Vision is arguably the most precious of the five senses. As such, the utmost caution must be used when treating athletes who have eye complaints. The training room is often the first site of patient contact, before the emergency department or the ophthalmologist, so having the proper equipment is crucial to make an accurate diagnosis and begin appropriate treatment. There are numerous eye conditions that can affect the athlete both on and off the field. Infections and trauma are the most common sources of eye complaints that affect the athlete’s performance on game day and beyond; however, other ocular complaints may be manifestations of a variety of systemic conditions. This can make for a challenging diagnosis, so a thorough review of the eye and its disorders is essential.

**ESSENTIAL EQUIPMENT**

The equipment available to the examiner will vary depending on location. Sideline management of eye conditions should be geared toward simple triage and protection from further injury. The training room should have all of the tools necessary to make roughly 90% of the diagnoses that come off the field of play. A slit lamp may be required to find additional problems, but is a piece of equipment that is not always readily available. A good “eye cart” should include at least the following: ophthalmoscope, pen light with blue filter, cotton-tipped swabs, a vision chart, topical anesthetic drops, mydriatic drops (for papillary dilation), fluorescein dye, antibiotic ointment, eye shields, sterile saline solution for flushing eyes and contact lenses, magnifying glasses, 18-gauge needles, fine-tipped pick-ups, a blue cobalt light, and a Tonopen (Medtronic, Minneapolis, Minnesota).

**HISTORY**

The evaluation should always begin with a thorough history of the onset of symptoms or mechanism of injury. Direct trauma to the orbit should always

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alert the physician or trainer to possible extra-ocular injuries. Atraumatic visual complaints should be evaluated as rapidly as traumatic ones. Subtle eye histories such as moderate to severe myopia can be pertinent, because this common refractive error predisposes patients to a higher risk of retinal detachments. Athletes who have a history of glaucoma, diabetes, migraine headaches, or Marfan’s syndrome may also display more severe manifestations of common eye problems.

**PHYSICAL EXAMINATION**

Once a history is obtained, and unless there is obvious trauma to the eye, the examination should begin with a measure of visual acuity. A standard eye chart needs to be available, and the athlete should use his or her glasses if normally required. If the athlete does not have the corrective glasses or contacts available, a pin-holed piece of paper can serve as a substitute lens for the examination. Newspaper-sized print may also be substituted and held about 14 inches from the patient’s face if no eye chart is available. Visual acuity should be documented for each eye separately as well as together. Serial examinations of visual acuity should be conducted to monitor for optic nerve compression in high-energy trauma cases. Corrected vision less than 20/40 warrants a more aggressive search for serious internal eye damage.

After visual acuity is assessed, inspection and manual palpation of the orbit and surrounding structures is done. Any suspicion of fluid leakage from the eye requires immediate transfer of the athlete to rule out a ruptured globe. No drops should be instilled; the eye should be protected with a shield, and the athlete transported for formal ophthalmologic evaluation. The same holds true for a penetrating foreign body, which should be secured to prevent further motion and the athlete urgently referred to an ophthalmologist. Examining pupil size for symmetry and reactivity is critical in ascertaining near-optical functions. Consensual light reflex should be tested to look for an afferent pupillary defect (APD). Tenderness or crepitus over the orbital rims alerts the examiner to more serious injuries to the skeletal structures of the face and skull. Lateral observations can detect proptosis or enophthalmos, which suggest retroglobal pathology including trauma and infection. Extra-ocular muscle function should be examined for palsies. An assessment of visual field defects must be performed. Once a primary survey is completed, a more thorough examination can be safely done. Instilling one or two drops of topical anesthetic can moderate tearing during the examination.

Reviewing the innervation of the eye and its surrounding structures is useful in pinpointing damaged areas. Cranial nerve (CN) II is the optic nerve responsible for carrying the electrical signal from the retina to the occipital lobe. Cutaneous innervation of the periorbital region and eyeball is supplied by the ophthalmic branch of the trigeminal CN V. CN III (oculomotor) directs the levator palpebrae superioris, superior rectus, medial rectus, inferior rectus, and inferior oblique muscles. It also provides parasympathetic fibers, which constrict the pupil. CN IV, the trochlear nerve, innervates the superior
oblique, and CN VI, abducens nerve, the lateral rectus. Table 1 depicts clinical findings with associated nerve injury. All palsies of ocular muscles require neuroimaging.

