveitis
   - Pediatrics (ophthalmia neonatorum, HSV neonatal conjunctivitis)
(present in 92% of patients) is upper and lower lid retraction. On downward gaze, the upper lid does not follow the eyeball (lid lag). The autoimmune process also affects the extraocular muscles, and patients may present with extraocular motor paresis. TRO is considered an ophthalmologic emergency, as 22% of patients develop severe vision loss if untreated. Treatment is begun with high-dose oral steroids. Radiation therapy and surgical decompression are reserved for non-responders.

**Tumors**

Both metastatic and primary orbital tumors generally present unilaterally, after a gradual onset, and with mild pain. There may be significant proptosis associated with EOM dysfunction and significant visual loss. Primary tumors tend to peak in childhood years, whereas metastatic disease follows the general pattern of increasing incidence of malignancies with aging. A CT scan of the orbit and consultation are essential.

**Pediatric Considerations Periorbital Cellulitis/Orbital Cellulitis**

The availability and widespread use of the *H. influenzae* type B (HIB) vaccine between 1985 and 1990 has had a profound effect on the microbiology, virulence, and treatment of POC/OC. Standard management prior to 1990 included aggressive evaluation with blood cultures and LP followed by admission for IV antibiotics. Such aggressive interventions are rare in the current era. Today, the pathogenesis and management of POC/OC more closely resembles adult disease, with organisms associated with sinusitis predominating. If the sinusitis is acute, *S. pneumoniae*, nontypeable *H. influenzae*, and *Moraxella catarrhalis* are most common. With chronic sinusitis, *S. aureus* and anaerobes must also be considered. If there is evidence of local trauma resulting in a break in the dermis, skin flora are often causative agents. Spread via the bloodstream may still occur in children younger than 24 months of age, who are at risk for *S. pneumoniae* bacteremia. In older children with bacteremia, Group A *Streptococcus* predominates. It is important to differentiate soft-tissue swelling due to an acute allergic reaction from POC. Children may also present with erythema and edema of either eyelid secondary to an acute allergic reaction. In these instances, the child is generally afebrile, looks well, and may complain of itching in the area. In contrast to the violaceous color seen in infectious causes, the erythema in allergic edema is less prominent, and the edema has a lighter, watery type of appearance. A trial dose of an oral antihistamine in the ED may confirm allergic erythema if the symptoms resolve after an hour of observation.

Children with POC/OC often present the day symptoms begin. There may be marked erythema, edema, fever, proptosis, ophthalmoplegia, or pain with EOM motion.

Although the evidence comes exclusively from retrospective chart reviews (the incidence of POC/OC is probably too low for any successful prospective trial), a less aggressive approach is likely safe in the HIB-vaccine era. Low-risk patients are defined as nontoxic, older than 12 months, and without meningeal or focal neurologic findings, vision loss, limitation of eye motion, eye malformation, or operation in the vicinity of the infection. These children can be evaluated with blood culture and CBC (what the CBC tells us is unclear, but it remains an accepted ritual), without LP. Low-risk patients can be considered for outpatient therapy; however, the parents should also be willing and able to return at any sign of worsening. Daily follow-up should be ensured. The American Academy of Pediatrics issued a position statement that “mild cases of periorbital cellulitis (eyelid < 50% closed) may be treated with appropriate oral antibiotic therapy as an outpatient with daily patient encounters.”

As with adults, CT of the orbits and cranium is very useful in defining the extent of the infection, sinusitis, or abscess collection. A CT should be ordered on the suspicion that the infection extends beyond the orbital septum. *Any orbital involvement mandates consultation with a surgical specialist (ophthalmologist, ENT surgeon).* Parenteral antibiotics are standard for inpatient therapy and are often administered as the first dose of an outpatient regimen. There are no prospective trials comparing the different regimens. Initial treatment is based on an estimation of the microbes involved and several clinical factors: the severity of the process, the toxicity of the child, and the suspicion of intracranial disease.

