The Physical Examination of the Eye

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There are more than 2 million visits annually to United States emergency departments for complaints involving the eye and its surrounding tissues [1]. Approximately one half of these visits are related to trauma to the eye and its adnexal tissues, 3% of which require admission to the hospital [1,2]. Six percent of eye injuries presenting to emergency departments annually are related to the use of drugs and/or alcohol [1]. Most of these patients are treated initially by emergency physicians, with or without ophthalmology consultation. Performing a complete, accurate, and efficient physical examination of the eye allows the emergency physician to treat most eye complaints and to communicate effectively with the ophthalmologist on call. The physical examination of the eye requires a Snelling Chart (or substitute), a light source, ophthalmoscope, slit lamp, a method for determining intraocular pressure, proparacaine ophthalmic solution, and fluorescein stain [3].

Knowledge of the anatomy of the eye is essential in emergency medicine. To properly diagnose ocular problems, one should have full knowledge of the eye, orbit, cranial nerves, musculature for the control of eye movements, and blood supply of the eye.

The orbit

The orbit is composed of four walls. The roof of the orbit is comprised of the frontal bone and the lesser wing of the sphenoid bone. The lateral wall is made of the zygomatic bone and the greater wing of the sphenoid bone. The lateral wall and roof are separated by the superior orbital fissure. The floor of the orbit is composed primarily of the maxilla. However, portions of the zygomatic and palatine bones contribute to the floor. The medial wall is formed by portions of multiple bones including the ethmoid, lacrimal,
sphenoid, frontal, and maxilla. The anterior portion of the orbit is narrower than the area behind the rim, which adds protection [4–7].

The orbit lies in close proximity to the paranasal sinuses (Fig. 1), allowing sinus infections to spread to the periorbital tissues (preseptal cellulitis) and into the orbit itself (orbital cellulitis) [8]. These conditions will be discussed in detail in an article by Mueller and McStay elsewhere in this issue.

**The external eye**

The external anatomy of the eye is made up of the eyebrows, the eyelids, and the lacrimal apparatus. The eyebrows consist of thick skin and hair with muscle fibers underneath. The eyelids function to protect the eyeball by closing. They are made of thin skin covering the orbicularis oculi muscle. This muscle closes the lids. Beneath the muscle is areolar tissue followed by a fibrous connective tissue layer called the “tarsal plate.” The lids are covered on their posterior surface by mucosa called the “palpebral conjunctiva.” Between the orbit and tarsal plate lies fascia that makes up the orbital septum.

The lacrimal apparatus is composed of the lacrimal gland, lacrimal sac, canaliculi, and the nasolacrimal duct. Many accessory lacrimal glands also exist. The lacrimal gland produces tears that coat the anterior portion of the eye. The tears are drained through the canaliculi to the lacrimal sac. The lacrimal sac drains into the nasal cavity via the nasolacrimal duct [5,6].

During examination of the external structures surrounding the eye, many diseases and injuries should be looked for specific to the structures being examined. Examination of the lids may reveal chalazion, hordeolum, or lid cellulitis, all of which will be discussed by Mueller and McStay in an article found elsewhere in this issue. Abnormalities of the orbicularis oculi muscle may be seen in seventh cranial nerve palsies. Lid lag (ptosis) may be seen in third cranial nerve palsies and in Horner’s syndrome. These entities will be discussed by Duong, Leo, and Mitchell in an article found elsewhere in this issue. Conjunctivitis

![Fig. 1](image-url)
may involve the inner lining of the eyelids (palpebral conjunctiva). The palpebral conjunctiva lining the upper lid is a common site for occult ocular foreign bodies. All elements of the tear-producing and collecting system are subject to infection (dacrocystitis) or obstruction and require special attention in the setting of trauma, because lacerations to these structures may require repair by an ophthalmologist. These injuries will be discussed in an article by Bord and Linden elsewhere in this issue.

