

Eye Conditions

Authors: Dr Tze Foon Lai; Dr Johnny Wu; Dr Tim Henderson; Dr Wilfred Win Law
(Department of Ophthalmology, ASH)

Topic Reviewers: Prof Hugh Taylor; Kenna Bistani (RAN, Pine Creek); Bernard Egan (RAN, Bilman Clinic); Robyn Dixson (RAN, Yirrkala); Colin Watson (RAN, Nyirripi Clinic)

[Editor: In this section the authors have collated more detailed and expanded instructions on the appropriate care of common (or frightening, e.g. penetrating eye injury) eye conditions seen in remote community clinics.

The evidence for the specialist care of diabetic retinopathy, cataract and trachoma has been collated by the Centre for Eye Research Australia for the Commonwealth Department of Health and Ageing, Office for Aboriginal and Torres Strait Islander Health in Specialist Eye Health Guidelines for use in Aboriginal and Torres Strait Islander Populations. Cataract, Diabetic Retinopathy, Trachoma (Commonwealth of Australia, 2001).

Though these guidelines are written for specialists, they also offer a good review of the evidence for most aspects of primary clinical care of these conditions. Many aspects of trachoma control and primary prevention of cataract and diabetic retinopathy are in the realm of population-based public health measures, and hence beyond the scope of the CARPA STM.

Good management of systemic aspects of diabetes are very important in minimising diabetic retinopathy, this is covered in detail in the diabetes chapter.

Cataract is not specifically dealt with in the CARPA STM. However, using the protocols (including the 'older persons adult health check') will direct people with significant visual disability from cataract to specialist eye services where appropriate treatment should follow. Other protocols also cover the potentially modifiable risk factors for cataract such as smoking, diabetes, alcohol and the judicious use of medicines that can be a risk factor for cataract.)

History and examination

Using the E chart or the letter chart?

Visual acuity is the 'vital sign' in ophthalmology. It should be the first step in the examination and should be performed accurately. The only time visual acuity should not be obtained before treatment starts is in chemical injuries, when washing out the eye is the most important thing to do first.^{1,2}

1. Patient is seated at an appropriate distance depending on the chart used. The standard distance is 6 m (20 feet). It does not matter which chart is used, the size of the figures is what is important. The patient should be briefed precisely what is required of them to allow an accurate assessment of their vision.
2. Test the vision:

- i. Unaided or with glasses if worn for distance (driving/TV spectacles).
- ii. Using a pinhole if not able to see 6/6

Always test one eye at a time while covering the other effectively. If you ask the patient to use their hand make sure it is the palm of the hand, as they can inadvertently get a very good pinhole effect between their fingers of the eye that is supposed to be covered.

If patients are literate it is easier to have them call out the letters. If using an E chart ask the patient which way the three limbs of the E point (not the two white gaps between them, which point the opposite way).

3. Record the vision for each eye with and without the pinhole. Take the number written on the chart corresponding to the line the patient can see (this is written below or above each line of letters, depending on the chart), i.e. if the patient reaches the line numbered 24 and completes this line then take this number. For example:

RVA 6/24	with glasses	LVA 6/6
6/9	with p.h.	6/6

(6 = the distance from the chart in metres, 24 = the number of the line corresponding to the distance a person with 'normal' vision would be able to see this letter).

Tip: Children are usually easier to examine when they do not realise that a procedure is being done. Therefore do not miss the opportunity to have a good look at the child's eye whilst he or she is busy reading the letters initially.

For very young children, visual acuity can be assessed by observing whether they can pick up 'hundreds and thousands' cake decorations.⁶ Other tests such as letter matching or preferential looking for babies are also employed.

Using an ophthalmoscope

Whenever possible put dilating drops (Tropicamide 1% eye drops) in the eyes to be examined, only after checking pupils.

1. Switch on the ophthalmoscope and make sure it is working.
2. Turn all the dials to zero.
3. Darken the room.
4. Hold the ophthalmoscope at arm's length from the patient and keep the eyepiece right up against your own eye. Unless you have one eye with poor vision use the ophthalmoscope 'Right eye to Right eye and vice versa. (This avoids kissing the patient by mistake!) It also makes it easier for the patient to look straight ahead while being examined. For each eye check the red reflex from a distance and compare both eyes. The red reflex is a reflection of the light directed into the eye. If it is speckled, dull or totally absent, there is opacity interrupting the passage of light, which is usually caused by a cataract but can be due to other causes, e.g. vitreous haemorrhage.
5. Then approach from the temporal side of the eye aiming for the centre of the head. Find something that looks like a blood vessel and, while looking at it, turn the dial on the ophthalmoscope to bring it into

sharp focus. Then follow the vessels until you find the optic disc. Once at the disc look up and temporally along the vessels then follow the lower vessels temporally. Finally, look temporal to the disc into the central area of vision known as the macular. This is bright for patients but if they can tolerate it the best way of looking at the very central part of the retina is to ask the patient to look straight at the light.^{1,3} This usually causes the maximal pupil constriction, and dilating drops may be necessary to see any real detail.

Visual field testing

The visual fields map the peripheral extent of the visual world. Testing the visual field may give clues to the site of lesion and the diagnosis. The visual field may be tested in various ways.

Confrontation test⁶

1. The patient is seated directly opposite the examiner closing his/her eye on the same side.
2. An object, such as a red top pin, is then brought into view from the periphery and moved centrally. The patient is asked to indicate when he/she first sees the test object.
3. Each quadrant is tested and the location of the blind spot determined, careful use of this technique can allow assessment of the size of the blind spot.
4. The patient's field is thus compared to that of the examiner.

Crude testing of the field can be performed as follows⁶:

1. The patient is seated directly facing the examiner.
2. The patient is asked to cover one eye.
3. The examiner hold up both hands in front of the unoccluded eye, palms facing the patient, one on either side of the midline. Ask whether the two palms appear the same.
4. This can be useful in picking up a bitemporal hemianopia (patients may also miss the temporal letters on the Snellen chart when their visual acuity is measured).

The patient is asked to count the number of fingers in each quadrant of the visual field while presenting two fingers on one side and one on the other.

How to evert or double evert an eyelid
See CRANA Clinical Procedures Manual.

Eye injuries

Ocular injuries, however trivial, are a frightening experience for the patient [Editor: and practitioner!], who may have a deep-rooted fear of blindness. The incidence of injuries varies with the environment and protective measures taken.

Trauma to the eye is managed differently according to the kind of injury the eyeball has received. There are two broad categories, sharp (penetrating) and blunt (non-penetrating) trauma, but if the force hitting the eye is severe enough then both types of injuries will result, irrespective of the size of the object.^{4,10}

In the assessment of eye injuries, a thorough history should be taken to elicit:

- **Mode, extent of injury** to the eye. Record, in the patient's own words, the events leading up to the injury and the direction the blow came from, e.g. if a stick injury, did the stick hit the eye end on or from the side. This information helps you to judge the amount of direct trauma to the eyeball. For sharp trauma, establish whether the object that caused the injury was intact on withdrawal from the eye. If the object is available do not discard it or any of its pieces. If the penetrating object is still in the eye do not be tempted to remove it.
- Other **relevant injuries** (head/neck)
- **Tetanus prophylaxis**
- Past ocular, medical and drug history including hepatitis/HIV status

Visual acuity (VA) should always be measured despite the eye being sore and swollen shut. If the eye is too painful to open²:

- Explain the importance of obtaining a visual acuity to the patient and relatives/parents.
- Instil some topical local anaesthetic drops. Warn the patient this will sting for approximately 30 seconds. This is especially important for children and their parents.
- Allow the anaesthetic to work. Ask the patient if he or she can open the eye after about 30 seconds or so. The patient may need help if the lids are swollen. Even if it means reading letters one at a time between opening the eye and the patient 'having a break', a visual acuity must be obtained.
- It may be necessary to exert gentle pressure to reduce lid oedema or even to use lid retractors to hold tight swollen lids open enough to check vision.

Do not be put off by an unenthusiastic patient and always encourage a patient further. The letter on the next line down may appear blurred but if the patient can read them, then this blurring is of significance.

For children, if the child is frightened or crying, this should be performed with great care to avoid aggravating any pre-existing injury. If at any time during the examination suspicion of a ruptured globe develops, no further diagnostic examinations should be performed.⁹

Do not force the eyelids open if there is any chance of a penetrating injury. Beware deep lacerations of the upper lid. Do not forget the VA of the other eye need to be recorded.

VA not only is a good indication of the severity of a condition; it may also be required for medicolegal reasons^{1,2,6,7} and to give some idea of the potential vision that may be recovered if multiple procedures are contemplated to rehabilitate the eye.⁶

Examine the eye systemically from the outside in.²

- **Eyelids:** Look for **laceration, bruising and surgical emphysema**. (It classically feels like the sensation of walking on dry leaves. Once felt never forgotten). The presence of surgical emphysema suggests a bony fracture into a sinus.
- **Conjunctiva:** This is often red and swollen. Look for **foreign bodies and lacerations**. If the patient was (at the time of the injury) wearing a contact lens, this may have been displaced. It needs to be found and removed. If possible, ask the patient to remove the lens themselves (most patients are very good at this). If there is any suspicion of a full-thickness laceration of the eyeball do not try to

open the eyelids, and any contact lens can be left in place as it may be holding things in check.

- Cornea: Look for **abrasions** (these may look like tiny yellow (when fluorescein is used) dots or straight lines). Locate, and if possible remove, any foreign bodies.
- Anterior chamber: Look for **hyphaema**, a fluid level of blood that can look bright red, dark red or even black. Try to judge how much of the anterior chamber is filled by roughly assessing how far up the cornea the top of the fluid level is situated, e.g. an anterior chamber totally filled with blood has a 100% hyphaema, one in which the blood level only comes half the way up has a 50% hyphaema. Measuring the height in minutes is helpful to indicate if it is settling with time.
- Iris and pupil: Check the pupils for **size, shape and reactions to light**. The pupil may be fixed and dilated or be an abnormal shape. Any tear of the iris will cause a distortion of the pupil. Any abnormally shaped pupil may indicate a ruptured eyeball. If the iris is protruding through a wound, this is known as **iris prolapse**. **A peaked pupil is the characteristic sign of a perforating corneal wound** (best to draw it).
- The deeper layers of the eyeball will not be visible without specialised equipment, but if there is an ophthalmoscope available check for a red reflex.
- Exclude a blow-out fracture of the orbit by testing the patient's eye movements. Testing for numbness on the upper part of the patient's cheek in the area below the eye, with either cotton wool or gently with a disposable needle. Compare with other side. Any numbness on the side of the injury suggesting that there might be a fracture in the orbital floor.
- X-rays: The floor and medial wall of the orbit are the weakest. A blow-out fracture may be confirmed by asking for 'occipitontal' and 'A-P' views. These do not have to be performed as an emergency.

Practice points

Penetrating injuries of the eye can easily be missed because they may seal themselves, and the signs of abnormality are subtle. Any history of high velocity injury (particularly a hammer and chisel injury) should lead one strongly to suspect a penetrating injury and request orbital X-rays on up-gaze and down-gaze to identify metallic fragments.

