42. Ophthalmology

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Ocular emergencies

It is not necessary to refer every patient with an eye disease to an ophthalmologist for treatment. In general, sties, bacterial conjunctivitis, superficial trauma to the lids, corneas, and conjunctiva, and superficial corneal foreign bodies can be treated just as effectively by the surgeon or primary physician as by the ophthalmologist. More serious eye disease such as the following should be referred as soon as possible for specialized care: iritis, acute glaucoma, retinal detachment, strabismus, contusion of the globe, and severe corneal trauma or infection.

In the management of acute ocular disorders, it is most important to establish a definitive diagnosis before prescribing treatment. The maxim "All red eyes are not pinkeye" is a useful one, and the physician must be alert for the more serious iritis, keratitis, or glaucoma. The common practice of prescribing "shotgun" topical antibiotic combinations containing corticosteroids is to be discouraged, because inappropriate use of steroids can lead to complications.

This chapter attempts to summarize the basic principles and technics of diagnosis and management of common ocular problems, with special emphasis on emergencies, particularly those caused by trauma.

Ocular emergencies may be classified as true emergencies or urgent cases. A true emergency is one in which the patient is suffering severe pain or in which a few hours' delay in treatment can lead to permanent ocular damage. An urgent case is one in which treatment should be started as soon as possible but in which a delay of a few days can be tolerated.

Foreign Bodies

If a patient complains of "something in my eye" and gives a consistent history, a foreign body is usually present even though it may not be readily visible. Almost all foreign bodies, however, can be seen under oblique illumination with the aid of a hand flashlight and loupe or other magnifying device.

Note the time, place, and other circumstances of the accident. Test visual acuity before treatment is instituted as a basis for comparison in the event of complications.

Conjunctival Foreign Body

A foreign body of the upper tarsal conjunctiva is suggested by pain and blepharospasm of sudden onset in the presence of a clear cornea. After instilling a local anesthetic, evert the lid by grasping the lashes gently and exerting pressure on the mid portion of the outer surface of the upper lid with an applicator. If a foreign body is present, it can be easily removed by passing a sterile wet cotton applicator across the conjunctival surface.

Corneal Foreign Body

When a corneal foreign body is suspected but is not apparent on simple inspection, instill *sterile* sodium fluorescein into the conjunctival sac and examine the cornea with the aid of a magnifying device and strong illumination. The foreign body may then be removed with a sterile wet cotton application. An antibiotic should be instilled, eg, polymyxin B-bacitracin (Polysporin) ointment. It is not necessary to patch the eye, but the patient must be examined in 24 hours for secondary infection of the crater. If the nonspecialist cannot remove the corneal foreign body in this manner, it should be removed by an ophthalmologist. If there is no infection, a layer of corneal epithelial cells will line the crater within 24 hours. It should be emphasized that the intact corneal epithelium forms an effective barrier to infection. Once the corneal epithelium is disturbed, the cornea becomes extremely susceptible to infection.

Early infection is manifested by a white necrotic area around the crater and a small amount of gray exudate. These patients should be referred immediately to an ophthalmologist. Untreated corneal infection may lead to severe corneal ulceration, panophthalmitis, and loss of the eye.

Intraocular Foreign Bodies

Foreign bodies that have become lodged within the eye should be identified and localized as soon as possible. They usually cause extensive damage to the eye upon entry, and the injury is made worse by leaving the foreign body in place.

In unusual cases, a metallic foreign body can enter the eye, cause minimal initial damage, and be overlooked by the physician. A metallic splinter from a hammer or chisel may enter the eye at high speed, causing minimal symptoms, but complications weeks to years later may result in loss of the eye. The important diagnostic points are a history of pounding "steel on steel" and x-ray search for the fragment. The anterior portion of the eye, including the cornea, iris, lens, and sclera, should be inspected with a loupe or slitlamp in an attempt to localize the entry wound. Direct ophthalmoscopic visualization of an intraocular foreign body may be possible. An orbital x-ray must be taken to verify the presence of a radiopaque foreign body.

An intraocular foreign body should be removed as soon as possible, preferably through the wound of entry. Foreign bodies with magnetic properties can be removed by holding the tip of a sterilized magnet in the wound of entry.