### Table 1
Clinical findings with associated nerve injury

<table>
<thead>
<tr>
<th>Injured nerve</th>
<th>Pupil</th>
<th>Affected eye position</th>
<th>Normal eye position</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>CN III</td>
<td>Dilated</td>
<td>Down and out</td>
<td>Centered</td>
<td>Rare</td>
</tr>
<tr>
<td>CN IV</td>
<td>Normal</td>
<td>Displaced up</td>
<td>Centered</td>
<td>Often bilateral</td>
</tr>
<tr>
<td>CN VI</td>
<td>Normal</td>
<td>Turned in</td>
<td>Centered</td>
<td></td>
</tr>
</tbody>
</table>

### TRAUMA

Sports can be classified by their level of risk to induce eye-related trauma. Common sense dictates high-risk sports such as boxing, football, wrestling, basketball, and baseball predispose the athlete to an increased number of injuries compared with activities such as cross-country, swimming, and gymnastics. Thus, proper equipment and protection is essential, but varies according to the level of each sport’s risk. The American Society for Testing and Materials (ASTM) provides proper standards for protective eyewear in sports. Proper fit, the strength of lens, and adequate coverage of the eye socket are the variables monitored by this group. Specific recommendations can be found at [www.cpsc.gov/cpscpub/pubs/reports/2000rpt.pdf](http://www.cpsc.gov/cpscpub/pubs/reports/2000rpt.pdf). Unfortunately, despite the obvious benefits of protective eyewear, more than 42,000 sports- and recreation-induced eye injuries were reported in the year 2000 in the United States [1]. The Protective Eyewear Certification Council can also verify appropriate eyewear for particular sports. Their database can be found at [www.protecteyes.org](http://www.protecteyes.org).

### Orbital Fracture

The skeletal structure of the orbit consists of thick portions of the temporal, frontal, zygomatic, and maxillary bones, which compose the rim. The interior portion is formed by the delicate bones of the ethmoid, lacrimal, orbital plate, sphenoid, and palatine. The term “blow-out fractures” refers to the pattern of orbit fractures when the energy of an impact is transmitted through one or more of the walls of the orbit. There are two descriptions of how this can occur. The “hydraulic” mechanism causes the inferior or side walls of the orbit to fracture in order to release the pressure that is transmitted to the globe and vitreous when the anterior-posterior diameter of the eye is compressed. The “buckling” mechanism describes a force applied to the bony orbital rim in a fashion whereby the energy travels posteriorly down multiple sides into the orbit until the force vectors collide, and then escapes via a fractured plate [2]. Stepping gently down on an empty styrofoam cup until one side of the cup buckles illustrates this effect. These high-energy injuries can also be associated with lid, corneal, lens, globe, retinal, and optic nerve complications.
Facial Fracture

When the athlete presents with a blunt trauma, timing from diagnosis to treatment is critical in order to lessen the degree of possible vision loss. If no globe rupture is suspected, evaluation of extra-ocular movements (EOM) should be performed next. The athlete should not be allowed to blow his nose, because the increased intrasinus pressure can further displace soft tissue and bone fragments. One study using CT scans of 55 children who had orbital trauma [3] showed that the majority of fractures (61%) occurred inferiorly through the orbital surface of the maxillae. When this occurs, soft tissue and the inferior rectus can herniate inferiorly into the maxillary sinus. The patient may complain of diplopia from the trapped rectus, and on examination may be unable to follow the examiner’s finger superiorly. Another study of adult facial-trauma patients [4] showed medial and posterior-lateral wall fractures to be the most common, often with more than one bone fractured. Even if EOM are normal, suspicion for fracture should be relative to the amount of energy sustained in the trauma. Facial radiographs are no longer warranted because the availability and increased sensitivity of the CT scan have proven far superior. The coronal planes are often best suited to see any disruptions in the bony contour. Other facial fractures may include the external orbital rim, zygomaticomaxillary (ZME), naso-orbito-ethmoidal (NOE), frontal sinus, and LeFort I–III patterns.