Eyelid abrasions/lacerations

Treatment

Eyelid abrasions should be cleaned and debrided to prevent infection.

Tetanus immunisation should be updated for deep, dirty or devitalised wounds.

Prophylactic topical antibiotic ointment (Chlorsig) four times daily is indicated to prevent periorbital cellulitis.

Referral to ophthalmologist needed if:^{7,9}

- Full thickness cut or those involving the lid margin.
- The lacrimal ducts have been involved.
- There is any suspicion of a foreign body or penetrating injury.

Periorbital or ocular contusion

History

Usually minimal pain and visual disturbance. The degree of periorbital oedema varies.

Examination

Periorbital ecchymosis and oedema are found superiorly and/or inferiorly and may involve the contralateral eye.

Subconjunctival haemorrhage.

Full oculomotor movement with minimal pain.

Treatment

Plain X-ray films and/or computed tomography (CT) scanning of the orbit is needed if orbital fracture is suspected (subject to availability).^{1,6,7}

Ocular involvement: refer to ophthalmologist.

Non-ocular involved cases: reassurance, cool compress and non-urgent follow-up.

Orbital fractures

Blow-out fracture

History

Orbital floor ('blow-out') fractures are common when orbit is struck by objects larger than or of similar size to the orbital opening, such as a ball (especially squash ball), fist, or the dashboard of an automobile.^{9,10}

Examination

Limitation of upward gaze is the most apparent clinical sign.

Others include lower eyelid ecchymosis, nosebleed, orbital emphysema, and hypaesthesia of the ipsilateral cheek and upper lid (results from disruption of the infraorbital nerve as it traverses the orbital floor).

Medial wall fracture

History

This can be caused by the same forces that cause orbital floor fractures, also with blows to the bridge of the nose.^{9,10}

Examination

Orbital emphysema, epistaxis, a depressed bridge of the nose, and enophthalmos (sunken eye).

If there is nasal septal fracture or haematoma, the nose is deviated and nasal breathing can be impaired. The nasolacrimal drainage system can be disrupted, resulting in epiphora (tearing), lacrimal sac mucocele, or dacryostenosis (blockage of the tear-draining system).

Orbital roof fracture

It is less common than inferior or medial wall fractures but is more life threatening. Central nervous system involvement, pneumocephalus, or an intracranial foreign body should be considered in the assessment. Late complications include brain abscess and infectious meningitis.^{9,10}

Examination

- Cerebrospinal fluid leakage (rhinorrhoea). Rhinorrhoea is usually transient because dural tears are generally self-sealing.
- Superior and lateral subconjunctival haemorrhage.
- Optic nerve can be involved on this injury.

Treatment

- Refer to ophthalmologist.
- With orbital emphysema, expulsion of air into the orbit can dramatically increase with sneezing, coughing, or blowing the nose, all of which can cause a sudden rise in paranasal sinus pressure. Therefore, patients should be forewarned against blowing their nose, and coughing episodes should be vigorously treated with antitussives.^{1,7}

Subconjunctival haemorrhage

History

Blunt trauma, forceful sneezing and eye rubbing are usually reported. It can also occur spontaneously.

Examination

- Painless, bright red accumulation of blood, usually limited to one section or quadrant of the eye. The size of the clot and the area affected varies from patient to patient.
- For spontaneous subconjunctival haemorrhage, measure the patient's blood pressure⁷ and consider checking their coagulation status.
- Whenever a traumatic subconjunctival haemorrhage occurs, a more severe underlying ocular injury should be ruled out. Accumulated blood can hide a retained foreign body or an occult scleral laceration.

Treatment

In uncomplicated cases, reassurance alone is adequate.^{2,7} Patient should be told that the haemorrhage could take several weeks to completely disappear. It may appear to become larger over the first several days secondary to gravity and local spread under normal conjunctiva, turned yellow and gradually resorbs over two to three weeks.²

Conjunctival laceration

History

A conjunctival laceration is common when a sharp object, such as a fingernail or glass, strikes the eye.

Examination

It is essential to look for any deeper or more severe injury to the globe.

Treatment

Small isolated conjunctival laceration rarely requires treatment. Prophylactic antibiotic ointment or drops (e.g. Chlorsig) to prevent secondary infection.

Corneal abrasion

History

- A history of mild trauma to the eye, possibly caused by dust, fingernail scratch, tree branch, contact lens, make-up brush, or foreign body.
- Photophobia and involuntary lid closure (blepharospasm).
- Pain and foreign body sensation may be quite severe as when the epithelium is scratched, abraded, denuded it exposes the underlying epithelial basement layer and superficial corneal nerves.
- Vision is disturbed if the abrasion is in the central cornea.

Examination

- Instil a drop of topical local anaesthetic if the patient has severe spasm of the eyelids that prevent examination.
- Conjunctival hyperaemia, swollen eyelids and tearing.
- Pen light may reveal a surface irregularity.
- Linear corneal abrasions should alert the examiner a foreign body is possible.
- Slit lamp examination shows an epithelial defect but often a clear cornea. Minimal cellular reaction is seen in the anterior chamber. If corneal haze or moderate to severe flare and cells noted in the anterior segment, especially with an associated discharge, bacterial superinfection should be considered.
- Fluorescein dye is absorbed by areas devoid of epithelium and outlines the defect, best seen when illuminated by light with a blue filter.

Differential diagnosis¹

- Viral keratitis (herpes simplex or zoster), often with corneal dendrites.
- Corneal or conjunctival foreign body, especially under the conjunctiva of the upper lid, which can cause 'ice-skate track' abrasions as the foreign body is repeated swept linearly over the epithelium.
- Recurrent erosion, which is identical to primary epithelial defects but occurs long after the initial corneal trauma.
- Ultraviolet corneal injury (welder's flash).

Treatment

Foreign body's management described as below. The aims of treatment are ²:

- To prevent infection: Topical local antibiotic drops and/or ointment.
 [Editor: The issue of eye patching for corneal abrasion has been the subject of review. We found one systematic review of RCTs (seven, total of 550 patients) with meta-analysis (309 patients) (Flynn CA, Damico F, Smith G. Should we patch corneal abrasion? A meta-analysis. Journal of Family Practice, 1998; 47(4):264-70).]
 They included a variety of eye-pad options trialed in people over six years old. Abrasions related to infection or contact lenses were excluded. Primary outcome was healing at one to two days, secondary outcomes were symptoms and complications.
 There was no significant difference in the likelihood of healing at one or two days. There was no significant difference in the risk of complications. Two of seven studies reported faster healing in the no-patch group and five found no difference. Four of seven studies found no difference in pain between groups and two studies found statistically less pain in the no-patch group.
 Conclusion: Eye patching was not found to improve healing rates or reduce pain in patients with corneal abrasions. Given the theoretical harm from loss of binocular vision and possible increased pain or

infection risk, we recommend no-patching in treating corneal abrasion.]

- To relieve pain: Instil a cycloplegic (e.g. cyclopentolate 2%); give oral analgesia if necessary.

Practice points^{2,7}

- Although patients often request them, under no circumstances should topical local anaesthetics be prescribed or given to the patient. Not only do anaesthetics retard wound re-epithelisation, but the loss of the cornea's normal pain response predisposes the patient to a much more severe injury.
- Patients with a large central abrasion or high-risk abrasion should be referred to an ophthalmologist for management and follow-up care.
- A pressure patch is never used for more than 24 hours.
- If visual acuity is markedly decreased, more severe ocular injury needs to be ruled out.

Follow-up

All abrasions are monitored daily until they have completely resolved. If healing takes longer than two or three days, the patient should be referred to an ophthalmologist.

Corneal laceration

A corneal laceration can be either full or partial thickness. A full thickness laceration should be suspected when the results of the Seidel test are positive.^{11,12}

The test is performed by applying a dry strip of fluorescein over the wound or a drop of 1% fluorescein whilst observing the cornea at the slit lamp with the cobalt blue light. A slow leak of aqueous fluid (a positive test) is diagnosed with progressive dilution of the green fluorescein dye like a yellow-green waterfall.

Treatment

A shield should be placed over an eye suspected of having a corneal laceration until the patient can be examined by an ophthalmologist.⁸

A pressure patch should never be applied to a potentially open eye.⁸

Foreign body

History

It is not uncommon for the patient not to recall a foreign body having entered the eye.⁷ Pain and ocular foreign body sensation are the common symptoms, but a high-speed foreign body may cause no more than a transient irritation.

Examination

Slit lamp examination of the conjunctivae and cornea is preferable to definitely locate the particle and assess the degree of injury. The upper and lower lids must be everted to search for debris.^{1,7,12,20} Linear abrasions are highly suggestive of a foreign body embedded in the superior conjunctiva; discovery and subsequent removal of the particle provides prompt relief to the patient.²⁰

Treatment

Conjunctival foreign bodies are often swabbed away easily with a moist cotton-tipped applicator following local topical anaesthesia. If this fails, foreign bodies can be removed with an 18 or 25 gauge needle.²⁰ An 18-gauge needle has a wider diameter and is used for large foreign bodies, while a 25-gauge needle is narrow and useful for small particles. Manipulation of the needle may be stabilised by attaching it to a tuberculin syringe or inserting a cotton-tipped applicator in the plastic end.

If the foreign body is located in the cornea, the depth of the particle should be established before removal is attempted.²⁰ Foreign bodies that lie deep within the stroma occasionally have the potential to penetrate into the anterior chamber when manipulated. Referral to an ophthalmologist is advisable for removal of dangerously deep foreign bodies that pose the risk of an aqueous leak. It should be remembered that the cornea is a very tough structure and it is quite difficult to penetrate through it.

The needle is held tangential to the surface of the cornea and never pointed in a perpendicular direction toward the patient. Improper angulation could drive the sharp point into the cornea with abrupt patient movement, but if held correctly, it is unlikely that the force will be sufficient to penetrate through Bowman's membrane or the stroma.²⁰ The examiner's hand should be stabilised on the side of the patient's face or nose so that any movement goes with the patient.

Sterile irrigation can be used to dislodge superficial foreign bodies from the ocular surface. Irrigating solution should not be pointed directly at the foreign body but rather at a slight angle since pressure exerted from the irrigation stream straight onto the particle could embed it more deeply into the cornea.

Not all foreign bodies require removal and, in fact, deeply embedded, non-toxic foreign bodies such as glass may best be left in situ rather than subject the eye to additional scarring by extraction. Such particles should be lodged well below the corneal surface, allowing the epithelium to heal over them.²⁰

Following removal, cyclopentolate 2% and antibiotic ointment (Chloramphenicol) should be instilled into the eye followed by firm double padding. Padding protects the eye whilst still anaesthetised but may be removed after two hours if the patient prefers.

Follow-up

Patients treated for corneal foreign bodies should be examined the next day to evaluate the level of healing and detect signs of infection. Foreign bodies from potentially contaminated substances – such as vegetative matter – deserve careful follow-up, since introduction of micro-organisms at the time of the injury could precipitate corneal ulceration.