Traumatic Cataract

Traumatic cataract is most commonly due to a metallic intraocular foreign body striking the lens. BB shot is a frequent cause; less frequent causes include arrows, rocks, overexposure to heat ("glassblower's cataract"), x-rays, and radioactive materials. Most traumatic cataracts are preventable. In industry, the best safety measure is a good pair of safety goggles.

The lens become white soon after entry of the foreign body, since the puncture of the lens capsule allows aqueous and sometimes vitreous to penetrate into the lens structure. The patient is often an industrial worker who gives a history of striking steel on steel. A tiny fragment of a steel hammer, for example, may pass through the cornea and lens at high speed and lodge in the vitreous, where it can usually be seen with the ophthalmoscope.

The patient complains immediately of blurred vision. The eye becomes red and the lens opaque, and there may be intraocular hemorrhage. If aqueous or vitreous escpaes from the eye, the eye becomes extremely soft. Complications include infection, uveitis, retinal detachment, and glaucoma.

A cataractous lens should be removed at the time of initial repair or later. In persons under age 25 or 30, lens material in the traumatic cataract will often absorb almost completely over a period of months without surgery. A thin membrane may remain, in which case treatment with a YAG laser may be necessary to improve vision.

The lens material may clog the anterior chamber angle, interfering with aqueous outflow and causing glaucoma. If glaucoma occurs and cannot be controlled medically, the lens must be removed without delay. Most eye surgeon now use a combination of irrigation and aspiration for traumatic cataract. If the posterior capsule is ruptured, anterior vitrectomy is required.

Lacerations

Note: Tetanus prophylaxis is indicated whenever penetrating eye or lid injury occurs.

Lacerations are usually caused by sharp objects (knives, scissors, a projecting portion of the dashboard of an automobile, etc). Such in juries are treated in different ways depending upon whether there is prolapse of tissue.

Lacerations Without Prolapse of Tissue

If the eyeball has been penetrated anteriorly without gross evidence of prolapse of intraocular contents, and if the wound is clean and grossly free from contamination, it can usually be repaired by direct interrupted sutures of fine silk or nylon or by absorbable sutures.

Lacerations With Prolapse

If only a small portion of the iris prolapses through the wound, it should be grasped with a forceps and excised at the level of the wound lip. In any type of uveal tissue injury, the possibility of sympathetic ophthalmia must be kept in mind during the period of recovery.

If the wound has been extensive and loss of contents has been great enough that the prognosis for useful function is hopeless, evisceration or enucleation is indicated as the primary surgical procedure.

Lacerations of the Lids

Many lacerations of the lids do not involve the margins and may be sutured in the same way as other lacerations of the skin. If the margin of the eyelid is involved, special technics are required to prevent notching of the lid margin.

Rarely, extreme edema of the tissue prevents apposition of the wound for primary closure, and the repair must be delayed (secondary repair) until the edema has subsided. Local debridement and irrigation, with use of antibiotics, should be carried out until it is possible to approximate the edges of the wound.

Lacerations of the lids near the inner canthus frequently involve the canaliculi. If these are not repaired, permanent strictures with epiphora will result. Small polyethylene tubes are usually placed in the canaliculi at the time of repair and left in place until healing occurs.

Canaliculus repair should be performed immediately, since later repair is much more difficult.

Nonpenetrating Injuries of the Eyeball

Corneal Abrasions

Abrasions of the cornea do not require surgical treatment. The wound should be cleansed of imbedded foreign material. To facilitate the examination, pain can be relieved by instillation of a local anesthetic such as 0.5% tetracaine (Pontocaine) solution, but routine instillation of a local anesthetic by the patient must not be permitted, since it may delay the diagnosis of complications and is conductive to further injury. Antibiotic ointment, eg, polymyxin B-bacitracin (Polysporin), helps prevent bacterial infection. An eye bandage applied with firm pressure lessens discomfort and promotes healing (in 48-72 hours) by preventing movement of the lids over the injured area. The dressing should be changed daily until healing is complete.

Corneal abrasions cause pain severe enough to require strong analgesics. If not treated properly they may lead to recurrent corneal erosion.