Treatment of exterior and interior orbital fractures depends on the age of the athlete, extent of soft-tissue involvement, and fracture pattern. Entrapment of soft tissue with diplopia usually mandates surgical repair, as do fractures greater than one half of the orbital floor or enopthalmos greater than 2 mm. Children have fewer orbital fractures but have a higher percentage of problems with persistent diplopia [5]. Other fractures can be closely monitored to see if the diplopia resolves after several days.

The athlete will likely be out of competition for several months, and perhaps permanently, if she sustains an injury of this magnitude.

Corneal Abrasion

Corneal abrasions are more commonly seen than orbital blowout fractures. The cornea consists of five layers. Listed from superficial to deep, they are: epithelium, Bowman’s membrane, stroma, Descemet’s membrane, and endothelium. The stroma is the thickest layer and composes 90% of the cornea’s depth. There are no blood vessels in the cornea, and it receives its oxygen supply from the atmosphere. Athletes presenting with corneal injuries often complain of a foreign body sensation, photophobia, tearing, and pain. Vision can be distorted, especially if the abrasion is directly over the pupil. When an abrasion is suspected, application of topical anesthetic drops usually allows for a more comfortable examination. Some corneal defects are visible to the naked eye, but if seen initially, one drop of fluorescein dye inside the lower lid is applied. A blue-cobalt light is then used to look for the dye’s uptake in the corneal defect. The size and location of the injury can then be evaluated with a rough idea of how deep the void is. Examination with a slit lamp, if available, gives a much better view of the depth
of the injury. A magnifying lens from the ophthalmoscope or examination headset should be used to search for small particles of debris. Every examination should include eversion of the lid to look for a retained foreign body. This is best accomplished by having the patient look down, grasping the upper lid eyelashes in one hand, and using a cotton-tipped swab to apply pressure on the superior tarsal plate, thus everting it. Ingrown eyelashes, cysts, and even contact lenses may be hidden there. If there is a foreign body embedded in the cornea, two approaches may be used to remove it. A moistened, cotton-tipped swab is often enough to catch the object and remove it, or a skilled examiner may be comfortable using the tip of a sterile 18-gauge needle to lift the object out, using extreme caution not to penetrate the cornea any further. If the object penetrates the entire cornea or is unable to be retrieved, an ophthalmologist should be consulted.

Fortunately, the cornea has the most rapidly regenerating cells in the body, with the entire epithelial layer being replaced every 48 to 72 hours. Therefore, superficial injuries to the cornea usually keep the athlete out of action only for a short time. Most ophthalmologists recommend prophylactic antibiotic use with abrasions in order to prevent infection, which can ultimately lead to corneal ulcers. Sulfacetamide use is often discouraged because many patients will become irritated from the drops themselves; erythromycin or gentamicin preparations are preferred. Patching of the affected eye for a corneal abrasion was previously considered standard of care; however, a recent meta-analysis [6], along with multiple other randomized controlled trials, showed no benefit in pain relief or healing rates using patches. Injuries to the basement membrane may require a different approach, but for an uncomplicated corneal abrasion, no good evidence exists in favor of patching. Follow up examination should take place in 24 hours to evaluate for signs of healing. The athlete should be pain-free with a normal examination before being allowed to return to play. Contact lenses should not be used again until the cornea is well healed. Large abrasions or those associated with iritis can be treated with mydriatics and oral pain relievers for 1 or 2 days, but prolonged use of topical anesthetics is strongly contraindicated.

**Hyphema**

Between the cornea and the iris is the anterior chamber, which is filled with perfectly clear aqueous fluid. Although less likely than injuries of the cornea, hyphemas—trauma-induced bleeding into the anterior chamber—may nevertheless be encountered by the training room clinician. Often a direct blow to the orbit is responsible for bleeding into the aqueous fluid, but blood dyscrasias, diabetes, vitreous hemorrhage, and neoplasia can cause them as well. Athletes who have sickle cell anemia are at considerable risk for complications if they sustain a hyphema. Prominent hyphemas are easily recognizable, but some may only be detected using a slit lamp. This should not be confused with the more common presentation of a subconjunctival hemorrhage, in which only the white sclera of the eye is bloodstained, and the corneal region is spared. Patients will
often have tearing, photophobia, and blurred vision, as with corneal abrasions. The bleeding is from tearing of the iris or ciliary vasculature, or from a concussive mechanism that ruptures the blood vessels. The blood may obstruct outflow of the aqueous fluid and result in elevated eye pressures and acute glaucoma. Grades 1 through 4 describe the level of blood that fills the anterior chamber on examination, from 25% to 100%, respectively.