Practice point

Any foreign body on or close to the visual axis should be left for removal by an ophthalmologist.

Iron-containing foreign bodies deserve special consideration. Within a few hours of the injury, the iron partially decays, and rust stains the adjacent epithelial cells as well as Bowman's membrane.¹³ The rust ring must be removed as it will act as a continued source of irritation, delays healing process and can permanently stain the cornea.

Wooden splinters are particularly dangerous as they may easily penetrate the eye and cause severe infection.¹³

Fluorescein stain is a useful diagnostic adjunct to the evaluation of a patient with anterior segment trauma; however, fluorescein instillation should follow examination of the anterior segment, since subtle flare may be masked by fluorescein in the tear film. As the fluorescein stain pools within zones of corneal disruption, the extent of the corneal injury from the foreign body is highlighted. Additionally, fluorescein is useful in the detection of perforations of the globe, because aqueous leakage through the wound will be observed as a bright green stream and then washed away. This percolation of aqueous through the injury site is known as Seidel's sign.²⁰

Patients who have been hammering/chiselling/ drilling/grinding will need an X-ray to exclude the presence of an intra-ocular foreign body. X-rays are very useful but not conclusive, and only useful if they have been taken with eyes looking up and down. Each X-ray department has its own protocol on which X-ray views should be taken. Be sure to write 'to exclude an intra-ocular foreign body' on the form, and ask for 'non-screen film', because this has fewer speckles (which may be misinterpreted as intra-ocular foreign bodies) on it.

Globe rupture/penetrating injuries

Severe trauma may result in globe rupture, gross lid and conjunctival swelling, sometimes hyphema, restricted eye movement and ocular contents may be visible outside the eye.

Treatment

In situations where a penetrating foreign body is visible it should not be removed, as intra-ocular contents may be lost in the process.

Initial treatment includes loose fitting shield (Cartella shield or plastic cup cut in half) over the injured eye, systemic antibiotics (e.g. cephazolin 1 g three times a day), anti-emetic, analgesias, and intravenous fluids as necessary.^{2,8} Keep patient nil by mouth and urgent referral to an eye care facility is mandatory.⁸ Transport should be smooth and avoid depressurisation (please refer to RFDS guideline).

[Editor: Previous editions of the CARPA STM had specifically recommended pethidine over morphine for pain relief in penetrating eye injury. 'The principle is to avoid opiates if possible in penetrating eye injury because of the theoretical possibility of retracting the iris when it is the only thing preventing collapse of the eyeball. The risk of posterior synechiae is of minor importance' (communication Tim Henderson ASH ophthalmologist). An enquiry through the National Prescribing Service revealed; The evidence for pethidine having a lesser pupillary constriction effect is weak¹, though the pupillary effect of morphine may last longer.² The emetic effects are likely to be similar for equi-analgesic doses, and an anti-emetic would be given in this circumstance anyhow.

Our recommendation is that if an opioid is needed, then Tramadol or morphine can be used.

1. Ghoneim MM, et al. Comparison of four opioid analgesics as supplements to nitrous oxide anesthesia. *Anesth Analg* 1984; 63:405-12.

2. Zachny JP, et al. A dose-response analysis of the subjective, psychomotor and physiological effects of intravenous morphine in healthy volunteers. *J Pharmacol Exp Ther* 1994; 268:1-9.]

Injuries to the iris

History

Blunt eye contusion can injure the iris sphincter muscle, resulting in pupillary constriction (traumatic miosis) during the first several hours, followed by dilatation (traumatic mydriasis).

Patient complains of pain, photophobia, and asymmetry in pupil size. An accommodative spasm and paralysis may be associated, resulting in blurred vision and difficulty with near tasks.

Examination

Hyphema, perilimbal conjunctival injection, anisocoria, and sluggish pupil response

Treatment

The management of traumatic hyphema is variable and controversial.⁹ There is no consensus as to whether patients should be at strict bed rest or allowed limited ambulation or whether they can watch television or read. There is also no agreement as to the efficacy of hospitalisation, ocular occlusion, patching of the traumatised eye, cyclopegics, topical corticosteroids, and antifibrinolytic agents.

Accepted practice is reduced activity, bed rest for 72 hours and topical steroids.

Injuries to the lens

History

Trauma is the most common cause of dislocation of the ocular lens. Other causes include congenital dislocation, systemic syndrome (e.g. Marfan, homocystinuria), inflammation, and congenital glaucoma with buphthalmos.^{3,4,9}

Treatment

A non-cataractous, dislocated lens may be stable and asymptomatic for years. Children and their families, however, should be forewarned of the symptoms of pupillary block glaucoma and be advised to wear eye protection for sports and hazardous labour.⁹

Surgical considerations for removal of a dislocated lens include pupillary block glaucoma, corneal touch, inflammation, and decreased vision uncorrected by other means.^{9,10}

Traumatic retrobulbar haemorrhage

History

- Significant sharp or blunt trauma to the globe/peri-orbital region.²
- A markedly swollen red eye, painful, decreased vision.

Examination

- Proptosis, eyelid and peri-orbital ecchymosis.
- Diffuse and massive subconjunctival haemorrhage and chemosis. The posterior border of the subconjunctival blood cannot be visualised.
- The pupils and extra-ocular motility are evaluated carefully. The presence or development of a relative afferent pupillary defect may

indicate optic nerve injury caused by compression or a traumatic optic neuropathy.

- Markedly elevated intra-ocular pressure; significant resistance on repulsion of the globe can be found and the peri-ocular area is tense.
- Extra-ocular movements may be limited.
- Intracranial penetration should be suspected in cases in which retrobulbar haemorrhage resulted from a penetrating injury.

Differential diagnosis ²

- Orbital cellulitis, which often can be ruled out by the patient's history.
- Orbital fracture.
- Carotid-cavernous fistula, which is possible after severe head trauma.
- CT scan may be useful (if available).

Treatment

For suspected retrobulbar haemorrhage, patients should be referred to an ophthalmologist urgently. Emergency lateral canthotomy and inferior cantholysis can be performed by those who are experienced in this procedure.

Scleral laceration or rupture

History

- Usually, sharp objects or missiles (e.g. metal-on-metal projectile injury, broken glass, knife wound, and bullet).
- Severe blunt trauma (e.g. from a fist, bottle, or club).
- Red eye, pain, decreased vision, and possible inadvertent lid closure (blepharospasm).

Examination

- A defect is noted in the conjunctiva or sclera, with or without subconjunctival haemorrhage.
- Marked haemorrhagic chemosis (swelling) may obscure the underlying scleral injury.
- Uveal or vitreous prolapse occurs through the scleral wound, which may appear as a brownish discolouration beneath the conjunctiva, sometimes mistaken for blood.
- Pupil may be abnormally shaped (e.g. peaked).
- The intra-ocular pressure is low in most cases. Less commonly, the intra-ocular pressure is normal or elevated.
- The anterior chamber (cornea to iris) depth may be increased making it look 'deep'.

Differential diagnosis^{1,2}

- Conjunctival laceration without scleral injury.
- Intra-ocular foreign body.
- Clear or haemorrhagic chemosis without obvious scleral injury.

Treatment

- If globe rupture is suspected, the eye is shielded and the patient immediately referred to an ophthalmologist. No patching is used. A light pad may be necessary to absorb any discharge or bleeding.
- In cases of partial-thickness lacerations of the sclera, the patient is referred to an ophthalmologist for evaluation and possible surgical repair.

- In cases of suspected or confirmed full-thickness lacerations, intravenous antibiotics are administered and the patient is immediately referred for ophthalmological evaluation and surgical repair.
- The patient's tetanus status should be updated.

Follow-up

The focus and frequency of follow-up vary depending on the extent of the injury

Traumatic optic neuropathy

History

A history of traumatic injury to the globe or orbit (causing direct injury of the optic nerve) or to the forehead or temporal region (causing indirect injury of the optic nerve) is reported. Optic nerve injury manifests as a reduction in visual acuity not explained by refractive error or injury to the cornea, lens, or retina.

Visual acuity is decreased.

Examination

- Decreased visual acuity.
- Colour vision and red saturation are decreased and may be accompanied by a visual field defect.
- The optic disc appears normal. If an atrophic disc is seen on examination, a chronic process is ongoing or the nerve sustained injury at least six weeks previously.¹
- Disc haemorrhage and oedema may be seen.
- A new relative afferent pupillary defect in a traumatised eye suggests the diagnosis (i.e. no retinal pathologic conditions or chiasmal damage).^{1,6}

Aetiology

- Injury to the optic nerve from a sharp injury or compression by fragments of bone, foreign body, blood, or oedema can lead to traumatic optic neuropathy.
- A shearing injury from blunt trauma has also been implicated.

Differential diagnosis^{1,2}

- Optic chiasm injury.
- Optic nerve head avulsion.

Treatment

Once the diagnosis is suspected, the patient is immediately referred to an ophthalmologist for complete assessment and management.

Follow-up

The prognosis depends on the mechanism of injury and degree of damage sustained by the optic nerve. With a clinically evident relative afferent pupillary defect, the optic nerve must have sustained a fairly severe injury; therefore ultimate visual functioning may be poor.

Ocular burns

A burn may result from contact with heat, flame, chemicals, electricity or radiation, with chemical and radiation being the most common causes of burns to the eye.¹⁴

Management

The underlying basis of management of burn injuries is to preserve the globe vision and to prevent and treat complications.¹⁶

The management is based on an understanding of the protective mechanisms of the globe, the reaction of ocular tissues to injury and the healing processes of various ocular tissues. These latter include the blink, Bell's phenomenon, the tears, protective bony structure of the orbit and shielding of the eyes and face by the hands and arms.¹⁶

Alkali burns

Causes of alkali burns¹⁵

- Ammonia (NH₃): fertilisers, refrigerants and cleaning agents
- Lye (Sodium hydroxide Na OH): drain cleaners
- Potassium hydroxide KOH (Caustic potash)
- Magnesium hydroxide Mg (OH)₂: sparklers, flares and fireworks
- Lime (calcium hydroxide): plasters, mortar, cement and whitewash
- Motor vehicle airbag injuries. (Note: a side product of the explosive reaction, which produces inflation of the airbag, releases alkali, which can burn the eyes, though eye injury in this case is usually mechanical effect)
- Household cleaners, fertilisers, and refrigerants contain ammonia. Plaster, cement, mortar and whitewash contain fresh lime. Sparkles and flares contain magnesium hydroxide.

With alkali burn the corneal epithelium is rapidly and extensively lost.^{15,16} The epithelium is an effective barrier against microbial pathogens and its loss deprives the cornea of one of its most effective barriers, defence mechanisms and subsequently delays corneal stromal healing. The rapidity with which alkali penetrates the cornea is reflected by the rapid rise in pH in the aqueous. **Ammonia** causes the most severe form of alkali burn and the rise in pH may occur within seconds.¹⁵ It usually returns to normal with or without external irrigation within 30 minutes to three hours. The more severe the burn, the deeper the injury and the greater likelihood of ischaemic change. The corneal epithelium has a strong regenerative power; the corneal re-epithelialisation is dependent upon the limbal stem cell population, if this is severely damaged then poor recovery is likely with chronic scarring and loss of vision.