Inpatient regimens for OC/POC consist of:

- Ceftriaxone (100 mg/kg/d in 2 divided doses) or
- Ampicillin-sulbactam (200 mg/kg/d in 4 divided doses) plus
- Vancomycin (60 mg/kg/d in 4 divided doses) if infection is either known or likely to be caused by highly resistant *Streptococcus pneumoniae*.

If intracranial disease is suspected or known, consult with a pediatric infectious disease specialist and a surgeon, and consider adding metronidazole 15-35 mg/kg/d divided q8h (maximum dose, 1-2 g/d) and nafcillin/vancomycin to the third-generation cephalosporin. The outpatient regimens for OC/POC are:

- Ceftriaxone 50 mg/kg IM or IV (maximum, 1 g) followed by *either* amoxicillin/clavulanate 45 mg/kg/d divided twice daily PO for at least 10 days or
- Azithromycin 12 mg/kg (maximum, 500 mg/d) for five days in the penicillin-allergic child.

When sinusitis is present or suspected, treatment duration should be extended to reflect accepted courses for this entity.

**Dacryocystitis**

Dacryocystitis (inflammation or infection of the nasolacrimal duct) may be acute or chronic. (See Figure 3 on page 20.) Dacryocystitis occurs in children with congenital or acquired nasolacrimal duct obstruction. Congenital nasolacrimal duct obstruction (CNLDO) may occur in up to 70% of healthy newborns. It presents as eye watering, crusting, and mucoid discharge without conjunctival injection in a newborn that is otherwise well. As most cases will resolve spontaneously by 1 year of age, conservative
management is generally the rule. Parents should massage the lacrimal ducts several times a day. The duct is “milked” by placing an clean index finger over the common canalicus, thus preventing material from exiting from the lacrimal punctum, and then applying gentle pressure in a downward motion. Massage improves the chance for early resolution. Acquired nasolacrimal obstruction is usually the result of trauma to the midface or orbit. Simple obstruction needs to be differentiated from both chronic inflammation and acute infection.

Acute dacryocystitis (AD) also presents with eye discharge and crusting, but it is associated with lacrimal sac or eyelid erythema and edema. It can occur as a complication of chronic dacryocystitis (CD) in neonates and older infants and in older children in association with facial trauma that disrupts the nasolacrimal duct. Some authorities believe neonates require a full sepsis evaluation, including CBC, blood and urine cultures, and LP, due to their relative immunosuppression. Such children should be covered with antibiotics to cover Staphylococcus and Streptococcus species, with early probing of the duct by the specialist. In older infants, an LP is not necessary unless the child is ill-appearing or there are signs of meningitis. All cases of acute dacryocystitis, no matter what the age, are treated with intravenous antibiotics to cover Staphylococcus and Streptococcus species, with early probing of the duct by the consultant. In the case of fracture-related AD, a stent may need to be placed in the duct. POC/OC is a recognized complication of AD.

Suggested antibiotic regimens include:
- Cefotaxime 200 mg/kg/d, divided q8h plus nafcillin 100-200 mg/kg/d, divided q6h plus erythromycin ophthalmic ointment
- Ampicillin/sulbactam (Unasyn) 100-200 mg/kg/d divided q6h (maximum, 6-12 g/d)

Chronic dacryocystitis is a low-grade inflammation of the lacrimal sac in the setting of nasolacrimal obstruction. It can be difficult to differentiate from CNLDO. In CD, there is a mucopurulent discharge from the lacrimal punctum and crusting of the lashes, but with the absence of erythema in either the lacrimal sac or the eyelid. Treatment consists of topical ophthalmic antibiotics and elective nasolacrimal duct probing by an ophthalmologist.

Tumors
Tumors that originate in the eye are far more common in childhood than metastatic disease. They generally present with vision loss and/or proptosis. These tumors may be either benign or malignant. Clues to the presence of tumor include proptosis, absence of light reflex, and decreased visual acuity. CT scan and appropriate referral are crucial.

Conclusion
Infections, most often the sequelae of acute or chronic sinusitis, are the most common causes of the unilateral swollen eye. The emergency physician should be aware of other etiologies, such as tumor or inflammation, the latter especially if the proptosis is bilateral. Current therapy is supported largely by small case series or retrospective reviews.