The eyeball

A review of daytime presentations to an ophthalmologic emergency department in Sydney, Australia found the following five most common diagnoses: conjunctivitis, keratitis, cataract, corneal abrasion, and iridocyclitis [9].

The conjunctiva

A thin mucous membrane covers the posterior lids and anterior sclera. This is the conjunctiva. It is continuous with the skin at the margin of the lid. In the United States, more than 500,000 patients present to the emergency department annually for inflammation of the conjunctiva [1]. Conjunctivitis will be discussed in detail in the article “Ocular Infection and Inflammation” by Mueller and McStay and “The Red Eye” by Mahmood and Narang elsewhere in this issue.

Tenon’s capsule

Tenon’s Capsule (also called the fascia Bulbi) is a fibrous globe that envelops the eyeball from the optic nerve to the limbus. Tubular reflections of the capsule enclose each extraocular muscle at its site of attachment and fuse with the fascia of these muscles and attach to adjacent orbital structures. These reflections limit the movement of these muscles [5,6].

The sclera and episclera

The fibrous coating of the eye is the sclera. This protective coating is made almost entirely of collagen. It is white in color and very dense. Anteriorly, it is continuous with the cornea. The episclera is a thin layer of elastic tissue that covers the anterior sclera [4,6].

Inflammation of these layers may be associated with connective tissue disorders. Scleritis is usually painful, whereas episcleritis is not. The redness associated with episcleritis blanches with instillation of phenylephrine drops, whereas the redness associated with scleritis does not. See the article by Mueller and McStay for a detailed discussion of these diseases.

The cornea

The transparent structure covering the anterior of the eye is the cornea. It is composed of five separate layers: the epithelium, Bowman’s layer, the
stroma, Descemet’s layer, and the endothelium. The epithelium is highly prone to damage [6].

Corneal abrasions and foreign bodies comprise the majority of corneal abnormalities presenting to emergency departments. Other entities include keratitis, corneal ulcers, corneal perforation, and corneal exposures. Proparacaine to make the patient comfortable and fluorescein instillation to visualize corneal defects, in conjunction with slit lamp evaluation, are essential to evaluate the cornea. Corneal abnormalities are discussed in the articles “The Painful Eye” by Dargin and Lowenstein and “Ocular Infection and Inflammation” by Mueller and McStay in this issue.

The anterior chamber

Between the cornea and iris is the anterior chamber. The anterior chamber is filled with aqueous humor. The aqueous is formed by the ciliary body. It is drained from the anterior chamber by the trabecular meshwork. The trabecular meshwork is a series of collagen and elastic tissue with trabecular cells that form a filter. The ciliary muscle can contract and increase the size of the filter pores. This enhances the drainage of the aqueous. The aqueous is absorbed at a venous channel (the canal of Schlemm) where the iris and cornea join. This is the angle of the anterior chamber [4–7].

People with a narrow anterior chamber angle are at risk for acute angle closure glaucoma (Figs. 2 and 3). This vision-threatening, sometimes elusive diagnosis will be discussed in the article by Mahmood and Narang in this issue.

The uvea

The uvea consists of the iris and the ciliary body (anterior uvea) and the choroid (posterior uvea) [6].
The iris

The amount of light entering the eye is controlled by the iris. It is an extension of the ciliary body. It is a flat surface with a central, round opening (the pupil). The posterior surface of the iris is heavily pigmented. The stroma of the iris contains the sphincter and dilator muscles. The size of the pupil is controlled by parasympathetic innervation (contraction) and sympathetic innervation (dilation) [4–7].

The iris is the site of abnormalities of both traumatic and atraumatic etiologies. Iritis (anterior uveitis) may be infectious, rheumatologic, or idiopathic. Trauma may cause traumatic iritis, traumatic midriasis, or tears of the iris. Anterior uveitis will be discussed in the articles entitled “The Red Eye” by Mahmood and Narang and “The Painful Eye” by Dargin and Lowenstein in this issue.