Alkali also acts directly on collagen resulting in trabecular meshwork distortion, therefore an early increase in intraocular pressure may result, and while damage to the nerve endings may produce corneal anaesthesia. Keratocytes may be completely obliterated, which further limits the possibility of a good recovery as they are pluri-potential cells with the unique ability to remodel damaged stroma.

In acute alkali injury there may also be damage to the corneal endothelium, to the lens, the iris and the ciliary body. There maybe an acute rise in intra-ocular pressure and, in extremely severe injury, hypotony may result. Hypotony indicates very severe injury – the ciliary body has been so badly involved that aqueous production has dropped – hence also intra-ocular pressure.

Acid burns

Causes of acid burns¹⁵

- Sulphuric acid (H₂SO₄): car battery acid

- Sulphurous acid (H₂SO₃): fruit and vegetable preservatives, bleach and refrigerants
- Hydrofluoric acid (HF): penetrates easily into the corneal stroma due to the presence of the fluoride ion
- Hydrochloric acid (HCL)
- Nitrous acid (HNO₂)
- Acetic acid (CH₃COOH)
- Fruit preservatives
- Glass-etching (HFI)

The description above of the response of alkali may apply equally to acid burns.^{8,15,17} However, acid burns are usually not as severe, since the coagulation of proteins in the corneal epithelium and superficial stroma caused by acid neutralises the acid and produces a barrier to deep penetration.

Superficial complications – such as vascularisation and scarring of the cornea – may occur, but corneal stromal thinning and sterile ulceration and perforation, along with the intra-ocular complications, are much less likely. The exception to this is an injury caused by **hydrofluoric acid**, which penetrates the cornea rapidly because of the presence of the fluoride ion. These injuries may be associated intra-ocular inflammation, including corneal endothelial damage and cataract formation.

Thermal burns

The response of the bulbar tissues to thermal injuries varies. Generally the instantaneous nature of the injury produces a burn that is limited to the superficial layers. In **flame injuries** frequently the globe is spared. In **contact injuries**, however, the severity may vary from superficial epithelial loss to perforation, if a burning object remains in contact with the eye wall and burns through it. Molten metal can collect in the fornix and remain in contact with the globe for some time while it cools.

Chemical injury

History

- A history of chemical exposure.
- Severe pain, redness, blurred vision and eyelid spasm.

Examination

Signs vary depending on the severity of the injury and the time since the chemical exposure.

In mild to moderate injuries^{1,17}:

Initial signs include corneal epithelial loss, chemosis and conjunctival hyperaemia, subconjunctival haemorrhage, intact episcleral and conjunctival vessels, and mild peri-ocular skin involvement (first-degree burns).

Chronic signs include minimal corneal scarring.

In severe injuries^{1,17}:

Initial signs include severe chemosis; corneal oedema and opacification; loss of conjunctival and episcleral vessels, which causes a very white-appearing sclera; severe peri-ocular skin involvement (second or third degree burns); and a marked anterior chamber reaction, which may not be visualised.

Chronic signs include: foreshortened fornices (loss of normal conjunctival cul-de-sac) with symblepharon formation (conjunctival and globe adhesions);

severe eyelid abnormalities such as trichiasis (misdirected eyelashes), entropion (lid turned in), and ectropion (lid turned out); severe tear film abnormalities (loss of mucus producing cells in the conjunctiva); corneal scarring and opacification and phthisis bulbi (shrunken, blind eye).

Treatment

As with other ocular injuries, the history guides the evaluation and treatment.

The severity of the chemical injury is directly related to:

- The nature of the chemical
- Whether or not the chemical was diluted with volume
- The volume of chemical that entered the eye
- The speed at which first aid was given, both on site and in the casualty department

In cases of severe exposure, initial treatment precedes the ocular evaluation. Copious irrigation with at least two litres of normal saline solution 0.9% over one hour is performed as an initial treatment in the emergency room. Topical local anaesthetic is instilled initially and every 10 to 15 minutes to make this a much less painful procedure. Lid speculum is used if significant orbicularis spasm is present.^{8,9}

The clinician should sweep the conjunctival fornices with a moistened cotton-tipped applicator to remove any retained foreign matter, especially lime, which exists as particulate matter.

It is also essential to evert the upper and lower lids to ensure that no retained chemical is present after sweeping the fornices.

In cases of less severe exposure or questionable history (e.g. the patient reports getting a drop of cleaner in the eye but washing it out at home), less copious irrigation with pH measurements is performed initially.

Irrigation continues until the conjunctival pH normalises (i.e. 7.3 to 7.6); the pH is checked with the pH section of a urinalysis strip or litmus paper. Two to three normal readings should be obtained at five to 10 minute intervals widely, and the eye should be reassessed.

The possibility of a ruptured globe is carefully assessed. Minimal pressure is placed on the globe during lavage when this diagnosis is a possibility or is indicated by the history (very rarely related to chemical/burn injuries). Specific treatment actions are as follows:

- Cyclopegic agents (e.g. homatropine 5%) and mydriatic agents (e.g. phenylephrine 2.5%) are instilled to dilate the pupil. Note: In severe injury, some researchers discourage instillation of phenylephrine because of the possibility of further vasoconstricting the conjunctival vessels.¹
- Topical antibiotic ointment is instilled and a pressure patch is placed over the eye.
- Immediate referral to an ophthalmologist is needed once the initial lavage is complete.
- Management of severe burns includes treatment of the intra-ocular pressure problems, exposure, scarring, and tear film dysfunction; therapy involves corticosteroid administration, ascorbate or citrate supplementation (in cases of alkali burns only), and surgery (e.g. conjunctival grafts, corneal transplants).
- The most important aspect of treatment of a chemical injury is the speed and efficiency of the washing out procedure.

Follow-up

Patients are usually monitored daily for several days.

Prognosis

The prognosis mainly depends on the type of injury.

Even in the most severe alkaline injuries the primary care physician can play a significant role in reducing the chronic sequelae. By instructing the patient to irrigate at the place of injury (e.g. home, work) using a sink, shower, or garden hose – rather than immediately summoning the patient to the emergency room – the chronic sequelae can be reduced. As stated, the prognosis is directly affected by the adequacy of the lavage immediately after exposure.

Practice points

Sometimes what seems like a mild injury on the first day can develop into a more serious condition later. Warn the patient of this and refer all chemical injuries for thorough assessment.

Thermal injury

History

- A history of exposure to a hot object (e.g. curling iron, tobacco ash, electrical arc, explosion).
- A red painful eye, tearing, a foreign body sensation, and decreased vision.

Examination

- Burns of the eyelids and peri-ocular region.
- Corneal whitening indicates an epithelial or a stromal burn.
- Conjunctival chemosis and injection.
- Corneal epithelial defect is evident.
- Minimal anterior chamber reaction.

Differential diagnosis¹

- Corneal abrasion or infection (especially if no history available).
- Ultraviolet injury (welder's flash).

Treatment

- Associated alkaline injury should be ruled out or confirmed if the thermal burn was caused by fireworks or flares (magnesium hydroxide).
- Double pad the worse eye for 24 hours with chloramphenicol ointment and cyclopentolate drops, and prescribe chloramphenicol ointment four times a day for the other eye and for the padded after this time.
- Analgesias (e.g. paracetamol, NSAIDS).
- Cold compresses are useful.
- In cases of deep burns of the cornea, patients should be referred immediately to the ophthalmologist.
- Peri-ocular burns are treated with ophthalmic preparations. Skin preparations may enter the eye and cause epithelial toxicity.

Follow-up

An ophthalmologist or oculoplastic surgeon is consulted if severe peri-ocular injury accompanies the ocular injury. Cicatrisation (scarring) of the eyelids from severe burns may lead to exposure and corneal scarring.

Practice points

Light induced burns to the eye: 'flash burns', 'arc eye'.¹

Patients exposed to the light of an arc-welding lamp may develop very painful red watery eyes a few hours after exposure. Although the eyes are very painful it is only the very front surface of the eye (the corneal epithelium) which has been affected, and this will heal completely in two to three days.

Conjunctivitis

The conjunctiva is a thin, transparent mucous membrane that lines the inner surface of the lids and outer surface of the eye. Conjunctivitis, inflammation of the conjunctivitis, is a common presenting problem to the doctor and can be broadly classified into the three common causative factors: viral; bacterial; or allergic conjunctivitis.

Conjunctivitis in young children is extremely important because the eye defences are immature and a severe conjunctivitis with membrane formation and bleeding may occur. Serious corneal disease and blindness may result. Conjunctivitis in an infant less than one month old (ophthalmia neonatorum) is a notifiable disease. Such babies must be referred to the ophthalmologist. Venereal disease in the parents must be excluded.

Viral conjunctivitis

Viral conjunctivitis occurs in clusters or mini-epidemics (pink eye). Most often it is caused by an adenovirus, a highly contagious organism with an incubation period of four to 10 days. Other viral causes include coxsackie and enterovirus, which also occur in epidemics.^{7,8,18,21}

History

- Acute onset. Red eye, photophobia, watery discharge, gritty and uncomfortable feeling.
- The second eye is usually involved three to seven days after the first, and the symptoms are less severe in most cases.
- This type of conjunctivitis usually lasts longer than bacterial conjunctivitis and may go on for many weeks.
- Photophobia and discomfort may be severe if the patient goes onto develop discrete corneal lesions.

Examination

- Visual acuity is normal (when the patient is not looking through a film of tears or discharge) Chemosis, watery discharge. In severe cases, erythema and oedema are often found in the lids.
- A follicular response (lymphoid aggregation) in the conjunctiva is evident in most cases.
- Pre-auricular adenopathy is common; patient may report tenderness in this region. Bacterial conjunctivitis is almost never associated with pre-auricular adenopathy, which can be a differentiating feature.
- Subepithelial infiltrates can develop in the cornea two to three weeks after the acute infection. They result from the body's immune response to viral antigens and can cause decreased vision and photosensitivity.

Treatment

Just as there is no cure for a cold, there is no treatment one can give a patient with a viral conjunctivitis. Treatment is symptomatic and includes (i) cold compresses, (ii) artificial tears and (iii) topical vasoconstrictors (such as phenylephrine 0.12%) (iv) topical Chloramphenicol

drops/ointment (to prevent secondary infection, and provide lubrication) and (v) avoidance of bright light.¹⁸

Viral conjunctivitis is extremely contagious and strict hygienic measures are important for both the patient and the doctor – for example, washing of hands, sterilising of instruments and so on.

Patients are counselled that they (and especially their tears) are infectious, so their handkerchiefs, towels, pillows etc. need to be kept separately: if other members of the family use them they may contract the virus.

Reassure the patient that their vision is unlikely to be affected, but the condition, although self-limiting, can take as long as three to four weeks to disappear.

Warn the patient that if their vision becomes blurred (even after they clear tears/discharge away from their eye) they should return. This symptom means that the infection may have spread to the cornea and the patient should be referred within 24 hours.