After reviewing the history for sickle cell, bleeding disorders, or prior eye surgeries or trauma, the examination consists of searching for other trauma to the orbit, cornea, iris, lens, and retina. Visual acuity should be measured and pupillary response noted. Hyphemas should always be examined with a slit lamp, and intraocular pressures measured with tonometry. The slit lamp can obtain a better look at the posterior cornea, which can be penetrated by the blood and permanently stain the endothelium. In general, the more blood in the chamber, the higher the risk of blockages occurring in the trabecular meshwork, inducing glaucoma, both acutely and long-term. It is recommended that these athletes receive a full examination from an ophthalmologist to carefully evaluate the posterior structures and guide the postinjury care. Traumatic, grade 1 hyphemas with no underlying pathology can be managed on an outpatient basis, doing serial examinations every 1 to 2 days. Aspirin and nonselective nonsteroidal anti-inflammatory drugs (NSAIDs) must be avoided to prevent rebleeds, and the head must remain elevated to keep the blood settled below the level of the pupil and central cornea. Mydriatics are often used to keep pressures as low as possible. Clearance for return to play varies with each case, and should be managed by the ophthalmologist. A rebleed in the first week is often more severe than the initial one and must be urgently treated. Annual checks by an ophthalmologist should be performed to watch for angle recession glaucoma in the future.

Retinal Injury
Deep beneath the anterior chamber, under the iris and through the vitreous, lies the retina. A neurosensory tissue, the retina contains the rods and cones responsible for changing the light images into nerve impulses for the optic nerve, which generates sight. The retina is a very delicate tissue, adhered to the retinal pigment epithelium (RPE), which contains the retinal blood supply and joins the inner surface of the eye itself. The macula is the fine-vision center found just lateral to the optic cup and disc. Injuries to the macula are the worst retinal injuries and, as with all detachments, are medical emergencies. The longer the retina is detached from its blood supply, the greater the likelihood of permanent blindness. Trauma is always a possible source of retinal detachment; the most common types are reviewed below.

With age, the consistency of the vitreous changes from a thick, gel-like substance to a thinner, more watery fluid that occupies less space. When this occurs, its pressure on the retina may lessen, and as space is created, the retina may peel off the back wall of the eye like old wallpaper. This tends to happen more frequently in very near-sighted individuals because their anterior-posterior distance
of the globe is longer than normal. “Rhegmatogenous” retinal detachments are the most common, and occur when there is a small tear in the retina that allows the liquid vitreous to fill in behind it and spread the size of the initial defect. Most patients are not aware that they have a small defect, and even a minor blow to the eye can shift fluid pressures enough to cause a symptomatic tear. Trauma is the leading cause of retinal tears in patients under 18 years of age [7]. A patient who had a retina tear may describe any or all of the following symptoms: sudden loss of vision, onset of or increase in “floaters,” bright flashes of light, or an expanding shadow growing over the field of vision.

Inflammatory problems of the eye or small tumors may cause “exudative” retinal detachments. There are no breaks or tears in the lining of retina with this type of detachment, merely a seepage of fluid behind the retina that separates it from the surface of the RPE. “Traction” detachments are the least common type, and occur when fibro-vascular tissue in the vitreous cavity adheres to the retina and pulls it off of its foundation. Diabetics often have this type from the proliferative retinopathy that can occur with long-standing disease. When the athlete presents with symptoms concerning for a retinal detachment, whether from injury or spontaneous causes, a dilated fundoscopic examination is essential. If adequate visualization of the retina is possible, the entire retina must be scanned to look for a tear, although it may be difficult to find the defect using the ophthalmoscope. The examiner’s level of comfort with the ophthalmoscope should dictate whether or not to perform an examination or to arrange for immediate transfer to an ophthalmologist. No special patching or drops are indicated before transfer.