Contusions

Contusions of the eyeball and its surrounding tissues are commonly produced by blunt trauma. The results of such injury are variable and are often not obvious upon superficial examination. Careful study and adequate follow-up are indicated. The possible results of contusion injury are hemorrhage and swelling of the eyelids (ecchymosis, "black eye"), subconjunctival hemorrhages, edema or rupture of the cornea, hemorrhage into the anterior chamber (hyphema), rupture of the root of the iris (iridodialysis), traumatic paralysis of the pupil (mydriasis), paralysis or spasm of the muscles of accommodation, traumatic cataract, dislocation of the lens (subluxation and luxation), vitreous hemorrhage, retinal hemorrhage and retinal edema (most common in the macular area, called commotio retinae, or Berlin's traumatic edema), detachment of the retina, rupture of the choroid posteriorly, and optic nerve injury.

Many of these injuries cannot be seen on casual external observation, and some may not develop for days or weeks following the injury. Careful follow-up of intraocular pressure is necessary if the iris root is torn and damage to the trabecular meshwork has occurred. Careful examination of the retinal periphery with indirect ophthalmoscopy is needed to ensure that retinal tears are not present. After a severe ocular contusion, the patient should be counseled regarding the symptoms of retinal detachment, since detachment may occur months or years later in a traumatized eye.

Except for cases involving rupture of the eyeball, intraocular hemorrhage, or retinal detachment, most ocular contusions do not require immediate definitive treatment.

Rupture of the Eyeball

Rupture of the eyeball may be direct, at the site of injury, or may occur indirectly as a result of sudden increase in intraocular pressure, causing the wall of the eyeball to tear at one of the weaker points. Common sites of rupture are the limbus and the area around the optic nerve. Anterior ruptures can be repaired surgically by interrupted sutures if the intraocular contents have not become deranged in a manner that will prohibit useful function of the eye. If this is the case, evisceration or enucleation is indicated. If either of these procedures is required, implantation of a plastic sphere is useful as a space-filler and to aid in movement of an artificial eye.

Chemical Conjunctivitis & Keratitis

Chemical burns are best treated by thorough irrigation of the eyes with saline solution or water immediately after exposure. It is wise not to try to neutralize an acid or alkaly by using its chemical counterpart, as the heat generated by the reaction may cause further damage. If the chemical irritant is an alkali, the irrigation should be continued longer, since alkalies are not precipitated by proteins of the eye, as acids are, but tend to linger in the tissues, producing further damage long after exposure. A local anesthetic solution is instilled before the irrigation in order to relieve pain. The pupil should be dilated with sterile 2% atropine or 0.25% scopolamine solution to prevent synechia formation.

Corticosteroid ointment is placed in the affected eye often enough to relieve pain and irritation. The frequency of instillation depends upon the severity of the burn. The patient must be watched carefully for such complications as symblepharon, corneal scarring, closure of the puncta, and secondary infection.

Ultraviolet Keratitis (Actinic Keratitis)

Ultraviolet burns of the cornea are usually caused by exposure to a welding arc or to the sun and snow when skiing ("snow blindness"). There are no immediate symptoms, but about 12 hours later the patient complains of agonizing pain and severe photophobia. Slitlamp examination after instillation of sterile fluorescein shows diffuse punctate staining of both corneas in the exposed areas.

Treatment consists of topical corticosteroids, systemic analgesics, and sedatives as indicated. All patients recover within 24-48 hours without complications.

Orbital Injury

There are many types of injury to the bony orbit. Only blowout fracture will be considered here.

Blowout Fracture

Isolated orbital floor or "blowout" fracture, without concurrent orbital rim fracture, may follow blunt injury to the eye. Orbital contents herniate into the maxillary sinus, and the inferior rectus or inferior oblique muscle may become incarcerated at the fracture site.

Signs and symptoms are pain and nausea at the time of injury and diplopia on looking up or down. Diplopia may occur immediately or within a few days. Enophthalmos may not be present until the orbital reaction clears. The fracture site is best demonstrated by antral roof deformation on Waters' view x-rays or laminagrams. There is limited movement of the eye even with forced ductions.

If the fracture is large or the muscle imbalance is great, prompt surgical reduction is imperative. If the vertical imbalance is small, surgery can be delayed a few days or weeks as long as steady improvement is noted. The orbital floor fracture is most commonly repaired using the Caldwell-Luc approach.

Infections of the Eye

1. Bacterial Corneal Ulcer

Corneal ulcers constitute a medical emergency. The typical gray, necrotic corneal ulcer is preceded by trauma, usually a corneal foreign body. The eye is red, with lacrimation and conjunctival discharge, and the patient complains of blurred vision, pain, and photophobia.