Practice points

- In view of the chronic course of some cases the patient may return for further treatment, but steroids must not be given without ophthalmological supervision.
- Never pad a discharging eye.
- If possible clear away debris and mucus with sterile normal saline solution before using medication.
- If there is no improvement after two days with antibacterial, review diagnosis and consider taking swabs after a 24-hour period without antibacterial.
- Viral conjunctivitis is infectious until redness and weeping resolves (usually 10-12 days after onset). Patients should avoid touching their eyes and sharing towels, and should wash hands before and after instilling medications, e.g. lubricant drops.
- Appropriate cultures and susceptibilities should be considered if clinically indicated.

Bacterial conjunctivitis

Incidence/prevalence

Currently there is no good evidence on the incidence or prevalence of bacterial conjunctivitis.

In adults, bacterial conjunctivitis is less common than viral conjunctivitis; although estimates vary widely (viral conjunctivitis has been reported to account for 8-75% of acute conjunctivitis).^{21,22} Staphylococcus species are the most common bacterial pathogens, followed by Streptococcus pneumoniae and Haemophilus influenzae.²³

In children, bacterial conjunctivitis is more common than viral, and is mainly caused by H. influenzae, S. pneumoniae, and Moraxella catarrhalis.²⁴

History

- Red eye, irritation, purulent discharge, adhesion of the lids (especially in the morning).
- There may be a history of contact with a person with similar symptoms.

Examination

- Normal visual acuity.
- A mucopurulent discharge is found in the fornix and on the lid margin associated with conjunctival papillae.

- In cases of diffuse conjunctivitis, erythema and oedema of the lids is sometimes observed
- *N. gonorrhoea* and *N. meningitidis* cause a 'hyperacute' conjunctivitis characterised by an exuberant mucopurulent discharge.^{7,36} Because the organism can rapidly invade the cornea, causing tissue destruction and ocular perforation, infection with *Neisseria* species results in a potentially serious form of conjunctivitis.^{7,36}

Treatment

The aim is to achieve rapid cure of inflammation and to prevent complications, with minimum adverse effects of treatment. Chloramphenicol eye drops should be instilled hourly for 24 hours, decreasing to four times a day, and chloramphenicol ointment applied each night for a week to hasten recovery.²⁵

Practice points

Swabs for microscopy, Gram's stain and culture are performed in any cases suggestive of conjunctivitis caused by *Neisseria* species.

Most cases do not require extensive investigation because broad spectrum antibiotics eradicate the infection and conjunctival swabs are only necessary in cases where significant improvement does not occur within three to four days of treatment.

Gonococcal or chlamydial conjunctivitis (see below) should be considered in patients who do not respond to treatment, and should be referred to an ophthalmologist for investigation

Gonococcal conjunctivitis

Gonococcal conjunctivitis is seen both sporadically and in epidemics in Central and Northern Australia.³⁶ The rare sporadic cases outside these areas should be treated in consultation with an ophthalmologist. Patient requires both topical and systemic antibiotic treatment.³⁶

Treatment^{35,36}

For sporadic cases, three days of therapy is adequate. Procaine penicillin (child: 50 mg/kg up to) 1.5 g intramuscularly daily for three days. or Amoxycillin (child: 75 mg/kg/day) for children or 3 g plus probenecid 1 g for adults for three days.

In the epidemics, all household and classroom contacts should be treated and single dose therapy of above regimen has been adequate to date.

Gonococcal ophthalmia in neonates

This is highly contagious and may rapidly lead to blindness. Patient should be admitted for both topical and systemic antibiotic treatment.³⁶

Cefotaxime 50 mg/kg intravenously, eight hourly or Ceftriaxone 50 mg/kg intravenously, daily for seven days.

In areas where the prevalence of penicillin-resistant *N. gonorrhoea* is low, or where the organism from the neonate or mother is known to be penicillin-susceptible, use²⁹ Benzylpenicillin 15 mg/kg intravenously, 12 hourly during the first week of life, and 7.5 mg/kg six hourly, thereafter, for a total of seven days.

Co-infection with *Chlamydia trachomatis* and *N. gonorrhoea* is common in some areas. Thus treatment of *C. trachomatis* conjunctivitis should be used in addition to the beta-lactam antibiotic.

Prognosis

Most bacterial conjunctivitis is self-limiting. One systemic review of randomised controlled trials reported clinical cure or significant

improvement on placebo within two to five days in 64% of people (99% CI 54% to 73%).²⁵

Some organisms cause corneal or systemic complications, or both; otitis may develop in 25% of children with *H. influenzae* conjunctivitis, and systemic meningitis may complicate primary meningococcal conjunctivitis in 18% of people.²⁶

Conjunctivitis in children is more likely to be bacterial than viral, warranting heightened awareness of possible systemic complications.

Adult chlamydia conjunctivitis

History

- Acute or subacute onset. Patients are usually young with a history of chronic bilateral conjunctivitis with a mucopurulent discharge.
- There may be associated symptoms of venereal disease.

Aetiology

Ocular inoculation usually results from chlamydial infection of the genitalia.

Examination

- Unilateral or bilateral.
- Diffuse conjunctival injection with a scant mucopurulent discharge and many lymphoid aggregates in the conjunctivitis (follicles). The cornea is usually inflamed (keratitis) and an infiltrate of the upper cornea (pannus) may be seen. Pre-auricular adenopathy are sometimes seen.

Investigations

Conjunctival swabs for antigen detection, polymerase chain reaction or ligase chain reaction test, or culture should ideally confirm the diagnosis, as successful treatment requires systemic therapy. There is no evidence that additional topical therapy provides any benefit.

For neonates (inclusion conjunctivitis) and children under 6 kg, use erythromycin 10 mg/kg orally, six-hourly for 21 days.

For adults and children over 6 kg, use azithromycin (child 20 mg/kg up to) 1 g orally, as a single dose.²⁷

In areas where trachoma is prevalent, the treatment of all household contacts is recommended. When the prevalence of trachoma in the community is above 20%, a community-based treatment program should be considered where practicable.³⁶

Allergic conjunctivitis

History

The prevalent symptom is intense itching.¹⁹ Almost always bilateral. Usually associated with a history of exposure to: pollen (hay fever), medication (topical eye preparation), insect bites, chemicals (e.g. make-up). There may be a family history of atopy. Similar symptoms may have occurred at the same time in previous years.

Examination

- Conjunctival injection and chemosis. The discharge is clear and stringy.
- Because of the fibrous septa that tether the eyelid (tarsal) conjunctivae, oedema, results in round swellings (papillae). When these are large they are referred to as cobblestones.

Treatment

For treatment of acute allergic conjunctivitis, the patient should be instructed to go home and lie down, with their eyes closed, and covered with a cold flannel for comfort.

The swelling of the conjunctiva settles spontaneously over a few hours, and requires no treatment.⁷ Reassure the patient and his/her parents that this is an acute allergic response and that the patient should avoid the causative factor from now on if identified.

Hay fever can affect the eyes badly and unfortunately antihistamine tablets seldom help. Topical sodium cromoglycate 'Opticrom' and the more recently introduced Iodoxamide 'Alomide' drops used four times a day for many weeks are very effective.^{2,7} These take two weeks to modify the mast cell population so must be used regularly for some time for maximum benefit.

Systemic allergy evaluation is performed with consideration of desensitisation treatment and removal of allergens from the patient's environment.

Systemic antihistamines are administered in severe cases. Several topical preparations may be useful such as: topical vasoconstrictor/antihistamine combinations, topical antihistamines, mast cell stabilisers, topical NSAIDS.

If these treatments fail, patients should be referred to an ophthalmologist for further treatment for consideration of topical corticosteroid.

Corneal ulcers

Examination

- A white corneal opacity. Most corneal ulcers are easily visible with the naked eye, have many causes and can be situated anywhere on the cornea.
- A corneal ulcer is likely to be infected and the infection can spread inside the eye. Assume a corneal ulcer is infected until proven otherwise.^{1,2}

Treatment

Corneal ulcers always need referral, even if they are very small.^{1,2,6,7}

Cellulitis (pre-septal or post-septal)

Pre-septal cellulitis is the less serious of the two, and involves the eyelids. Orbital cellulitis is the more severe condition that affects the contents of the orbit. Orbital cellulitis is a medical and ophthalmic emergency, as the infection can spread to the brain and the situation can become life threatening in a matter of hours.¹

Pre-septal cellulitis

History

Warm, red, tender swelling of the lids may extend over the nasal bridge to the opposite side. This may be associated with a stye, traumatic or surgical lacerations.

Examination

- Usually a low-grade fever and elevated white blood cell count.

- The eye is usually white or may be red if there is a bacterial conjunctivitis secondary to an underlying cause, e.g. discharging eye.
- Normal visual acuity.
- There is neither proptosis, i.e. the eyeball is not pushed forward, red desaturation or RAPD.
- Blood cultures are usually negative unless the organism is Haemophilus influenzae or Streptococcus pneumoniae.

Aetiology

- Upper respiratory tract infection or sinusitis (commonly arises from infection in ethmoid sinuses). The most common causative organisms in adults are Streptococcus species, Staphylococcus aureus, and mixed flora.
- Lid trauma (blunt or perforating). The most common causative organisms are Streptococcus pyogenes, S. aureus and fungus (if organic material was involved).
- Superficial lid infections such as a stye (hordoleum) or impetigo.
- Conjunctivitis.
- Dacryocystitis.
- Surgical procedures that violate the orbital septum – such as strabismus, retinal detachment repair, and orbital surgery – can lead to the disorder.

Differential diagnosis ¹

Orbital cellulitis, orbital pseudotumour, carotid-cavernous fistula.

Investigation: FBC, swabs of an open wound, purulent nasal drainage, conjunctival discharge, or any weeping vesicles for microscopy, culture and sensitivity. Computer tomography of orbits and sinuses if indicated. Blood cultures are performed if H. influenzae or S. pneumoniae is suspected.

Treatment³⁶

Refer to hospital for admission if systemically unwell.

Children under five years: For the severely ill child, give cefotaxime 50 mg/kg up to 2 g intravenously, eight-hourly, or ceftriaxone 50 mg/kg up to 2 g intravenously, daily, until clinically improved, followed by amoxicillin/clavulanate 15 mg/kg up to 500 mg orally, eight-hourly to complete a total treatment period of one week.

When associated with local lesions such as styes, dacryocystitis or impetigo that suggest staphylococcal or streptococcal infection, di(flu)cloxacillin should be added to the regimen above.

Adults and older children: di(flu)cloxacillin 500 mg (children 12.5 mg/kg up to 500 mg) orally, six-hourly. In severe, use di(flu)cloxacillin 2 g (children: 50 mg/kg up to 2 g) intravenously, six-hourly.

Follow-up

The focus of follow-up is to ensure that orbital cellulitis does not develop. Warn the patient that if their symptoms increase, they should return for further examination. The patient does not need referral unless the diagnosis is in doubt or the condition deteriorates.

Orbital cellulitis

History

Symptoms are the same as those for preseptal cellulitis. But the patient is in severe pain, is feverish and systemically unwell and shows evidence of orbital involvement.