Acute Vision Loss
In general, patients presenting to the ED with acute vision loss should be referred quickly to an ophthalmologist. In truly emergent conditions, minutes may count in preserving vision. This section will not cover neurologic causes of vision loss, such as aneurysms, TIAs (except to the retinal artery), and strokes, nor does it address diseases of the optic nerve beyond the orbit (optic chiasm, optic radiation, or visual cortex).

History
Dividing patients by age and type of visual loss is the first step in the approach to acute vision loss. Partial loss or complaints of flashing lights or “floaters” often denote retinal detachment. Non-traumatic vision loss under the age of 50 is almost always due to optic neuritis (ON). Over the age of 60, vascular causes predominate, with temporal arteritis rarely occurring below 65 years of age.

Besides the age of the patient and the extent of visual impairment, it is important to ask about perception of color, central vs. peripheral vision loss, current medications, and the presence of underlying medical conditions. Determine how quickly the visual loss occurred and whether the patient has pain.

Physical Examination
The examination should be systematic and is best approached in the following order.

1. Visual Acuity And The Pinhole Test
   If visual acuity improves with looking through a pinhole, the etiology of visual loss is likely to be optical (uncorrected refractive error, lens or corneal opacity, or vitreous disease) and not due to diseases of the CNS or the eye.

2. Swinging Flashlight Test
   Relative afferent papillary defect (RAPD), formerly referred to as the Marcus Gunn pupil sign, is detected by the swinging flashlight test. This is performed by brisk alternat-
ing stimulation of the eyes with a strong light source. The normal response is constriction of the pupil when light is shown directly in the eye. Paradoxical dilatation to direct light is a positive RAPD test. The abnormal pupil will initially dilate to direct light, but then constrict when the light is shone in the opposite eye. This greater response to consensual vs. direct stimulation establishes an afferent defect in the eye with the positive test. This strongly suggests disease of the optic nerve.

3. Evaluation Of Pupil Size, Shape, And Response To Light And Accommodation
The size of the pupil depends on a balance between sympathetic and parasympathetic tone. The parasympathetic fibers travel in the oculomotor (III) nerve to the ciliary ganglion. Postganglionic axons innervate the pupillary constrictor muscle. The sympathetic fibers exit the spinal ganglion. Postganglionic fibers then ascend via the carotid plexuses and the nasociliary nerve to the superior cervical ganglion. Postganglionic fibers then ascend via the carotid plexuses and the nasociliary nerve to innervate the pupillary dilator muscle. Significant pupil findings include:

- Adie’s tonic pupil: a dilated pupil that reacts poorly to light, but better to accommodation. It is usually unilateral. The defect is usually localized to the third cranial nerve ciliary ganglion and is usually due to a viral infection or inflammatory process.
- Argyll-Robertson pupil: a small, irregular pupil associated with CNS syphilis.
- Horner’s syndrome: a miotic pupil that reacts to light. It is the result of sympathetic denervation of the pupil. Causes include apical lung tumors, trauma to the spinal cord, lateral brainstem vascular lesions, or syringomyelia. The pupil will usually dilate to instillation of topical mydriatics. Patients may demonstrate ptosis of the involved eye and inability to sweat on the side of their face.

4. Visual Fields
Evaluating visual fields by direct confrontation is important in ON. Patients with this condition may have central scotoma with sparing of the periphery. Abnormalities of visual fields may also detect optic chiasm or associated neurologic disease. For instance, a pituitary tumor compressing the optic chiasm may present with a bitemporal hemianopia, whereas a large hemispheric stroke may result in a homonymous hemianopsia.

5. Evaluation Of The Extraocular Muscles
Have patients hold their head still and use their eyes to follow the examiner’s finger up and down and side to side. The third cranial nerve innervates the medial, superior, and inferior rectus muscles as well as the inferior oblique and levator palpebri muscles. The parasympathetic fibers also run with the third nerve. An eye with a third nerve palsy will be deviated down and out due to unopposed actions of the IVth and VIth nerves and will not move up or medially. The fourth cranial nerve innervates the inferior oblique muscle. An eye with a fourth nerve palsy will be externally rotated at rest (extorted). The sixth cranial nerve innervates the lateral rectus muscle. An eye with a sixth nerve palsy will be slightly adducted at rest and unable to abduct.