Prompt treatment is essential to prevent complications. Otherwise, visual impairment may occur as a result of corneal scarring or intraocular infection.

Corneal ulcers may result from many causes, including allergic disorders and bacterial, viral, and fungal infections. Only the most serious types will be discussed here.

Pneumococcal ("Acute Serpiginous") Ulcer

Streptococcus pneumoniae is the commonest bacterial cause of corneal ulcer. The early ulcer is gray and fairly well circumscribed.

Since the pneumococcus is sensitive to both sulfonamides and antibiotics, local therapy is usually effective. If untreated, the cornea may perforate. Concurrent dacryocystitis, if present, should also be treated.

Pseudomonas Ulcer

A less common but much more virulent cause of corneal ulcer is *Pseudomonas aeruginosa.* The ulceration characteristically starts in a traumatized area and spreads rapidly, frequently causing perforation of the cornea and loss of the eye within 48 hours. *P aeruginosa* usually produces a pathognomonic bluish-green pigment.

Early diagnosis and vigorous treatment with topical polymyxin and gentamicin are essential if the eye is to be saved.

2. Herpes Simplex Keratitis

Corneal ulceration caused by herpes simplex virus is more common than any type of bacterial corneal ulcer. It is almost always unilateral and may affect any age group of either sex. It is often preceded by facial "cold sores" and upper respiratory tract infection with fever.

The commonest finding is of one or more dendritic ulcers (superficial branching gray areas) on the corneal surface. These are composed of clear vesicles in the corneal epithelium; when the vesicles rupture, the area stains green with fluorescein. Although the dendritic figure is its most characteristic manifestation, herpes simplex keratitis may appear in a number of other configurations.

Treatment consists of mechanical removal of the virus-containing corneal epithelium without disturbing Bowman's membrane or the corneal stroma. This is best done by an ophthalmologist.

Frequent instillation of acyclovir ointment is used by many ophthalmologists in addition to or instead of removing the corneal epithelium. The drug is still investigational for ophthalmologic use. Alternative antiviral agents are idoxuridine, trifluridine, and vidarabine.

3. Acute Iritis (Endogenous Nongranulating Uveitis)

Nongranulomatous uveitis is primarily an anterior autoimmune noninfectious disease, but it may occasionally be associated with ankylosing spondylitis or Crohn's disease. The iris and ciliary body are primarily affected, but occasional foci are found in the choroid.

The onset is acute, with marked pain, redness, photophobia, and blurred vision. A circumcorneal flush, caused by dilated limbal blood vessels, is present. Fine white keratic precipitates (KPs) on the posterior surface of the cornea can be seen with the slitlamp or with a loupe. The pupil is small, and there may be a collection of fibrin with cells in the anterior chamber. If posterior synechiae are present, the pupil will be irregular and the light reflex will be absent.

Local corticosteroid therapy tends to shorten the course. Warm compresses will decrease pain. Atropine, 2%, 2 drops in the affected eye, will prevent posterior synechia formation and alleviate photophobia. The frequency of instillation will depend upon the severity of the symptoms and may vary from once a day to several times a day. Recurrences are common, but the prognosis is good.

4. Orbital Cellulitis

Orbital cellulitis is manifested by an abrupt onset of swelling and redness of the lids, often accompanied by proptosis. Fever is common. It is usually caused by a pyogenic organism. Immediate treatment with systemic antibiotics is indicated to prevent brain abscess or rapid increase in the orbital pressure, either of which may interfere with blood supply to the eye. The response to antibiotics is usually excellent, but surgical drainage may be required if an abscess forms.

Diplopia

Double vision is due to muscle imbalance or to paralysis of an extraocular muscle as a result of inflammation, hemorrhage, trauma, tumefaction, congenital defect, or infection of the third, fourth, or sixth nerve. The sixth nerve is most commonly affected.

Angle-Closure (Acute) Glaucoma

Acute glaucoma can occur only with the closure of a preexisting narrow anterior chamber angle. If the pupil dilates spontaneously or is dilated with a mydriatic or cycloplegic, the angle will close and an attack of acute glaucoma is precipitated; for this reason, it is a wise precaution to estimate the depth of the anterior chamber angle before instilling these drugs. About 1% of people over age 35 have narrow anterior chamber angles, but many of these never develop acute glaucoma.