Examination

- Low-grade fever. Both lids are red, swollen and tender, which can be so severe that the patient cannot open their eyes.
- Normal visual acuity.
- Proptosis (when viewed from above the patient's head, and (if possible) with the eyelids pulled out of the way, the eyeball will be seen to protrude further out of the socket when compared with other eye).
- Conjunctival chemosis, sluggish pupillary reflex.
- Eye movements are restricted. Early restrictions of movement can be difficult to detect but the patient will tell you that they see double. This is a very important sign.
- The signs of severe disease are reduced visual acuity, red desaturation, and presence of an RAPD indicating optic nerve compression.

Investigations

- Same as that for cases of preseptal cellulitis.
- A fundoscopic examination may reveal retinal haemorrhages, venous congestion, and disc oedema.
- A CT scan displays diffuse infiltration of orbital fat may progress to abscess formation.
- Blood cultures are usually negative.

Differential diagnosis

Differential diagnoses are the same as those for preseptal cellulitis.

Treatment

- Urgent referral to ophthalmologist and intravenous antibiotics.
- Urgent surgical drainage of the sinuses or of an abscess may be required, lest vision be lost.

Practice points

- The adequate examination of children presenting with orbital cellulitis can be difficult. Therefore, have a high index of suspicion and refer early.
- The possibility of orbital cellulitis should always be kept in mind, especially in children, and patients should be referred immediately.

Acute red eye

Acute angle closure glaucoma

Acute glaucoma should always be considered in a patient over 50 (can occur younger than 50, but rarely) with a painful red eye. The diagnosis must not be missed or the eye will be permanently damaged.

History

- Acute onset, characteristically in the evening when the pupil becomes semidilated. The patient may have had similar attacks in the pasts that were relieved by going to sleep (the pupil constricts during sleep, so relieving the attack).
- Intense ocular pain and photophobia.
- Blurred vision and halos are seen round light fixtures due to oedema of the cornea.
- It can be associated with vasovagal symptoms such as diaphoresis, nausea, and vomiting.

Examination

- A fixed and mid-dilated pupil.
- Lid oedema and conjunctival injection.
- Hazy cornea with fragmented light reflex.
- Vision is impaired according to the state of the cornea.
- On gentle palpation, the eye feels harder than the other eye.
- The anterior chamber seems shallower than usual, with the iris being close to the cornea.
- If the patient is seen after the resolution of a reversible attack the signs may have disappeared – hence the importance of the history.

Associated factors and diseases

- Women are affected three to four times more commonly than men.¹
- The average age of presentation is 60 and increases thereafter.¹
- Shorter, smaller, far-sighted eyes with narrow chamber angles.¹
- Stress, a darkened room, and drugs that can dilate the pupil may precipitate an acute angle-closure attack.¹ Many systemic medications with anticholinergic or sympathomimetic action carry a warning against use in persons with glaucoma. This applies to patients with a narrow chamber angle only, not to patients with open-angle glaucoma. Because most glaucoma cases are open-angle, these medications are rarely contraindicated in medical practice.
- Two studies have shown that the incidence of mydriasis-induced acute angle glaucoma is only approximately 0.02%.^{32,33} [Editor: This is ~2 in 10 000, so don't let this put you off using mydriatic drops to examine the fundus when needed.]

Treatment

- Acute angle-closure glaucoma is an ophthalmic emergency and requires immediate referral to an ophthalmologist.
- Initial medical treatment to lower the IOP involves a topical B-blocker (e.g. timolol 0.5% – 1 drop), carbonic anhydrase inhibitors (e.g. Diamox 500 mg intravenously or 250 mg orally two times), and osmotic agents if Diamox fails to reduce IOP (e.g., oral isosorbide 50-100 g or intravenous mannitol 1-2 g/kg given over 45 minutes – 500 ml of mannitol 20% contains 100 g of mannitol).¹
- In most cases, laser iridotomy (creating a full thickness opening in the peripheral iris) reopens at least a portion of the angle with marked lowering of the IOP. This is the definite treatment of acute angle-closure glaucoma, but cannot always be done in the acute stages.

Follow-up

- After an acute-angle closure event, a portion of the angle may remain closed because of scarring of peripheral iris tissue to the cornea, causing chronic angle-closure glaucoma.
- Chronic angle-closure glaucoma may also occur without any symptoms, just like open-angle glaucoma. Treatment involves medications and incisional surgery such as trabeculectomy.
- Laser iridotomy may be recommended for individuals with very narrow angles who are at significant risk for the development of either acute or chronic angle-closure glaucoma. Such patients are described as having occludable drainage angle.

Acute iritis/uveitis

Acute iritis is usually idiopathic, but the commonest systemic association is in patients who are HLA-B27 positive.³⁴

History

- The patient who has had past attacks can often feel an attack coming on before physical signs are present.
- Painful, photophobic, red eye with reduced vision.

Examination

- Reduced visual acuity.
- The diagnosis is made on the slit lamp, but the history obtainable from the patient is often typical.
- Small and maybe irregular pupil (this is more likely if the patient has suffered with recurrent attacks of iritis).
- Ciliary flush, anterior chamber flare (cells and proteinaceous exudate), keratitic precipitates on the cornea or may settle to form a collection of cells in the anterior chamber of the eye (hypopyon).

Treatment

The diagnosis is difficult without the use of a slit lamp and the patient should be referred to an ophthalmologist.

Where the patient has had previous episodes and is confident as to the similarities with this acute presentation, topical steroids (Prednefrin Forte two hourly)³⁰ and homatropine 2% three times daily can be commenced while ophthalmic review is arranged.

Medications

There are a wide variety of topical ophthalmic preparations available as diagnostic aid and rationale treatment for many eye diseases. Drugs administered topically act either within the eye or on its surface. Blinking distributes and eliminates drugs dissolved in tears. As tears containing the drug pass through the lacrimal drainage system and into the nasopharynx, it may be absorbed through the mucosa into the systemic circulation (without first having to pass through the liver) and lead to systemic side effects.^{31,35}

Choice of preparation is limited by the range of products available. Adverse effects due to nasal drainage and systemic absorption are more common with eye drops than with ointments.³⁵

Ointments are appropriate to use at night, in children, and when action depends on sustained concentrations of drug, e.g. antivirals. Drug action is prolonged by decreasing drug dilution and drainage. Ointments are often difficult to self-administer, blur vision may cause contact dermatitis.

Diagnostic medications

Topical fluorescein³⁵

Fluorescent stains the cornea at epithelial defects (orange when concentrated, bright green when diluted) taken up by devitalised tissue.

Indications

It is used to diagnose corneal abrasions and ulcers, fit rigid gas permeable contact lenses, test patency of lacrimal passage (Jones tests), detect aqueous leak and for applanation tonometry.

Dosage

Eye drop, 1 drop (0.25% ideal for applanation tonometry).

Paper strips, moisten tip with a drop of either sterile 0.9% normal saline solution or topical anaesthetic; avoid abrading the cornea.

Specific considerations

It is safe to use in pregnancy and lactation.

Patient counselling

Fluorescein stings initially, and can cause temporary yellow staining of skin, urine, nasal secretions and tears. It can permanently stain soft contact lenses and clothes (comes out with soap and water but is unresponsive to dry cleaning).

Rose Bengal ³⁵

Stains nuclei of corneal and conjunctival epithelial cells at defects in the precorneal tear film (where mucus layer is deficient); also stains 'sick' cells (supravital stain) and abnormal mucin.

Indications

It is used to demonstrate superficial corneal and conjunctival tissue changes, especially herpes simplex epithelial disease, Sjogren's syndrome and keratoconjunctivitis sicca.

Dry eye syndrome: rose Bengal causes intense stinging; use topical anaesthetic first or instil only a fraction of a drop.

Dosage

Eye drops, 1 drop.

Specific considerations

It is safe to use in pregnancy and lactation.

Patient counselling

Rose Bengal stings initially (stings more than fluorescein) and can cause temporary red staining of skin, urine, nasal secretions and tears. It permanently stains soft contact lenses and clothes.

Ocular local anaesthetics³⁵: Amethocaine, lignocaine, oxybuprocaine and propavacaine

Indications

They block nerve conduction reversibly and provide short-term ocular surface anaesthesia.

Specific considerations

Prolonged use impairs corneal epithelial healing, prevents reflex ocular protection and masks progression of keratopathy; use only for short procedures (<20 minutes).

Corneal scrapings: use preservative-free drops (preservative may affect microbiological culture).

Adverse effects

Common: stinging on instillation, punctate epithelial epithelial damage of cornea (do not use long term because of epithelial toxicity, i.e. acute corneal ulceration).

Rare: allergy.

Dosage

1 drop, repeated in five minutes if necessary; maximum five doses.

Specific considerations

It is safe to use in lactation. However, for pregnancy lignocaine is classified as Australian Drug Evaluation Committee (ADEC) A and others not categorised.

Patient counselling

Warn patients (especially children) about the initial stinging.

Close eyes after instillation and dab away tears without rubbing eyes.

Practice points

- Never prescribe for home use; anaesthetics are toxic to epithelial cells and thus will delay or prevent wound healing.
- Topical anaesthetics increase corneal permeability and intra-ocular bio-availability of other topical drugs; they also reduce the initial stinging of other topical drugs and should be instilled first.
- Single-use drops are useful if infection is suspected.

Table 2: Comparison of ocular local anaesthetics³⁵

Drug	Amethocaine	Oxybuprocaine	Propavacaine	Lignocaine
Duration (minutes)	20	10-20	10-20	20-30
Duration (minutes)	20	10-20	10-20	20-30
Sting (seconds)	30	10	10	30
Punctate epithelial damage	+++	++	++	+

Mydriatics and cycloplegics^{1,35}

Mydriatic and cycloplegic agents can be used for diagnostic and therapeutic purposes. Mydriatics dilate the pupil, and cycloplegic agents paralyse the ciliary muscle. Dilating the pupil is necessary for adequate examination of the internal ocular structures.

Therapeutic benefits are numerous: paralyzing the ciliary muscle reduces pain associated with traumatic iritis, prevents posterior synechiae formation (adhesion between the iris and lens), and stabilise the blood ocular barrier during bouts of intra-ocular inflammation.

Topical anticholinergics are the main drugs used for both mydriasis and cycloplegia. Phenylephrine (a sympathomimetic) is used to supplement anticholinergic mydriasis.

Diagnostic use

- Adult, 1 drop tropicamide 0.5% or 15, repeated in five minutes (add phenylephrine 2.5% if dilation is inadequate).
- Child one to 12 years, 1 drop proxymetacaine followed by 1 drop cyclopentolate 1%, repeated in five minutes.
- Infants under one year, 1 drop of proxymetacaine followed by 1 drop cyclopentolate 0.5%.
- Preterm infant (<32 weeks), 1 drop proxymetacaine followed by 1 drop cyclopentolate 0.1%.
- Examine after 20 minutes (30 minutes for darkly pigmented eyes and for cycloplegic refraction).