6. FunduscopY
In early central retinal artery occlusion, funduscopy may reveal the classic “box car” pattern of stagnant blood in the arteries or veins. A pale retina with a spared fovea (the cherry red spot) is pathognomonic of late retinal artery occlusion. Venous congestion and hemorrhage may be present in central retinal vein occlusion. Optic disc edema or congestion may be evident in ON and pseudotumor cerebri.

7. Tests For Malingering
Emergency physicians are occasionally faced with patients who claim sudden bilateral blindness. While such events occur, they are quite rare, and several simple tests performed without sophisticated lenses or prisms can substantiate the impression of malingering. The first step is to know how the truly blind eye responds to certain maneuvers. Patients who are bilaterally blind cannot make eye contact, nor will they have spontaneous accommodation and convergence. However, when asked to look at their own finger, malingers will demonstrate accommodation and convergence. A sudden bright light should not cause any blinking or flinching. A menacing action, likewise, should cause no response, although throwing a punch toward the face can cause blinking if the air from the force of the blow hits the patient’s cornea. (Besides, just in case the patient can see, don’t be too menacing.)

Unless the etiology of the blindness is cortical or subcortical, the pupils will be moderately dilated and unreactive to light. (However, the patient with cortical blindness will still have a pupillary response.)

With unilateral blindness, there should be a positive RAPD test in the affected eye (unless the lesion is cortical or subcortical). The truly blind eye will deviate first when following an object. If the “good” eye is suddenly covered, the “blind” eye should not continue to follow the object. Moving a mirror in front of the “blind” eye should not result in any movement of the eye. Having a patient stare straight ahead while passing a piece of paper with wide stripes in front of the eyes will produce involuntary nystagmus in a patient with intact vision. (Cardiac monitor paper with alternating blackened wide blocks works well for this.)

Some patients may even place anticholinergic preparations in their eye to create a “blown pupil.” Unlike a neurologically dilated pupil, the pharmacologic-blockaded eye will not constrict when 1% pilocarpine is placed in the eye. If there is no structural abnormality of the iris on slit-lamp examination and if the IOP is normal, then it is likely that the dilated pupil is pharmacologically induced. Only a few cases of an acute Adie’s pupil or traumatic iritis will produce the same result.

Retinal Detachment
Retinal detachment may present as sudden onset of light flashes or floaters, or with the classic description of a “shade coming down.” It is most commonly idiopathic,
but it may be associated with inflammation, trauma, surgery, or infections in immunocompromised patients. If the detachment involves the macula, visual loss will be severe. Many detachments are peripheral and thus difficult to appreciate on direct fundoscopy of the undilated eye (indirect ophthalmoscopy being more accurate in diagnosis). Using a short-acting miotic such as tropicamide (Mydriasil) may aid in visualization of the fundus. If visualized, the detachment will appear as an elevated gray area. All retinal detachments should be referred to an ophthalmologist for treatment.

“All seems infected that the infected spy / As all looks yellow to the jaundic’d eye.”—Alexander Pope (1688 - 1744), English poet, “An Essay on Criticism.”

Double Vision
Double vision (diplopia) may be monocular or binocular. A prospective survey of all patients presenting to an ophthalmologic ED (in the United Kingdom) over a nine-month period found that monocular diplopia represented about 20% of cases. Monocular diplopia is almost always due to abnormalities in the eyeball itself or to problems with contact lenses or bifocal glasses. In contrast, binocular diplopia is due to dysfunction of the extraocular muscles. Most cases of binocular diplopia are due to abnormalities of the cranial nerves (from systemic illnesses such as diabetes or vasculitis), extraocular muscle pathology (either congenital or acquired), or trauma. The most frequent traumatic cause is a “blow-out” fracture of the orbit with subsequent entrapment of the inferior rectus muscle. A small percentage of patients develop diplopia as a result of retro-orbital tumors or inflammatory processes. Other rare causes include from supra-nuclear palsy, brainstem ischemia, and pituitary tumors.