A quiet eye with a narrow anterior chamber angle may convert spontaneously to angle-closure glaucoma. The process can be precipitated by anything that will dilate the pupil, eg, indiscriminate use of mydriatics or cycloplegics by the patient or the physician. The cycloplegic (anticholinergic) can be administered in the form of eyedrops or systemically, eg, by the anesthesiologist ordering scopolamine or atropine before a general surgical procedure. Increased circulating epinephrine in times of stress can also dilate the pupil and cause acute glaucoma. Sitting in a darkened movie theater can have the same effect.

It should be emphasized that about 95% of patients with glaucoma have the open angle (chronic) type and are in no danger of converting to angle-closure glaucoma. It is particularly important to understand this when doing a general surgical procedure on a patient with open angle glaucoma. It is quite safe to premedicate with scopolamine, atropine, or other anticholinergic drugs. Acute glaucoma is usually precipitated by these drugs in patients with narrow anterior chamber angles without a history of glaucoma.

Patients with acute glaucoma seek treatment immediately because of extreme pain and blurring of vision. The eye is red, the cornea is steamy, and the pupil is moderately dilated and does not react to light. Intraocular pressure is elevated.

Acute glaucoma must be differentiated from conjunctivitis and acute iritis.

Laser peripheral iridectomy within 12-28 hours after onset of symptoms will usually result in permanent cure. Untreated acute glaucoma results in complete and permanent blindness within 2-5 days after onset of symptoms. Before surgery, the intraocular pressure

must be lowered by mitotics instilled locally and osmotic agents and carbonic anhydrase inhibitors administered systemically. The fellow eye should undergo prophylactic laser iridectomy.

Occlusion of the Central Retinal Artery

This uncommon unilateral disorder (the ocular equivalent of coronary thrombosis) occurs only in older people. Occlusion may be the result of thrombin formation on a preexisting plaque or may be due to subintimal hemorrhage with resultant displacement of the plaque. Spasm of the artery is often a complicating factor. Emboli may occur. There is sudden, painless, complete loss of vision in the affected eye. Ophthalmoscopic examination soon after onset reveals segmentation of the blood in the veins and arterioles as a result of absence of retinal blood flow. The disk is pale, and there is marked retinal edema in the posterior pole associated with a cherry-red spot in the macula. If the occlusion is complete, total light perception is permanently lost, and the pupil will not react directly to light (although the consensual pupillary light reflex is normal).

If the patient is seen within 30-60 minutes after onset, an effort should be made to restore blood flow through the obstructed artery by vigorous massage of the eyeball or paracentesis of the anterior chamber. Systemic administration of a rapid-acting vasodilator, eg, tolazoline (Priscoline), 75 mg intravenously, has not proved to be useful.

Because the retina can survive hypoxia longer than brain tissue, the prognosis is not hopeless if treatment is instituted promptly. If treatmed is delayed for over 30-60 minutes, the visual prognosis is all but hopeless, and the value of any type of treatment is questionable. Efforts should be made to locate the source of emboli or treat underlying hypertension, which is often responsible for atherosclerotic disease.

Retinal Detachment

Essentials of Diagnosis

- Blurred vision in one eye becoming progressively worse. ("A curtain came down over my eye.")

- No pain or redness.

- Visible detachment ophthalmoscopically.

General Considerations

Detachment of the retina is usually spontaneous but may be secondary to trauma. Spontaneous detachment occurs most frequently in persons over 50 years old. Predisposing causes such as aphakia and myopia are commonly present.

Clinical Findings

As soon as the retina is torn, a transudate from the choroidal vessels, mixed with vitreous, combines with abnormal vitreous traction on the retina and the force of gravity to strip the retina from the choroid. The superior temporal area is the most common site of detachment. The area of detachment rapidly increases, causing correspondingly progressive visual loss. Central vision remains intact until the macula becomes detached.

On ophthalmoscopic examination, the retina is seen hanging in the vitreous like a gray cloud. One or more retinal tears, usually crescent-shaped and red or orange, are always present and can be seen by an experienced examiner.

Differential Diagnosis

Sudden partial loss of vision in one eye may also be due to vitreous hemorrhage or thrombosis of the central retinal vein or one of its branches.

Treatment

All cases of retinal detachment should be referred immediately to an ophthalmologist. If the patient must be transported a long distance, the head should be positioned so that the detached portion of the retina will recede with the aid of gravity. For example, a patient with a superior temporal retinal detachment in the right eye should lie supine with the head turned to the right. Position is less important for a short trip.