Depending on the type, size, and duration of the retinal tear, several surgical interventions may be used to repair as much of the damaged retina as possible while attempting to also prevent further tearing. A scleral buckle is a common procedure done to the external eye. By tightening the outer surface of the globe with a silicone buckle, the potential space of the inner globe and vitreous is decreased, which in turn creates more pressure to push the retina back onto its base. Cryotherapy and laser therapy can help scar the retina back down to prevent future tears. Pneumatic retinopexy works well for superior retinal tears. This procedure involves injecting a gas bubble, typically perfluoroproprane, into the vitreous to migrate over the tear and press it back into position. After a few days, the gas dissolves and the eye produces more vitreous to take its place. Laser or cryotherapy is then used in conjunction to adhere the retina down. Regardless of which procedure is used, the athlete who sustains a retinal tear is held from contact sports indefinitely. The risk of a repeat tear is high with any trauma to the eye and may result in permanent vision loss.

**SPECIAL MEDICAL CONDITIONS**

As our population ages and the benefits of regular exercise are recognized by more people, the odds of witnessing more atypical eye disorders also rises. Some individuals may suffer from migraines, other patients will present with
complications of Marfan’s syndrome, both diagnosed and undiagnosed. Still others who have ocular manifestations of cerebrovascular diseases may seek treatment for their eye complaints in your training room.

**Migraine Headache**

Migraine headaches affect millions of people each year in the United States, both athletes and nonathletes alike. Although the true mechanism is not fully understood, cerebral vascular dilation is felt to be a major source of pain and disability. Precipitating factors may be different for each individual, but well-described triggers include lack of sleep, stress, alcohol, chocolate, aged cheese, sunlight, strong odors, or even exercise. Differentiating which type of migraine an athlete is experiencing can be quite challenging, but is imperative to ensure proper treatment.

The classic migraine begins with an aura that precedes the onset of the headache by 10 to 30 minutes. The aura may contain a vast array of different symptoms that often affect the patient’s vision in one or more ways. Flashing lights, wavy lines, visual field defects, or complete vision loss may occur with different migraines. The patient may have limb weakness, facial numbness, and even be confused during the aura and with the headache. If the athlete presents with their first classic migraine, any of the above symptoms can easily be mistaken for a true ocular emergency or a cerebral vascular accident. Age, lack of trauma, and other pertinent past medical problems can help decipher the source of the visual complaints. After the aura, the patient usually experiences a unilateral or bilateral headache that is typically intense, throbbing, and incapacitates the patient for hours to days. The common migraine has a similar headache pattern, with a milder prodrome, if any. Photophobia, vomiting, or phonophobia may all appear in the patient experiencing a migraine. One key in differentiating a migraine with aura from the ocular migraine is the source of the symptoms. Migraines with aura symptoms come from the occipital lobe, whereas ocular migraines arise from the retinal vessels. Therefore, covering the affected eye can relieve some symptoms with the ocular migraine but does little for classic migraine. Table 2 lists differences among these headaches with a vascular origin.

If the migraine is of new onset, special precautions should be taken to ensure the retina is not the source of symptoms, and often a CT scan of the brain is needed to confirm the absence of intracranial hemorrhage. Treatments for migraines have improved dramatically with the introduction of triptans, which are serotonin receptor (5HT) agonists that can lessen cerebral blood vessel diameters. Classically, migraines were treated with anti-inflammatories, caffeine, and other pain relievers. It is now widely accepted that triptans are the first line of medication for true migraine attacks. Oral, intranasal, intravenous, and intramuscular preparations are available. Access to those medications in the training room can be of great benefit because earlier administration appears to lessen the length and the intensity of the attack. Keeping athletes informed as to potential triggers for their particular type of migraine is essential as well. An
<table>
<thead>
<tr>
<th>Migraine type</th>
<th>Aura</th>
<th>Headache</th>
<th>Visual symptoms</th>
<th>Neurological symptoms</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Classic</td>
<td>Yes</td>
<td>Intense, often behind eyes</td>
<td>Photophobia, flashing, blurry</td>
<td>Facial tingling, numbness</td>
<td></td>
</tr>
<tr>
<td>Common</td>
<td>No</td>
<td>Same</td>
<td>Same</td>
<td>Same</td>
<td>Rare</td>
</tr>
<tr>
<td>Ophthalmoplegic</td>
<td>Possible</td>
<td>Possible</td>
<td>Pain, ptosis, diplopia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemiplegic Basilar artery</td>
<td>Yes</td>
<td>1–2 hours after onset Intense</td>
<td>Blurry, flashes Diplopia</td>
<td>Vertigo</td>
<td>Vertigo, muscle coordination.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 2
Differences among headaches with a vascular origin