The ciliary body

The ciliary body is composed of a series of veins and capillaries. The epithelium covering this body produces the aqueous. The ciliary muscle is made up of a series of muscle fibers arranged in a longitudinal, circular, and radial direction. These fibers can contract to change the shape of the lens and provide for different focal lengths [6].

The choroid

The choroid lies between the retina and sclera. It is comprised of a series of blood vessels that provide flow to the outer portion of the retina. There are three layers of these blood vessels [6].

Posterior uveitis, or choroiditis, is a vision-threatening emergency requiring urgent treatment. It is discussed in detail in the article by Mueller and McStay elsewhere in this issue.

The lens

The lens is approximately 4 mm thick and 9 mm in length. It is convex, colorless, and transparent. It is held behind the iris by the zonule. The
zoniule connects to the ciliary body. As one ages, the lens becomes larger and less flexible. The lens contains no nerves or blood vessels [6].

The lens may be dislocated in the setting of trauma. Also, trauma may cause cataract formation. See the article by Bord and Linden elsewhere in this issue.

The retina

The specialized neural tissue lining the posterior, inner part of the globe is the retina. It is semitransparent and multilayered. It is composed of ten layers containing nervous tissue, pigmented epithelial cells, and photoreceptors. The retina is easily detached from its epithelium, except at the optic disk [4–6].

Retinal detachment, a vision-threatening ophthalmologic surgical emergency, can be recognized by historical features (visual loss, floaters, flashing lights) and on physical examination (elevated portion of retina on fundoscopic examination). This will be discussed in detail in the by Vortmann and Schneider found elsewhere in this issue.

The vitreous

Making up two thirds of the volume and weight of the eye, the vitreous fills the space between the lens and retina. It is clear and gelatinous. It is composed of collagen and hyaluronic acid but is 99% water [4–6].

Vitreous floaters are defined as dots or spots floating in the field of vision that change position when the patient shifts his or her gaze to a different direction [10]. They may be from blood or debris in the vitreous fluid. They may be caused by retinal detachment or hemorrhage.

The extraocular muscles

The extraocular muscles control the movements of the eye. There are six extraocular muscles.

The rectus muscles

The eye has four rectus muscles that are named by their insertion: medial, lateral, superior, and inferior. They originate from a common site, a ring shaped tendon called the annulus of Zinn. These muscles adduct, abduct, elevate, and depress the eye [4–6].

The oblique muscles

There are two oblique muscles of the eye. They provide some upward and downward movement of the eye but primarily provide rotational movement. These muscles are the superior oblique and the inferior oblique [4–6].

Disorders of upward gaze in a traumatized eye may be caused by orbital floor fracture with inferior rectus entrapment. This will be discussed in detail
In the article by Bord and Linden elsewhere in this issue. Inability to move the eye, associated with proptosis, may be caused by orbital cellulitis or orbital hemorrhage.

**Innervation of the eye**

The eye is innervated by the cranial nerves listed below.

*The optic nerve (II)*

The optic nerve is composed mainly of visual fibers. It arises from the retina and consists of approximately one million axons. After leaving the globe, the nerve becomes myelinated. Each nerve is then enwrapped in a sheath of fibrous tissue that is contiguous with the meninges. It passes through the orbit and joins the nerve from the other eye at the optic chiasm. At the chiasm, nasal fibers from each optic nerve cross to the opposite tract. These axons then course to the lateral geniculate nucleus. After a synapse at the lateral geniculate nucleus, the nerves end in the primary visual cortex of the occipital lobes [4–6].

*The oculomotor nerve (III)*

The oculomotor nerve originates from the brainstem. It passes through the cavernous sinus and splits into inferior and superior branches. The superior branch innervates the superior rectus muscle. The inferior branch innervates the inferior and medial rectus muscles and the inferior oblique muscle. The inferior branch also provides parasympathetic innervation to the ciliary body [4–6].