Retinal detachment is a true emergency if the macula is threatened. If the macula is detached, permanent loss of central vision usually occurs even though the retina is eventually successfully reattached by surgery.

Treatment consists of closure of the retinal tears by cryosurgery or scleral buckling (or both). This produces an inflammatory reaction that causes the retina to adhere to the choroid. Photocoagulation is of value in a limited number of cases of minimal detachment. It consists of focussing a strong light from various sources through the pupil to create an inflammatory adhesion between the choroid and the retina.

The main use of photocoagulator and laser is in the prevention of detachment by sealing small retinal tears before detachment occurs.

Prognosis

About 85% of uncomplicated cases can be cured with one operation; an additional 10% will need repeated operations; the remainder never reattach. The prognosis is worse if the macula is detached, if there are many vitreous strands, or if the detachment is of long duration. Without treatment, retinal detachment almost always becomes total in 1-6 months. Spontaneous detachments are ultimately bilateral in 20-25% of cases.

Vitreous Hemorrhage

Hemorrhage into the vitreous is an uncommon but serious disorder. It is usually due to traumatic rupture of a retinal vessel but may be related to diabetes mellitus, hypertension, perivasculitis, blood dyscrasia, or retinal detachment. One or both eyes may be affected, depending on the cause.

There is a sudden loss of vision in the affected eye. The fundus reflection is absent, but the anterior chamber, cornea, and lens are clear.

Since retinal detachment is often the cause of vitreous hemorrhage in nondiabetic patients, ultrasound examination should be performed in all patients with vitreous hemorrhage where the retina cannot be visualized. A vitrectomy may be necessary if blood prevents visualization. In diabetic patients with decreased vision caused by vitreous hemorrhage, vitrectomy may be effective in restoring vision.

Hordeolum

Hordeolum is a common staphylococcal abscess that is characterized by a localized red, swollen, acutely tender area on the upper or lower lid. Internal hordeolum is a meibomian gland abscess that points to the skin or to the conjunctival side of the lid; external hordeolum or sty (infection of the glands of Moll or Zeis) is smaller and on the margin.

The chief symptom is pain. The severity of the pain is directly related to the amount of swelling.

Warm compresses are helpful. Incision is indicated if resolution does not begin within 48 hours. An antibiotic or sulfonamide instilled into the conjunctival sac every 3 hours is beneficial during the acute stage. Without treatment, internal hordeolum may lead to generalized cellulitis of the lid.

Chalazion

Chalazion is a common granulomatous inflammation of a meibomian gland characterized by a hard, nontender swelling on the upper or lower lid. It may be preceded by a sty. The majority point toward the conjunctival side.

If the chalazion is large enough to impress the cornea, vision will be distorted. The conjunctiva in the region of the chalazion is red and elevated.

Treatment consists of excision by an ophthalmologist.

Dacryocystitis

Dacrocystitis is a common infection of the lacrimal sac. It may be acute or chronic and occurs most often in infants and in persons over 40. It is usually unilateral and is always secondary to obstruction of the nasolacrimal duct.

Adult Dacryocystitis

The cause of obstruction is usually unknown, but a history of trauma to the nose may be obtained. In acute dacryocystitis, the usual infectious organism is *Staphylococcus aureus* or the beta-hemolytic *Streptococcus;* in chronic dacryocystitis, *Streptococcus pneumonia. Haemophilus influenzae* is not a cause of dacryocystitis in adults. Mixed infections do not occur.

Acute dacryocystitis is characterized by pain, swelling, tenderness, and redness in the tear sac area; pus may be expressed. In chronic dacryocystitis, tearing and discharge are the principal signs. Mucus or pus may be expressed from the tear sac.

Acute dacryocystitis responds well to antibiotic therapy, but recurrences are common if the obstruction is not surgically removed. The chronic form may be kept latent by using antibiotic eye drops, but relief of the obstruction is the only cure.

Infantile Dacryocystitis

H influenzae is the only organism that causes infantile dacryocystitis.

Normally, the nasolacrimal ducts open spontaneously during the first month of life. Occasionally, one of the ducts fails to canalize, and a secondary pneumococcal dacryocystitis develops. When this happens, forceful massage of the tear sac is indicated, and antibiotic or sulfonamide drops should be instilled in the conjunctival sac 4-5 times daily. If this is not successful after a few weeks, probing of the nasolacrimal duct is indicated regardless of the infant's age. When probing the nasolacrimal duct, the probe should be passed through the upper canaliculus; it is easier to do and avoids possible trauma to the lower canaliculus, the more important tear drainage canal.