Acute Vision Loss Under The Age Of 50
Optic neuritis is almost exclusively a disease affecting individuals between 15 and 45, with a significant preponderance of white females. It has been reported in children; however, less than 1% of cases occur in individuals over 50 years of age. The etiology is as yet undetermined. Significant visual improvement will occur in the first 4-6 weeks, with 95% of patients having visual acuities of 20/40 or better. The amount of visual loss varies from mild to severe. Many patients also complain of alterations in color vision. Almost all patients complain of periorbital pain that is exacerbated by extraocular movements. In the majority of patients, the disease is retrobulbar, so that the disc may appear normal (the origin of the old saw regarding ON—the patient doesn’t see anything and the doctor doesn’t see anything). In the third of patients who have anterior ON, the disc may appear edematous. The diagnosis is largely clinical, relying on the presence of decreased vision (either central or peripheral) and an afferent pupillary defect. Routine lab tests to screen for etiologies other than ON, in the absence of clinical findings, are of very little use.

There is an association between isolated ON and multiple sclerosis (MS). ON can be the initial presentation of MS in 20% of cases and will affect 50% of patients who have MS sometime during their lifetime. IV methylprednisolone (250 mg IV QID x 3 days) improves short-term visual outcome and may slow the progression of MS over the subsequent two years. Consultation with an ophthalmologist or neurologist should be done prior to initiating treatment.

Other Causes
There are certain clinical findings not characteristic of ON that should prompt a further workup: visual function that worsens after two weeks or shows no improvement after six weeks, bilateral ON (collagen vascular disease or vasculitis), severe optic disc swelling or hemorrhage (possible external compression of the optic nerve), bitemporal hemianopsia (pituitary masses), and age less than 18. Possible causes include:

- Infections: Cat-scratch disease, Lyme disease, syphilis, toxoplasmosis, toxocariasis, and histoplasmosis can present with painless visual loss and optic disc edema.
- Sarcoidosis generally affects the anterior chamber (uveitis); however, the optic nerve is the second most affected cranial nerve after the VIIth nerve. Ninety percent of patients will have abnormalities on chest radiography or CT scan.
- Acute methanol intoxication: Accidental or intentional overdose with methanol presents with visual disturbances often described as “being in a snow storm.” The visual complaints, abdominal pain, and accompanying severe anion and osmolar gap acidosis should suggest the diagnosis. Treatment involves bicarbonate and fomepizole, followed by dialysis to remove the formic acid that forms the basis for methanol toxicity.

Vision Loss Over The Age Of 60
Central Retinal Artery Occlusion
Central retinal artery occlusion (CRAO) may be caused by atherosclerotic obstruction, vasospasm, embolism, or systemic hypotension. It is associated with systemic diseases such as hypertension, diabetes, atherosclerosis, vasculitis, hypercoagulable states, and migraines.

The classic presentation is sudden, painless, and severe visual loss. The classic fundoscopic finding is a pale, edematous retina with a “cherry red spot” representing the unaffected choroidal vascular bed in the ischemic fovea. When the cherry red spot is present, it implies ischemia of three hours or longer.

The general prognosis is poor; however, there are case reports of return of vision even after 72 hours of arterial occlusion. Interventions of low and unproven efficacy are generally recommended for the emergency physician more on the basis of “something is better than nothing.” Interventions include placing the patient supine and performing ocular massage. To do this, apply pressure five seconds on then five seconds off, for 15-30 minutes. Because carbon dioxide dilates cerebral vasculature (and could allow an arterial clot to “move downstream”), have the patient rebreathe CO2. If a mixture of 95% O2 5% CO2 is unavailable,
a paper bag may be used. Intravenous acetazolamide (500 mg) may lower IOP. Paracentesis by an ophthalmologist is sometimes attempted, although it has a high complication rate and must be repeated every two hours to maintain a low IOP. It is not recommended in recent literature or textbooks.