*The trochlear nerve (IV)*

The trochlear nerve begins on the dorsal surface of the brain stem. It travels along the lateral wall of the cavernous sinus. It innervates the superior oblique muscle [4–6].

*The trigeminal nerve (V)*

The trigeminal nerve begins in the pons and forms the trigeminal ganglion. It then splits into three divisions. The first division is the ophthalmic division (V1). The ophthalmic division passes through the cavernous sinus and divides to provide sensation to the brow, forehead, cornea, iris, and ciliary body. It also provides sensation to the conjunctiva, eyelids, and the tip of the nose. The second division is the maxillary division (V2). It passes through the infraorbital canal and forms the infraorbital nerve. This provides sensation to the lower lid and cheek. The third division is the mandibular division (V3). It provides sensation over the lower portion of the face and mouth. It also provides innervation to eight of the muscles of the face and jaw [4–6].
The abducens nerve (VI)

Originating between the medulla and pons, the abducens nerve passes within the cavernous sinus. It innervates the lateral rectus muscle [4–6].

Multiple disorders involve the innervation of the eyes. Optic neuritis may herald the onset of multiple sclerosis. It causes decreased visual acuity, an afferent papillary defect, and pain on eye movement. Fundoscopic examination may be normal (if the inflammation is retrobulbar) or may reveal edema of the optic disc (if the inflammation is anterior) [11].

Unilateral or bilateral abducens nerve palsy (inability to abduct one or both eyes) may be an early sign of increased intracranial pressure [12]. A posterior communicating artery aneurysm may compress the ophthalmic nerve, causing lid lag and ophthalmoplegia [12]. These entities will be discussed in detail in the article by Duong, Leo, and Mitchell found elsewhere in this issue. Furthermore, trauma can cause an orbital hemorrhage, which may lead to increased orbital pressure and ischemia of the optic nerve.

The blood supply of the eye

The first branch of the intracranial section of the internal carotid artery is the ophthalmic artery. The ophthalmic artery and its subsequent branches supply the main blood source to the eye and its structures. After the ophthalmic artery enters the orbit, the first branch is the central retinal artery. As the artery continues to course within the orbit, multiple other branches supply the structures of the eye until the most superficial branches reach the eyelids. Here they form arcades that anastomose with the external carotid artery.

The main venous drainage of the eye occurs via the superior and inferior ophthalmic veins. These two veins receive the drainage from the eye including the central retinal vein. They then empty into the cavernous sinus. The venous supply of the periorbital skin is contiguous with the deeper venous structures. Superficial skin infections can therefore spread via the venous system into the cavernous sinus and deeper structures [4–6].

The blood supply to the eye can be threatened in several ways. Embolization of plaque or clot to the central retinal artery can cause central retinal artery occlusion and acute painless monocular visual loss. Central retinal vein occlusion also causes visual loss over a longer time period than central retinal artery occlusion. Inflammation of the ophthalmic artery can result from temporal arteritis, causing painless visual loss, which may be unilateral or bilateral [11]. These disorders will be discussed in the article by Vortmann and Schneider in this issue. Also, trauma can disrupt the blood supply to the eye.

Physical examination of the eye

In performing the physical examination of the eye, one should be able to evaluate both anatomy and function of each eye. The evaluation of the anatomy should focus on whether the problem arises from the globe, the orbit,
or the external structures. The evaluation of the function of the eye should include vision, alignment, and movements. Generally, the physical examination of the eye should begin with a measurement of visual acuity (an exception to this is when ocular exposure to toxic substances requires irrigation before visual acuity measurement [13]—in this case, a quick measurement of ocular pH can precede irrigation if it can be achieved in a few seconds and does not delay irrigation), then the examiner should start peripherally with the periorbital area and work centrally to examine the orbits, lids, the extraocular muscles, the conjunctiva, sclera and episclera, the uvea, the intraocular pressure, the cornea, the pupils, the anterior chamber, and the fundus.