Therapeutic

Iridocyclitis, 1 drop homatropine 5% every four to six hours, or 1 drop atropine 1% three to four times daily (phenylephrine 10% three times daily is occasionally used as an adjuvant).

Postoperative, 1 drop atropine 1% three to four times daily.

Patient counselling

To reduce initial stinging, shut eyes and avoid rubbing eyes after instillation. Enlarged pupils result in blurred vision and glare from bright light (suggest dark glasses). It is generally inadvisable to drive while vision is disturbed (approximately the duration of action). Report back if eye becomes painful or red, or vision deteriorates.

Practice points

- Do not use after head injury or if anterior chamber angle is narrow.
- Reversal of mydriasis with a cholinergic agent (pilocarpine) is not recommended and can precipitate an angle-closure attack in susceptible patients.

Anticholinergic

Atropine, cyclopentolate, homatropine, tropicamide³⁵

They act by reversibly blocking acetylcholine receptors on iris sphincter and ciliary muscle.

Contra-indications

Iris clip intra-ocular lens implant.

Specific considerations: Coexisting conditions

Significant head injury: uses only short-acting agents, and with care; consult patient's neurosurgeon or intensivist. Always make a note that pupils were dilated intentionally.

Narrow anterior chamber angle: mydriasis may rarely precipitate acute closed angle glaucoma.

Previously treated acute closed angle glaucoma: should be dilated under specialist supervision as not all laser iridotomies remain functional.

Lenticular subluxation: small risk of anterior lens displacement.

Children

Use with extreme caution if at all in neonates, preterm infants and children with spastic paralysis or brain damage, as they have increased susceptibility to systemic reactions. One drop of 0.5% atropine can cause systemic reactions in infants. In young children, long-term cycloplegia may induce amblyopia.

Adverse effects

Common: sting (especially 1% cyclopentolate), intolerance to bright light (glare) blurred vision (especially near vision), transient intra-ocular pressure elevation (especially in ocular hypertensives).

Infrequent: contact allergic blepharitis (atropine), persistent ocular irritation (mucus discharge, severe watering discharge, superficial punctate keratopathy and characteristically no itch), punctal stenosis with prolonged use (years), insomnia, drowsiness (cyclopentolate).

Rare: systemic toxicity, e.g. dryness of skin and mouth, fever, facial flushing, tachycardia, irritability, disorientation, ataxia, visual

hallucinations, incoherent speech, delirium, psychosis, seizures, hyperactivity in children.

Special considerations

It is safe to use in pregnancy and lactation.

Table 3: Comparison of Ocular Anticholinergics³⁵

Drug	Effect	Peak effect	Duration	Systemic side effects
Atropine 1%	Mydriasis cycloplegia	30-40 mins 3-6 hours	7-10days 7-14 days	+++
Cyclopentolate 0.5-1%	Mydriasis cycloplegia	30-60 mins 25-75 mins	1 day 6-24 hours	++
homatropine 2%	Mydriasis cycloplegia	20-30 mins 30-90 mins	6 hours-4 days 10 hours-2 days	+
tropicamide 0.5-1%	Mydriasis cycloplegia	20-40 mins 30-40 mins	6 hours 2-6 hours	rare

Sympathomimetics

Phenylephrine

It is a relatively selective alpha 1 agonist; stimulates pupil dilator muscle. Maximal mydriasis occurs after 60-90 minutes; duration of action is five to seven hours. It does not affect accommodation.

Specific considerations

Caution: With recent myocardial infarction or unstable angina. Blood pressure elevation can occur with repeated doses of 10% drops.

Elderly: Increased risk of systemic adverse effects.

Children: Increased risk of systemic adverse effects, especially hypertension and intraventricular bleeding in the first two to four weeks of life in preterm infants. Do not use 10% drops in preterm infants.

Drug interactions

MAOIs (antidepressants): hypertensive crisis may result; avoid use while taking phenelzine or tranylcypromide, and for 14 days after ceasing MAOI.

Methyldopa, alpha antagonist hypertension may occur; avoid combination.

Adverse effects

Common: rebound miosis, hyperaemia, stinging on instillation.

Infrequent: liberation of iris pigment (probably has no deleterious effects).

Rare: systemic effects (most frequently with 10% drops), e.g. hypertension, tachycardia, tremor, anxiety.

Dosage

Generally used as an adjunct.

Mydriasis: 2.5%, 1 drop once only as adjunct if mydriasis difficult, 10%, 1 drop once only as adjunct for rapid maximal mydriasis.

Uveitis: 10%, 1 drop three times daily as adjunct to mydriatic if trying to release posterior synechiae.

Episcleritis: diagnosis (vasoconstriction test) 2.5% 1 drop once only; episcleral vessels should blanch after five minutes.

Dilating drops are contraindicated in patients with known angle-closure glaucoma. Dilating drops may be less effective in patients with dark irides or intra-ocular inflammation. Administration of dilating drops to premature infants or children with cardiac disease or hypertension is high risk. These agents should be used cautiously in these patients.

Special considerations

It is not recommended to be used in pregnancy as theoretically it can induce placental vasoconstriction and fetal hypoxia. It is safe to use in lactation.

Practice points

Can reduce systemic absorption of the drug by pressing on the tear duct and closing eyes for three minutes after instilling drops

Antibacterial agents

Bacterial conjunctivitis³⁵

Acute bacterial conjunctivitis is frequently a self-limiting condition, lasts for two to three days.²⁵

Antibacterial is used to hasten recovery, prevent complications and limit the spread of infection to other people.

Drug choice

There are few comparative randomised controlled studies that compare antibiotics, and none have found a significant difference in rates of clinical or microbiological cure.

Aminoglycosides are active against gram-negative bacteria. Framysetin and especially, neomycin are associated with contact allergic reactions. Framycetin and, especially, neomycin are associated with contact allergic reactions. Gentamicin and tobramycin are more expensive, are active against Pseudomonas, and are indicated for neonatal gram-negative conjunctivitis.

Neomycin-polymycin combination is inexpensive and broad spectrum, but can cause contact allergy.

Sulfacetamide is irritant, avoid use.

Antiseptics (e.g. aminacrine, Aminopt) are ineffective, even for mild conjunctivitis.

Chloramphenicol

Chloramphenicol (chlorsig ointment, eyedrops) is the most common antibiotic used. It is bacteriostatic against Staphylococcus aureus and Enterobacteriaceae by inhibiting the protein synthesis at the ribosomal level.³⁷ It is bactericidal against Haemophilus influenzae, penicillin-susceptible Streptococcus pneumoniae, and Neisseria meningitidis, but not group B streptococci.³⁷

It is a broad spectrum antibiotic active against many gram-positive, gram-negative (except Pseudomonas species), and anaerobic bacteria, rickettsiae, chlamydiae and mycoplasma.³⁷

It has good ocular penetration and is safe in pregnancy and not expensive.

Contra-indications

Allergy to chloramphenicol.

Rare: allergic reactions, e.g. angioedema, anaphylaxis, contact dermatitis, dermatitis (often moderately severe).

Adverse effects

Local stinging, burning and unpleasant taste.³⁵ Topical chloramphenicol may very rarely cause aplastic anaemia as an idiosyncratic reaction resulting from systemic absorption.³⁸ There has been much debate about the association but it remains tenuous.

Dosage³⁵

Bacterial blepharitis: Massage ointment into lid margin two to three times daily. Blepharitis can often be managed with diluted baby shampoo used to wash the lid margins.

Bacterial conjunctivitis: 1 drop every two to four hours for two days; then if there is improvement, 1 drop four times daily for five days.

Ointment may be used as an adjunct to drops at night, or as a single agent three times daily, e.g. in children.

Prevention of infection (after superficial trauma or surgery): 1 drop four times a day until epithelium healed (rarely more than four days).

Ceftriaxone

It is a broad-spectrum cephalosporin. It is a popular agent due to its broad spectrum and its long half-life, allowing for once-daily dosing in non-central nervous system infections.

In ophthalmology, it is used as empirical treatment of orbital cellulitis and penetrating eye injuries.

Adverse effects³⁹

It can cause dose-dependent asymptomatic and reversible biliary sludge formation (pseudolithiasis), especially in children. This has been mistaken for gallstones on ultrasound scans and usually resolves after ceasing treatment. Pancreatitis and cholecystitis have also been reported. Associated symptoms may include nausea, epigastric distress, vomiting and right upper quadrant abdominal pain.

Dosage^{36,37}

Adult: 1 g IM/IV daily, maximum 4 g IV daily. Child: 50 mg/kg IM/IV as a single daily dose or divided dose 12-hourly.

Specific considerations³⁷

Neonates and preterm infants: Ceftriaxone is not advisable as it binds to serum proteins and displaces bilirubin from albumin and this may increase risk of bilirubin encephalopathy. Therefore, cefotaxime is preferred in neonates and preterm infants.

Antivirals

Viruses are obligate intracellular pathogens that use the host's metabolic processes for their survival and replication. Antiviral agents are designed to target the pathogen while leaving uninfected host cells essentially unaffected from their toxic side effects. To date, the most effective antiviral agents target viral enzymes and proteins that are essential for viral assembly.

Topical antivirals are indicated for treatment of herpes simplex keratitis and herpes zoster ophthalmicus with corneal involvement. Antivirals improve cure rates and reduce recurrences versus placebo. There is no good evidence of differences between topical antiviral agents.⁴¹

Aciclovir³⁵

Aciclovir is the common antiviral agent used.

Adverse effects

Common: mild transient stinging sensation.

Infrequent: superficial punctate keratitis, allergic reactions.

Dosage

Apply about 1 cm of ointment into the lower conjunctival sac, five times daily for 14 days or for three days after corneal epithelium healed, whichever is shorter.

There is conflicting evidence about the role of debridement before application of topical antivirals.^{40,42}

Specific considerations

It is safe to use in pregnancy (ADEC category B3) and lactation.

Practice points

- Herpes simplex keratitis is best managed by an ophthalmologist. Treatment usually requires topical steroid to suppress inflammation and topical antiviral to prevent viral replication. Therapy may be prolonged.
- For frequently recurring herpes simplex epithelial keratitis, advise patients to keep a spare tube of ointment and start treatment at the first sign of recurrence.

Drugs for allergic and inflammatory conditions

Allergic conjunctivitis

There are two main types, seasonal (hay fever) and perennial. Drug treatment is aimed at symptom control.^{19,35}

Treatment regimens³⁵

Identify triggers (e.g. pollens, house dust mites, cosmetics, ophthalmic drugs, contact lenses and solutions); avoid where possible.

Mild symptoms: irrigate with normal saline solution twice daily, use artificial tears four to eight times daily, and cold water compresses as required.

Moderate symptoms: use topical drugs, e.g. ketorolac, levocabastine. Topical decongestant-antihistamine combinations are not recommended in the long term due to rebound conjunctivitis.

Severe symptoms: ophthalmology referral; topical corticosteroids may be required.

Recurrent disease: use topical mast cell stabiliser as a preventative.

Factors influencing drug selection

- Response to cromoglycate may be delayed.
- Topical levocabastine and NSAIDs are expensive.
- Consider potential for adverse effects (e.g. raised intra-ocular pressure with corticosteroids).