Recently, intra-arterial urokinase has been investigated. However, a recent meta-analysis has shown only a modest improvement with this aggressive approach (27% vs. 18%-21% with conservative treatment).118

Central Retinal Vein Occlusion
Central retinal vein occlusion (CRVO) presents with either abrupt or gradual decrease in vision. The pathogenesis of CRVO is generally unknown, although in older individuals it is associated with hypertension, atherosclerosis, diabetes, cardiovascular disease, and hyperlipidemia.

Visual acuity may range from mild impairment to light perception. Funduscopic examination early in the course will usually reveal retinal hemorrhages, dilated veins, and a swollen optic disc.

There is no generally effective medical therapy. Certain subsets of patients (those with diabetes or glaucoma) are candidates for specific laser techniques. Surgery and rTPA are still considered experimental.119 Prognosis for recovery is directly related to visual acuity at presentation: 65% of patients with 20/40 maintained that vision at three-year follow-up, whereas 80% of patients with 20/200 vision remained at that level.120

Temporal Arteritis
Temporal arteritis (TA) is a granulomatous inflammation of extracranial arteries that may lead to rapid or sudden visual loss either by ischemic optic neuropathy (90% of cases) or acute central retinal artery occlusion. It is a disease of the elderly and rarely occurs before the age of 65. The majority of cases occur in Caucasians, especially those of Scandinavian descent. The role of the emergency physician is the identification of the patient with TA before visual loss occurs, because when it does, it is often rapid and permanent. TA may present in association with polymyalgia rheumatica, a complex of anemia, elevated erythrocyte sedimentation rate (ESR), proximal joint pain, fatigue, low-grade fever, and weight loss.107

TA should be considered in any elderly patient who presents with a headache and temporal tenderness. Jaw claudication (pain in the masseter muscles with chewing) and tender or indurated temporal arteries may also provide clues to the diagnosis. The ESR is almost always elevated, often greater than 100. Although there are isolated cases of TA with a low or normal ESR, this is so rare that an alternative diagnosis should be sought.120,121 Temporal artery biopsy is the gold standard for diagnosis; however, if the patient has visual symptoms on presentation, IV methylprednisolone is recommended while awaiting the biopsy results.120

Pediatric Considerations

Infants
Parents, especially with infants or pre-verbal children, are often the ones who note an abnormality with the eye or with vision. Congenital cataracts and glaucoma are usually discovered in the newborn nursery. Cataracts are detected as an absent red reflex, or leukocoria, when an ophthalmoscope is shone directly on the pupil. All cataracts should be urgently referred to an ophthalmologist, as the treatment of choice is surgical. Once the cataract is removed, the ultimate prognosis is closely related to compliance with amblyopia therapy.122

Congenital glaucoma (or infantile glaucoma), although rare (less than 1/10,000 live births), is a significant cause of childhood blindness.122 Infantile glaucoma should be suspected when excessive tearing and photophobia accompany an increase in corneal size or an enlarged globe. Clouding of the cornea may also be evident. The treatment of congenital glaucoma is primarily surgical, and ophthalmologic referral should be rapid whenever the diagnosis is suspected.

Children
Amblyopia (or abnormal vision in a structurally normal eye) occurs before the age of 10. It develops when an unclear image falls on the retina and is then transmitted to the immature visual cortex. Amblyopia may result from strabismus (lazy eye) or from external obstruction to vision (capillary hemangioma of the eyelid). It is diagnosed when there is a large discrepancy in visual acuity between the two eyes. Amblyopia should be referred to an ophthalmologist for outpatient management.

Adolescents
ON is an uncommon cause of vision loss in children. In children, ON frequently presents with bilateral sudden vision loss occurring after a recent viral illness.123 ON has been associated with measles, mumps, chickenpox, pertussis, EBV infections, immunizations,124 and Lyme disease.123 Retrospective studies by Morales et al and Brady et al provide conflicting data on visual prognosis. However, there appears to be a significant risk of permanent and considerably diminished vision (22% of patients in Brady et al and 29% in Morales et al).125,126 Children are less likely to develop MS than adults.123 Unilateral disease has a better visual prognosis and an increased risk of subsequent MS vs. bilateral disease.