**Vision**

The standard way to test vision is by reading a Snellen chart at 20 feet (Fig. 4). Each eye should be tested separately. The vision is then assigned a two-digit score (for example, 20/50) for each eye. The first number represents the distance from the chart, and the second digit represents the smallest line readable by the patient. Recording the vision separately for each eye, even if the vision is the same in each eye, is essential (for example, visual acuity: right eye 20/50, left eye 20/50). If a patient can read three letters on the next line, this can be recorded as, for example, 20/50 +3. If a patient gets 2 letters wrong on the last line, this can be recorded as, for example, 20/50 –2.

![Standard Snellen chart](image)
Patients who cannot read English can generally be tested with a tumbling E chart, in which the patient identifies the orientation of the letter E (facing to right, left, up, down) [14]. Testing vision in children will be discussed in detail in the article by Prentiss and Dorfman in this issue.

If the subject wears glasses and they are unavailable, vision can be tested through a pinhole. By reading the Snellen chart through multiple pinholes, the image is transmitted centrally through the lens onto the retina, excluding peripheral light. This allows for a clearer image to be formed and substitutes for eyeglasses [6].

If the patient cannot read the chart, he or she should be moved closer to the chart until he or she is 5 feet away. If he or she is still unable to read the chart, the examiner should test vision by having the patient count fingers at 2 feet. If the patient cannot count fingers accurately, the next step is to test whether he or she can detect hand motion. If the patient is unable to detect hand motion, the next step is to determine if he or she has light perception. The eye not being tested must be completely covered to test for light perception. If the patient can perceive light, the examiner can check to see if the patient can determine which direction the light is coming from (light perception with projection) or not (light perception without projection) [12]. If the patient is unable to detect light in an eye, that eye is considered totally blind (no light perception) [6].

Near vision may be tested at the bedside with a near-vision card, held 14 inches from the patient’s face [12,15]. Poor performance on near-vision testing can be caused by acute visual impairment but may also be caused by presbyopia [16], which is age-related chronic impairment of near vision [12].

**External examination**

To begin the physical examination of the eye, one should start with the external structures. The eyelids and surrounding areas should be examined for swelling, erythema, warmth, skin growths, and tenderness. Trauma or infection (periorbital cellulites or zoster) may become apparent on evaluation of the peri-orbital region.

Proptosis can easily be identified with gross inspection of the orbits. Unilateral proptosis may be caused by orbital cellulitis, orbital hemorrhage, cavernous sinus thrombosis, or tumors. Bilateral exophthalmos is most commonly caused by hyperthyroidism [12]. Proptosis can be quantified by the ophthalmologist using an exophthalmometer [14]. Enophthalmos may be caused by an orbital blow-out fracture. The bony structures should be palpated to ascertain tenderness [6].

Lid evaluation may find ptosis, which may be congenital or acquired. Newly acquired ptosis may represent Horner’s syndrome, third nerve palsy, botulism, or myasthenia gravis [10]. Inability to close the lids may result from weakness of the orbicularis oculi muscle, as in seventh cranial nerve palsy. It is important to flip the upper lid if you are searching for a foreign body. Careful evaluation of the lids may reveal infections of the lid (lid
cellulitis) or dacrocystitis, which is an infection of the tear collection system (puncta, canaliculi, nasolacrimal duct). Other lid findings include hordeolum and chalazion. A hordeolum is an acute inflammation of a meibomian gland, and a chalazion is a chronic obstruction and inflammation of a meibomian gland [10,11].

**Extraocular movements**

When testing extraocular movement, special note should be made of the range, symmetry, smoothness, and speed of the eye movements. Nystagmus can also be observed. To test these movements, the patient is asked to fixate on a target with both eyes. The examiner then moves the target in four directions [6]. Disorders of ocular motility may be caused by cranial nerve dysfunction, extraocular muscle entrapment, or increased pressure within the orbit (orbital cellulitis or hemorrhage).