Strabismus

Any child under age 7 with strabismus should be seen without delay to prevent or to threat the beginning of amblyopia. In adults, the sudden onset of strabismus usually follows head trauma, intracranial hemorrhage, or brain tumor.

About 5% of children are born with or develop a malfunction of binocular coordination known as strabismus. In descending order of frequency, the eyes may deviate inward (esotropia), outward (exotropia), upward (hypertropia), or downward (hypotropia). The cause is not known, but fusion is lacking in almost all cases. If a child is born with straight eyes but has inherited "weak fusion", strabismus may develop.

Clinical Findings

Children with frank strabismus first develop diplopia. They soon learn to suppress the image from the deviating eye, and the vision in that eye therefore fails to develop. This is the first stage of amblyopia ex anopsia.

Most cases of strabismus are obvious, but if the angle of deviation is small or if the strabismus is intermittent, the diagnosis may be obscure. The best method for detecting strabismus is to direct a light toward each pupil from a distance of 1-2 feet. If the corneal reflection is seen in the center of each pupil, the eyes can be presumed to be straight at that moment.

As a further diagnostic test ("cover test"), cover the right eye with an opaque object ("cover") and instruct the patient to watch the examining light with the left eye. If fusion is weak, covering the right eye will disturb the fusion process sufficiently to allow the right eye to deviate, and this can be observed behind the cover. The right eye may swing back into alignment when the cover is removed (phoria). In obvious strabismus, the covered eye will maintain the deviated position after the cover is removed (tropia). Ask the patient to follow the examining light with both eyes to the right, left, up, and down to rule out extraocular muscle paralysis. If there is a history of deviation but it cannot be demonstrated, the patient should be reexamined in a few months.

Prevention

Amblyopia due to strabismus can be detected by routine visual acuity examination of all preschool children. Visual acuity testing is best done with an illiterate E card close to the fourth birthday by the child's mother but is often performed in the physician's office as a routine procedure. Treatment by occlusion of the good eye is simple and effective.

The prevention of blindness by these simple diagnostic and treatment procedures is one of the most rewarding experiences in medical practice.

Treatment

The objectives in the treatment of strabismus are (1) good visual acuity in each eye; (2) straight eyes, for cosmetic purposes; and (3) coordinate function of both eyes.

The best time to initiate treatment is around age 6 months. If treatment is delayed beyond this time, the child will favor the straight eye and suppress the image in the other eye; this results in failure of visual development (amblyopia) in the deviating eye.

If the child is under age 7 years and has an amblyopic eye, the amblyopia can be cured by occluding the good eye. At age 1 year, patching may be successful within 1 week; at 6 years, it may take a year to achieve the same result, ie, to equalize the visual acuity in both eyes. Surgery is usually perfored after the visual acuity has been equalized.

Prognosis

The prognosis is more favorable for strabismus which has its onset at age 1-4 than for strabismus which is present at birth; better for divergent (outward deviation) than for convergent strabismus; and better for intermittent than for constant strabismus.

Other Diseases of the Eye

Ocular Tumors

Many tumors of the ocular adnexa can be completely excised if they are diagnosed in an early stage. Malignant intraocular tumors (other than iris tumors) nearly always require enucleation. The 2 most common intraocular tumors are retinoblastoma and malignant melanoma.

Optic Nerve Pathology

Optic nerve disorders such as optic neuritis, optic atrophy, and papilledema are quite serious and may indicate accompanying intracranial or systemic disease. Neurologic examination is indicated as well as ophthlamologic examination.

Sympathetic Ophthalmia (Sympathetic Uveitis)

Sympathetic ophthalmia is a rare, severe bilateral granulomatous uveitis. The cause is not known, but the disease may occur at any time from 1 week to many years after a penetrating injury near the ciliary body. The injured (exciting) eye becomes inflamed first and the fellow (symphatizing) eye second. Symptoms and signs include blurred vision with light sensitivity and redness.