There are no large, randomized, controlled studies on its management. Current practice is based mainly on the Optic Neuritis Treatment Trial (ONTT), a multicenter, prospective, randomized trial that did not include pediatric patients.123 Intravenous steroids and a slow taper are recommended to treat ON (again, based exclusively on adult clinical trials).124

Conclusion
The causes of acute vision loss are strongly tied to the age of the patient. In the neonate, congenital cataracts, glaucoma, and tumors are most frequent. In the child, refractive errors, amblyopia, and tumors predominate. In the older child or adolescent, the emergency physician must also consider the diagnosis of optic neuritis. ON is the overwhelming cause of
acute vision loss in patients under 50 years of age, while vascular disease is the main culprit in patients over 60. The emergency physician should also consider temporal arteritis in the geriatric population, as rapid institution of therapy can prevent further loss of vision.

“The next best thing to being clever is being able to quote someone who is.”
—Mary Pettitbone Poole, “A Glass Eye at a Keyhole,” 1938.

General Conclusion

With a little detective work, a thorough exam, and an understanding of the incidence and presentation of the most common and most serious disorders, the ED physician should be well-prepared to deal with non-traumatic eye emergencies. We must recognize when urgent consultation with an ophthalmologist is needed and when it can safely be delayed. ▲

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study, will be included in bold type following the reference, where available. In addition, the most informative references cited in the paper, as determined by the authors, will be noted by an asterisk (*) next to the number of the reference.


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Physician CME Questions

81. **Blepharitis is:**
   a. an inflammation of the eyelids.
   b. an inflammation of the cornea.
   c. an inflammation of the subconjunctival connective tissue and the blood vessels that course between the sclera and conjunctiva.
   d. an inflammation of the iris and ciliary body (also called iritis or iridocyclitis).
   e. an inflammation of the choroid.

82. **Keratitis is:**
   a. an inflammation of the eyelids.
   b. an inflammation of the cornea.
   c. an inflammation of the subconjunctival connective tissue and the blood vessels that course between the sclera and conjunctiva.
   d. an inflammation of the iris and ciliary body (also called iritis or iridocyclitis).
   e. an inflammation of the choroid.

83. **Episcleritis is:**
   a. an inflammation of the eyelids.
   b. an inflammation of the cornea.
   c. an inflammation of the subconjunctival connective tissue and the blood vessels that course between the sclera and conjunctiva.
   d. an inflammation of the iris and ciliary body (also called iritis or iridocyclitis).
   e. an inflammation of the choroid.

84. **Anterior uveitis is:**
   a. an inflammation of the eyelids.
   b. an inflammation of the cornea.
   c. an inflammation of the subconjunctival connective tissue and the blood vessels that course between the sclera and conjunctiva.
   d. an inflammation of the iris and ciliary body (also called iritis or iridocyclitis).
   e. an inflammation of the choroid.

85. **Posterior uveitis is:**
   a. an inflammation of the eyelids.
   b. an inflammation of the cornea.
   c. an inflammation of the subconjunctival connective tissue and the blood vessels that course between the sclera and conjunctiva.
   d. an inflammation of the iris and ciliary body (also called iritis or iridocyclitis).
   e. an inflammation of the choroid.

86. **Visual acuity:**
   a. is the “vital sign” of the eye.
   b. cannot be assessed in children.
   c. should be measured in both eyes at once.
   d. cannot be measured in patients without glasses.

87. **Normal intraocular pressure:**
   a. is less than 12 mmHg.
   b. ranges between 12 and 21 mmHg.
   c. ranges between 21 and 30 mmHg.
   d. is greater than 30 mmHg.

88. **Fluorescein staining that reveals a superficial criss-cross of mostly vertical lines suggests:**
   a. herpes infection.
   b. corneal abrasion.
   c. a foreign body under the upper lid.
   d. a corneal ulcer.