Mild aura possible
Mimics CVA, CN III dysfunction
Origin in childhood
Females, may lead to stroke.
athlete known to have migraines with new visual symptoms can make a
diagnosis obscure, but a detailed history and thorough examination of the
retina and optic disc/cup will help management decisions, ranging from obser-
vation and treatment with triptans to an ophthalmology consult or a CT scan.

Cerebrovascular Disease
Yet another source of eye symptoms is the cerebrovascular system. Although
cerebrovascular disease presents most commonly in older patients, arteriove-
nous malformations, blood clots, and cerebral ischemia can present at any age.
Visual symptoms of arterial or venous disease in the eye or brain can mimic
many of the conditions previously described, thus the examiner must be thor-
ough and precise with the history and examination.

It is vital to stratify each patient based on age, sex, race, health history, medi-
cations, and current symptoms. When just focusing on the eye complaints
themselves, it can be harder to narrow down the differential diagno-
sis, because similar presentations can have very different pathologies. Tran-
sient ischemic attacks (TIA) can present with any symptoms of a stroke,
and may include slurred speech, limb weakness, or altered vision. Amauro-
sis fugax, a progressive shadelike dimming of vision, is a classic symptom
of carotid artery disease, but can also develop from a thromboembolic oc-
cclusion of the retinal artery. Similar findings arise in some migraine suf-
ferers as well, so evaluating the patient for risk factors of a true ischemic
cause for vision changes is essential. Questioning for a history of hyper-
tension, atherosclerotic disease, heart valve defects, or thromboembolic dis-
ease may help. Examining the fundus to look for arterio-venous (AV) nicking
(an indication of intrinsic vascular disease), and listening to the carotids for
bruits and the heart for significant murmurs may also aid in the diag-
nosis. Treatment for vascular ischemic events is based on the diagnosis,
should be determined by an ophthalmologist or neurologist, and may range
from observation to anticoagulation with heparin or tissue plasminogen acti-
vator (TPA).

Diabetes
When the presenting athlete is diabetic, a vast array of eye pathologies
may surface. Diabetic retinopathy is well known to decrease vision, and should
be followed closely with annual dilated eye examinations and laser photother-
apy as necessary. Acute vision loss associated with or without pain may occur
with branch or central retinal vein occlusion. Retinal veins, which drain blood
from the retina, become blocked, typically at the intersection of a hypertro-
phied artery. This results in venous congestion, macular edema, and possibly
macular ischemia. The fundoscopic examination often shows the vessel dilation,
hemorrhages, and edema in and around the veins. Hypercoagulable patients
and those who have various vasculitides may experience this eye disorder as
well. Treatment typically involves laser therapy, which is more effective for
branch retinal vein occlusions than the central vein. Bilateral cases often indicate
a systemic cause.
The current medical therapies of anticoagulants, troxerutin, corticosteroids, or thrombolytic agents still produce unsatisfactory results. Surgical interventions are not yet supported by randomized, controlled trials [8].

**Marfan’s Syndrome**

Tall, long-limbed athletes are desired in basketball, football, and volleyball, and the majority of these athletes are healthy, normal individuals. Occasionally a tall athlete will demonstrate physical features that are concerning for Marfan’s syndrome. Classically inherited as an autosomal dominant trait, Marfan’s is caused by mutations in the gene for fibrillin-1 which is important in collagen and elastic tissue formation. Between 10% and 30% of cases have no family history and are thought to result from a new mutation. Overgrowth of the long bones, a pectus excavatum deformity, and fragility of the skin, aorta, and eyes are the typical manifestations. Although the inadequate aortic root strength is the most life-threatening complication of the disease, problems with the eyes can lead to life-altering changes. Early recognition and accurate diagnosis are critical to avoid tragedies such as the death of Olympic volleyball great Flo Hyman, who died abruptly with an aortic root dissection and was subsequently found at autopsy to have Marfan’s syndrome.