**Examination of the conjunctiva**

It is important to evaluate the conjunctiva lining the lids (palpebral conjunctiva) and the conjunctiva on the surface of the eye (bulbar conjunctiva). Conjunctival injection (prominence of vessels) may be diffuse or perilimbal (radiating outward from the limbus, which is where the cornea meets the sclera). Diffuse conjunctival injection usually results from inflammation or infection within the conjunctiva itself (conjunctivitis), whereas perilimbal injection may be the result of inflammation or infection within the uvea or anterior chamber. Chemosis refers to edema of the conjunctiva [11]. Conjunctivitis will be discussed in detail in the article by Mahmood and Narang found elsewhere in this issue.

Examination of the conjunctiva may also reveal a subconjunctival hemorrhage, which may be spontaneous or traumatic. Although these usually are benign and self-limited, severe subconjunctival hemorrhages (360° of bulbar conjunctiva) may be secondary to ruptured globe or coagulopathy [10].

**Examination of the sclera and episclera**

Inflammation of the episclera and sclera may be difficult to distinguish from conjunctivitis. Episcleritis is usually painless and causes injection of a sector of episcleral vessels. These vessels will blanch with application of phenylephrine. Vision usually is preserved. It is self-limited and may be associated with collagen vascular diseases [11].

Scleritis causes injection of scleral vessels with a characteristic violaceous color. It is painful and often causes decreased vision. The injection will not clear with topical phenylephrine. It is associated with collagen vascular diseases and certain infections (zoster, tuberculosis, syphilis). Treatment depends on severity but often includes systemic steroids [11]. See the article by Mahmood and Narang in this issue for detailed discussion of scleritis and episcleritis.
Examination of the uvea

Inflammation of the anterior uvea (anterior uveitis, iritis, iridocyclitis) can be detected by the associated photophobia and unilateral constricted pupil that results from ciliary spasm as well as cells and flare in the anterior chamber. Causes include trauma, viral, Reiter’s Syndrome, sarcoidosis, juvenile rheumatoid arthritis, and retinoblastoma, and it often is idiopathic [11]. The iris should also be examined for traumatic tears at the papillary margin and for iridodialysis (separation of the iris from the ciliary body) [11].

Inflammation of the posterior uvea can be seen as exudates around retinal vessels and hazy vitreous humor. It can be caused by cytomegalovirus, toxoplasmosis, sarcoidosis, syphilis, herpes, and other causes [10]. Anterior and posterior uveitis will be discussed in the article by Mahmood and Narang in this issue.

Intraocular pressure

The intraocular pressure (IOP) should be measured if glaucoma is suspected. There is a detailed description of various tonometers in the article by Babineau and Sanchez elsewhere in this issue. High pressures may result from acute angle–closure glaucoma, open-angle glaucoma, hyphema, carotid-cavernous fistula, retro-bulbar hemorrhage, and other causes [10]. Low IOP may be seen in ruptured globe, retinal detachment, ocular ischemia, glaucoma medications, and other conditions.

Examination of the cornea

Corneal evaluation is accomplished with the aid of proparacaine, fluorescein, and the slit lamp. Defects in the corneal surface will appear bright yellow-green under the cobalt blue light on the slit lamp when stained with fluorescein [17]. It is an essential part of the emergency eye examination to carefully evaluate the cornea with the slit lamp for foreign bodies, abrasions, and ulcerations [17]. Many causes of infection and inflammation of the cornea (keratitis) have specific patterns of fluorescein uptake on slit lamp examination (herpes simplex, herpes zoster, adenovirus, ultraviolet keratitis) [11]. Corneal edema, seen in acute angle closure glaucoma, will appear to the examiner as a “steamy” cornea, and the patient will see “halos” around lights. Keratitis will be discussed in detail in the article by Dargin and Lowenstein in this issue.