89. **The slit lamp examination:**
   a. should be performed in all patients who present with ocular complaints.
   b. can be reserved for patients in whom iritis or small foreign bodies are suspected.
   c. can substitute for measuring a patient’s visual acuity.
   d. should not be performed in children.

90. **All of the following about bacterial conjunctivitis are true except:**
   a. It can cause deep orbital pain and vision loss.
   b. Patients report a rapid onset of irritation, redness, and a purulent discharge.
   c. It is generally a benign and self-limited condition.
   d. Broad-spectrum topical antibiotics can shorten the duration of illness.

91. **Treatment of blepharitis involves:**
   a. a daily regimen of lid hygiene (warm compresses, massage, cleaning the lids).
   b. erythromycin ophthalmic ointment when staphylococcal blepharitis is suspected.
   c. a one- to three-week course of topical steroids for resistant and severe inflammation.
   d. counseling the patient about the likelihood of recurrent flare-ups and the need for a regular regimen of lid hygiene.
   e. all of the above.

92. **Which of the following concerning eye patches for corneal abrasions is true?**
   a. They should only be used in contact lens wearers.
   b. They should be used for all corneal abrasions.
   c. They should only be used in patients with small-to moderate-sized abrasions.
   d. A recent meta-analysis has shown that they do not promote healing, nor do they help to control pain.
   e. All of the above.

93. **Which of the following conditions can threaten vision and should be referred to an ophthalmologist?**
   a. Corneal ulcers
   b. Acute anterior uveitis
   c. Ocular herpes infections
   d. Acute angle-closure glaucoma
   e. All of the above

94. **Which of the following may present as a swollen eye?**
   a. Bacterial or fungal infections
   b. Inflammatory conditions such as idiopathic orbital inflammatory syndrome or thyroid-related ophthalmopathy
   c. Tumors
   d. Periorbital cellulitis and orbital cellulitis
   e. All of the above
95. Acute, non-traumatic vision loss in adults under the age of 50 is most likely due to:
   a. optic neuritis.
   b. temporal arteritis.
   c. central retinal artery occlusion.
   d. glaucoma.

96. Acute, non-traumatic vision loss in patients over the age of 60 is most likely due to:
   a. vascular causes.
   b. infections.
   c. optic neuritis.
   d. acute methanol intoxication.

This test concludes the January through June 2002 semester testing period of Emergency Medicine Practice. The answer form for this semester and a return envelope have been included with this issue. All paid subscribers are eligible to take this test. Please refer to the instructions printed on the answer form.

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Each action in the clinical pathways section of Emergency Medicine Practice receives an alpha-numerical score based on the following definitions:

Class I
• Always acceptable, safe
• Definitely useful
• Proven in both efficacy and effectiveness

Level of Evidence:
• One or more large prospective studies are present (with rare exceptions)
• High-quality meta-analyses
• Study results consistently positive and compelling

Class II
• Safe, acceptable
• Probably useful

Level of Evidence:
• Generally higher levels of evidence
• Non-randomized or retrospective studies: historic, cohort, or case-control studies
• Less robust RCTs
• Results consistently positive

Class III
• May be acceptable
• Possibly useful
• Considered optional or alternative treatments

Level of Evidence:
• Generally lower or intermediate levels of evidence
• Case series, animal studies, consensus panels
• Occasionally positive results

Indeterminate
• Continuing area of research
• No recommendations until further research

Level of Evidence:
• Evidence not available
• Higher studies in progress
• Results inconsistent, contradictory
• Results not compelling

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Target Audience: This enduring material is designed for emergency medicine physicians.

Needs Assessment: The need for this educational activity was determined by a survey of medical staff, including the editorial board of this publication; review of morbidity and mortality data from the CDC, AHA, NCHS, and ACEP; and evaluation of prior activities for emergency physicians.

Date of Original Release: This issue of Emergency Medicine Practice was published June 1, 2002. This activity is eligible for CME credit through June 1, 2005, the latest review of this material was May 7, 2002.

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