Athletes who have known Marfan’s disease should be disqualified from competitive sports if they have a suspected or diagnosed aortic root dilation. Eye manifestations occur in more than two thirds of the patients, however, and are not an absolute contraindication to participation in contact or other sports. Corneal flattening, upward lens dislocation, myopia, and retinal detachment may occur in the Marfan athlete. Because of the myopia, they are also at higher risk for glaucoma. A retrospective review of 104 patients (208 eyes) who had visual disturbances from Marfan’s [9] found that 72% of the eyes had a lens dislocation, fewer than 10% had retinal detachments, and 2% had glaucoma. Relative risk to the individual in his sport should guide the physician and athlete in their decisions to play or not. Protective eyewear should be highly recommended if the athlete competes in any sport, even though the visual problems can be spontaneous.

In the non-Marfan’s general population, a lens dislocation is a rare phenomenon, but nevertheless requires rapid recognition and management. During the course of a full eye examination, an ellipse of the lens can often be seen contrasted on the red reflex in the pupillary window. It may or may not be painful, and is treated on an urgent basis to prevent damage of other internal structures. The treatment procedures vary, but usually involve lensectomy after vitrectomy with an implantation of a synthetic lens sutured behind the iris.

**Vision Correction Surgery**

In the quest to become faster, stronger, and better, athletes may pay any price to achieve their objectives. Vision is one of the key components of an athlete performing her best on game day. Interestingly, however, one study found that 28% of 939 athletes screened had less than 20/25 vision and did not know it [10]. Years ago, the switch from glasses to contact lenses allowed a sense of freedom.
in performance. Today the surgical techniques of radial keratotomy (RK), laser in-situ keratomileusis (LASIK) and photorefractive keratectomy (PRK) can permanently improve sight for the athlete. A large percentage of athletes at the high school, college, and professional levels require vision-correcting lenses to play their sports, especially those in which hand-eye coordination is critical. Athletes may seek your advice regarding these procedures, and rarely present with complications of them.

Roughly two thirds of the refraction power in the eye comes from the cornea, with the remainder from the lens. Thus reshaping the cornea’s surface with these techniques can dramatically improve the sharpness of the image focused on the retina. Currently, LASIK is the most commonly used treatment. It involves using a surgical microkeratome (blade) to create a hinged corneal flap that is retracted. The underlying surface of the cornea, the stroma layer, is then partially excised with a laser. The flap is then repositioned and heals back in place. Steroid and antibiotic drops are instilled for 4 days afterwards. Athletes are advised to avoid contact competitions for 2 to 3 weeks postprocedure. Complications of the procedures include loss of corneal sensitivity, glare, hazes, and decreased contrast sensitivity.

Because these procedures are relatively new, long-term risks for athletes are not yet well-defined because no prospective trials have been published. Radial keratotomy may weaken the structural integrity of the eye and thus the risk of globe rupture from trauma appears to be increased [11]. The corneal flap created with the LASIK procedure does produce weakened fibers at the hinged point from which the flap can be lifted off with trauma. Ideally though, the flap can be resealed and allowed to heal back in position. Four cases of traumatic dislocation were reported between 10 days and 2 months following the LASIK procedure [12]. All the flaps were resituated and all patients recovered their pretrauma level of vision. If an athlete dislodges a corneal flap after vision correction surgery, the eye should be covered and he should be referred immediately to an ophthalmologist experienced with the LASIK and RK procedures for treatment and repair.

**SUMMARY**

The eye remains an incredibly important yet delicate structure of the human body. The number of preventable eye injuries resulting in permanent partial or full vision loss remains unacceptably high. Convincing athletes to overcome preconceived notions of protective eyewear use for sports can be challenging, but well worth the time and energy if we can reduce the incidence. Regardless, injuries will occur, and the challenge to diagnose and treat them quickly and safely remains. Maintaining the skills needed to perform a good fundoscopic examination will help to methodically work through the differential diagnoses. Cultivating a working relationship with an ophthalmologist and feeling comfortable consulting with him is always beneficial when an obvious cause of vision loss is not apparent. Although the majority of ophthalmologic issues in athletes can be managed well in the training room setting,
always maintain a high level of vigilance for rarer and more serious ophthalmologic pathologies.

References