Examination of the pupils

On gross examination, the pupils are symmetric and circular. As many as 20% of normal individuals may have anisocoria (unequal pupils) of less than 1 mm [18,19]. Anisocoria also may be caused by uveitis, trauma, uncal herniation, oculomotor nerve palsy, or Horner’s Syndrome and iatrogenically by dilating or constricting drops or nebulized ipratropium [20]. The
examiner should test the pupils’ response to direct light. As the pupil reacts to direct light, a consensual response (constriction of the opposite pupil) will also occur. After the examiner notes the degree and speed of response of both the direct and consensual response in each eye, one should swing the light between the two eyes (swinging flashlight test) [15]. Each pupil should constrict with both direct and consensual light. If the pupil dilates as the light is swung to it from the opposite pupil, this means that the pupil constricts more vigorously to consensual light than direct light, the hallmark of an afferent papillary defect. This is referred to as a “Marcus Gunn pupil” (Fig. 5) [4–6]. The Marcus Gunn pupil is also discussed in the article by Duong, Leo, and Mitchell in this issue.

Examination of the anterior chamber

Hyphema (blood in the anterior chamber) and hypopyon (layer of white blood cells [WBCs] in the anterior chamber) often can be seen without magnification, but a slit lamp evaluation is essential to adequately evaluate the anterior chamber. With a slit lamp, inflammation of the anterior chamber will be seen as cells (WBCs floating in the aqueous) and flare (increased protein causing the aqueous to appear cloudy) [10]. Cells and flare in the anterior chamber can be seen in uveitis, scleritis, and trauma [11]. The use of the slit lamp is discussed in detail in the article by Babineau and Sanchez in this issue (Fig. 6).

The depth of the anterior chamber can be approximated using a penlight. The penlight is shone tangentially across the cornea from the temporal side. If the entire cornea is illuminated, this implies a deep anterior chamber with

![Fig. 5. Testing for relative afferent papillary difference. (A) The right optic nerve is damaged. The light is shone in the left eye; both pupils constrict (the direct and consensual light reflex). (B) The light is moved to the right eye, and there is no afferent limb to the reflex, thus both pupils dilate. (From James B, Benjamin L. Ophthalmology: investigation and examination techniques. China: Butterworth Heinemann (an imprint of Elsevier); 2007; with permission.)](image-url)
a wide open angle. If the nasal portion of the cornea is in shadow, this implies a shallow anterior chamber with a narrow angle [21].

**Ophthalmoscopy**

The use of a hand-held ophthalmoscope enables the examiner to visualize the fundus. In a darkened room, the patient fixes his vision across the room, and the examiner then brings the ophthalmoscope as close as possible to examine each eye. The dial on the ophthalmoscope can then be adjusted to focus the image. This allows for examination of the structures in the retina. The examiner notes the optic disk, the vasculature, and the macula (Fig. 7). Dilation of the pupil can assist in the visualization of the retina. Examination of the periphery of the retina requires the use of an indirect ophthalmoscope and usually is performed by an ophthalmologist [4–6].

**Summary**

This article has provided a review of the anatomy of the eye and its surrounding tissues. A working knowledge of the functional anatomy of the eye will aid the emergency physician in performing a thorough yet efficient
physical examination of the eye. A goal-directed physical examination of the eye will allow the emergency physician to attempt to identify (or exclude) vision-threatening disease processes and facilitate communication with the ophthalmologist.

Fig. 7. Examples of optic disc abnormalities. (A) Normal disc: Note the distribution of the neuroretinal rim around the disc. (B) A swollen disc: The margin is not clearly demarcated; the patient had papilloedema. (C) The cup is enlarged, the neuroretinal rim thinned, and the inferior-superior-nasal-temporal pattern lost. The patient has glaucoma. (D) New vessels are growing at the disc: this patient had diabetes. (E) The disc has an irregular lumpy appearance and optic disc drusen. (F) Myelinated nerve fibers at the disc margin. (From James B, Benjamin L. Ophthalmology: investigation and examination techniques. China: Butterworth Heinemann (an imprint of Elsevier); 2007; with permission.)
References