The best treatment of sympathetic ophthalmia is prevention by removing the damaged eye. Any severely injured eye (eg, one with perforation of the sclera and ciliary body, with loss of vitreous and retinal damage) should be enucleated within 1 week after the injury. Every effort should be made to secure the patient's reasoned consent to the operation. In established cases of sympathetic ophthalmia, systemic corticosteroid therapy may be helpful. Without treatment, the disease progresses gradually to bilateral blindness.

Chronic Glaucoma

Antiglaucoma therapy should be instituted without delay in order to decrease the intraocular pressure and preserve the remaining visual field.

Unilateral Exophthalmos or Recent Origin

The most common cause of bilateral exophthalmos is hyperthyroidism, although exophthalmos may also appear after thyroidectomy. Unilateral exophthalmos may be due to an orbital tumor, cavernous sinus thrombosis, or atrioventricular shunt from the internal carotid artery to the cavernous sinus. Some of these disorders are treatable.

Technis Used in the Treatment of Ocular Disorders

Instilling Medications

Place the patient in a chair with head tilted back, both eyes open, and looking up. Retract the lower lid slightly and instill 2 drops of liquid into the lower cul-de-sac. Have the patient look down while finger contact on the lower lid is maintained for a few seconds. The patient must not squeeze the eyes shut.

Ointments are instilled in the same general manner.

Self-Medications

The same techniques are used as described above, except that drops should usually be instilled with the patient lying down.

Eye Bandage

Most eye bandages should be applied firmly enough to hold the lid securely against the cornea. Gauze-covered cotton is usually sufficient. Tape is applied from the cheek to the forehead. If more pressure is desired, use 2 or 3 bandages. Black eye patches are difficult to sterilize and therefore are seldom used in modern medical practice.

Water Compresses

A clean towel or washcloth soaked in warm tap water is applied to the affected eye 2-4 times a day for 10-15 minutes. Standard procedure is to use warm compresses for infections and cold compresses for allergies.

Removal of a Superficial Corneal Foreign Body

Record the patient's visual acuity, if possible, and instill sterile local anesthetic drops. With the patient sitting or lying down, an assistant should direct a strong light into the eye so that the rays strike the cornea obliquely. Using either a loupe or a slitlamp, the physician locates the foreign body on the corneal surface. It may then be removed with a sterile wet cotton applicator or spud, with the lids held apart with the other hand to prevent blinking. An antibacterial ointment (eg, Polysporin) is instilled after the foreign body has been removed. It is preferable not to patch the eye, but the patient must be seen on the following day to make certain healing is under way.

Precautions in the Management of Ocular Disorders

Use of Local Anesthetics

Unsupervised self-administration of local anesthetics is dangerous because they delay healing and because the patient may further injure an anesthetized eye without knowing it.

Pupillary Dilation

Cycloplegics and mydriatics should be used with caution. Dilating the pupil can precipitate an attack of acute glaucoma if the patient has a narrow anterior chamber angle.

Local Corticosteroid Therapy

Repeated use of local corticosteroids presents several serious hazards: herpes simplex (dendritic) keratitis, fungal overgrowth, open angle glaucoma, and cataract. Furthermore, perforation of the cornea may occur when the corticosteroids are used for herpes simplex keratitis.

Contaminated Eye Medications

Ophthalmic solutions must be prepared with the same degree of care as fluids intended for intravenous administration.

Tetracaine (Pontocaine), proparacaine (Ophthaine, Ophthetic), physostigmine, and fluorescein are most likely to become contaminated. The most dangerous is fluorescein, as this solution is frequently contaminated with *Pseudomonas aeruginosa,* an organism that can rapidly destroy the eye. Sterile fluorescein filter paper strips are now available and are recommended in place of fluorescein solutions.

Plastic dropper bottles are popular, and solutions from these bottles are safe for use in uninjured eyes. Whether in plastic or glass containers, eye solutions should not be used for more than about 2 weeks after the bottle is first opened.

If the eye has been injured accidentally or by surgical trauma, it is critical to use sterile medications supplied in sterile, disposable, single use eyedropper units.

Fungal Overgrowth

Since antibiotics, like corticosteroids, favor the development of secondary fungal corneal infection when used over a prolonged period in bacterial corneal ulcers, sulfonamides should be used whenever they are adequate for the purpose.

Sensitization

A significant portion of a soluble substance instilled in the eye may pass into the bloodstream. An antibiotic instilled into the eye can sensitize the patient to that drug and cause a hypersensitivity reaction upon subsequent systemic administration.