Excessive mucus³⁵

- Decongestants include naphazoline, phenyl ephrine, tetrahydrozoline and xylometazoline.
- Antihistamines combined with decongestants in eye preparations include antazoline and pheniramine.

Mode of action

Alpha-adrenoreceptor agonists constrict conjunctival blood vessels, reducing ocular redness and discomfort.

Contra-indications

Narrow anterior chamber angle.

Drug interactions

Monoamine oxidase inhibitors (MOIs): wait 14 days after ceasing MOAI before using decongestants; risk of hypertensive crisis.

Adverse effects

Common: rebound hyperaemia, stinging on instillation.

Others: mild mydriasis, blurred vision, epithelial erosion, narrowing of the tear duct, acute and chronic conjunctivitis with prolonged use (months), corneal and conjunctival pigment deposition with prolonged use (years).

Comparative information

All decongestants have similar efficacy, adverse reactions and cost.

Dosage

Phenylephrine, 1-2 drops every three to four hours as required. Others, 1-2 drops every six to 12 hours as required.

Special considerations: It is safe to use in pregnancy and lactation.

Patient counselling

Although advertised as being useful for relieving eye redness due to minor irritations (such as dust, smoke and contact lens wear), a cool compress is beneficial and is safer.

Seek medical attention if symptoms do not improve within 48 hours.

Do not use continuously for more than five days.

Practice points

Decongestant eye drops, used widely in non-prescription products, are not recommended, as their benefit is doubtful and rebound hyperaemia leads to overuse.

Levocabastine³⁵

It is a selective H1 antagonist.

Adverse effects

Stinging, mild ocular irritation, headache

Dosage

Adult and child more than six years, 1 drop twice daily, increasing to three to four times daily if necessary.

Specific considerations

It should be avoided in pregnancy as there is no human data available (ADEC B3) and can be used in lactation if needed

Mast cell stabilisers³⁵

Cromoglycate, lodoxamide³⁵

They act by inhibiting mast cell degranulation by unknown mechanism.

Adverse effects

Generally well tolerated, stinging on instillation.

Comparative information

Cromoglycate may take three to six weeks to take effect; lodoxamide's effects occur more rapidly (one to two weeks).

Dosage

Cromoglycate: 1 drop four to six times daily; Lodoxamide: adults and children over four years, 1 drop four times daily.

Special considerations

Cromoglycate is safe to use in pregnancy and lactation, however there is limited data on lodoxamide.

Patient counselling

These drugs can take four to six weeks to reach full effect.

Practice points

Mast cell stabilisers have delayed onset of action; trial for at least two to four weeks before evaluating effect. For best results, start treatment one month before the onset of the hay fever season, or give in combination with a low potency topical steroid for the first month

Anti-inflammatory agents

Anti-inflammatory agents are used most frequently to suppress immunologic mechanisms of all types, both externally and within the eye. Suppression of severe external inflammation is necessary to prevent synechiae (scarring), some forms of glaucoma, and postoperative inflammation. Topical administration allows excellent penetration into the anterior chamber. Some agents penetrate easier than others depending on the chemical composition.

Topical corticosteroids should be used with caution because they can cause cataracts and glaucoma and potentiate herpes simplex viral replication.³⁵

Non-steroidal anti-inflammatory agents (NSAIDs)³⁵

NSAIDs inhibit cyclo-oxygenase, decreasing prostaglandin synthesis and prostaglandin-mediated inflammation.

They are prescribed for 1) inhibition of miosis during cataract surgery (diclofenac, flubiprofen), 2) allergic conjunctivitis (ketorolac) and 3) prevention of inflammation after cataract surgery (diclofenac, ketorolac), and can also be used as an alternative to steroids or as steroid-sparing agents (e.g. episcleritis, allergic conjunctivitis, inhibition of postoperative inflammation) and analgesia following photorefractive surgery.

Contra-indications

Aspirin or NSAID induces anaphylactoid reactions (e.g. asthma, urticaria or rhinitis).

Specific considerations

ADEC category C, although short-term low-dose use should not be a concern in pregnancy. They are safe to be used in lactation.

Adverse effects

Commonly cause stinging on instillation and ocular irritation.

Practice points

These agents have an insignificant effect on intra-operative miosis (marked indication) and are used more as an alternative or adjuvant to topical steroids.

May mask symptoms of ocular infections.

Ketolorac³⁵

Ketolorac is used for short-term (two to four weeks) treatment of seasonal allergic conjunctivitis and prevention and reduction of inflammation after cataract surgery.

Adverse effects

Common: local allergic reactions, superficial keratitis.

Rare: systemic allergic reactions.

Dosage

1 drop four times daily.

Practice points

Start drops 24 hours before cataract surgery then continue for two to four weeks if needed.

Corticosteroids³⁵

They act on intracellular receptor-mediated inhibition of inflammatory cascade, fibroblast and keratocyte activity.

They are indicated for allergic and selected inflammatory conditions of lids, conjunctiva, cornea, iris and ciliary body, including postoperative inflammation.

Contra-indications

Ocular infection, especially herpes simplex epithelial keratitis and fungal keratitis.

Adverse effects

Topical ocular steroids, alone or in combination with antibacterials, should not be prescribed without close supervision by an ophthalmologist as they have major, potentially vision-threatening, adverse effects.

Common: Ocular hypertension (usually reversible) proportional to dose, potency, penetration and duration of treatment; retarded corneal healing, rebound inflammation.

Infrequent: Opportunistic infection.

Rare: Refractive changes, ptosis, chemosis, lid swelling, exophthalmos (slowly, incompletely reversible).

Cataracts: Posterior subcapsular cataracts may occur with long-term (greater than one year) high-dose use; mostly asymptomatic and partially reversible.

Dosage

Titrate to disease severity and treatment response. Usual: 1 drop (or application of ointment) two to four times daily. Intensive: 1 drop every hour.

Specific considerations

Contact lens wearers: risk of indiscriminate long-term use of steroids to relieve ocular irritation.

Glaucoma: may be aggravated.

It is safe in pregnancy and lactation.

Practice points

Do not prescribe ocular corticosteroids for longer than two weeks without supervision by an ophthalmologist unless facilities are available to monitor corneal epithelium and intraocular pressure, and never without first staining with fluorescein to exclude an ulcer of any sort.

Therapy of tear deficiency states

The tear film is a highly complex layer responsible for vision (the primary ocular refractive surface) and ocular comfort. A large percentage of patients have tear film abnormalities and experience symptoms related to 'dry eyes.' Tears contain mucous, lipid and aqueous components but the most common cause of tear deficiency states is lack of the watery component. Such conditions are mild in most cases and caused by senile atrophy of the lacrimal gland, or by neural or humoral factors.¹⁹ Patients with ocular surface abnormalities usually respond well to artificial lubricants, although the symptomatic relief may be only temporary.^{19,35}

Drug choice^{19,35}

Use of ocular lubricants provides symptomatic relief. Patient preference after an empirical trial usually determines final choice.

Topical drops should be given as frequently as is necessary to alleviate symptoms. These may vary from two or four times daily to every hour depending on severity of symptoms.

Preservative-containing: if drops are used infrequently, use an inexpensive lubricant containing a preservative that prevents microbial contamination.

Preservative-free preparation: if drops are required more than four to six times daily, unpreserved lubricants such as Polytears and Genteal should be considered to avoid the ocular surface toxicity caused by preservatives. It is non-irritant but more expensive and packaged in bulky single-dose containers that are harder to use. Single-use vials can be used safely more than once (if uncontaminated and kept refrigerated) but must be discarded 24 hours after opening. Ointments are also beneficial as nocturnal ocular lubricants, the major difference between them being the inclusion of lanolin in Lacri-lube. Practitioners should be aware that lanolin can act as a sensitiser at the ocular surface and, if ocular inflammation is increasing, it may be reduced by introduction of a lanolin-free ointment. Lacrisert is a sustained-release lubricant designed to sit in the inferior conjunctival recess and provide continuous tear film supplementation. However, sufficient tears to dissolve the lubricant are required.

Eye ointments/gels

Used before bedtime if symptoms interrupt sleep or occur on awakening, and may be used throughout the day if dry eye is severe. Some people have incomplete lid closure at night and may benefit from having lids taped closed overnight.

Specific considerations

It is safe to use in pregnancy and lactation.

Patient counselling

Lubricants can be used safely as often as possible.

Practice points

- Reduce evaporation by using cold water, humidifiers, avoiding air conditioners that dry the air, and hair driers, and by wearing wrap-around glasses or even swimming goggles.
- Patients often find a suitable lubricant by trial and error.
- Encourage regular use if required (e.g. hourly).
- Ocular delivery system is usually a last resort; it is difficult to insert and often poorly tolerated.

References

1. Palay DA, Krachmer JH. Ophthalmology for the Primary Care Physician. Mosby-Year Book, Inc. USA, 1997.
2. Okhravi N. Manual of Primary Eye Care. Torino, Italy: Reed educational and Professional Publishing Ltd, 1997.
3. Berson FG. Basic Ophthalmology for Medical Students and Primary Care Residents. 6th ed. United States of America: American Academy of Ophthalmology, 1987.
4. MacCumber MW. Management of Ocular Injuries and Emergencies. United States of America: Lippincott-Raven Publishers, 1998.
5. Specialists Eye Health Guidelines for use in Aboriginal and Torres Strait Islander populations. Office for Aboriginal and Torres Strait Islander Health, Commonwealth Department of Health and Aged Care, 2001.
6. James B, Chew C, Bron A. Lecture Notes on Ophthalmology. 8th ed. United Kingdom: Blackwell Science.Ltd, 1997.
7. Elkington Ar, Khaw PT. ABC of Eyes. Great Britain: British Medical Journal, United Medical Journal, 1990.
8. Manolopoulos J. Emergency Primary Eye Care: Tips for diagnosis and acute management. Australian Family Physician 2002; 31(3):233-7.
9. Catalano RA. Eye injuries and prevention. Pediatric Clinics of North America 1993; 40(4):827-39.
10. Cassen JH. Ocular Trauma. Hawaii Medical Journal 1997. 55:292-4.
11. Pavan-Langston D. Manual of ocular diagnosis and therapy. Boston, United States of America: Little Brown, 1980.
12. Bartlett JD, Jannus SD, eds. Clinical ocular pharmacology 189. USA: Butterworth, Stoneham.
13. Zuckerman BD, Lieberman TW. Corneal rust ring. Arch Ophthalmol 1960; 63:254-65.
14. White WL, Hollsten DA. Burns of the Ocular Adnexa 1994. 5:74-7.
15. Hammerton ME. Burns to the eye: an overview 1995; 24(8): 998-1003.
16. Hammerton ME. Management of Ocular Burns 1995; 24(8):1006-20.
17. Wagoner MD. Chemical Injuries of the Eye: Current Concepts in Pathophysiology and Therapy. Survey of Ophthalmology 1997; 41(4):275-313
18. Coote MA. Sticky Eye, Tricky Diagnosis. Australian family Physician 2002; 31(3):225-231.
19. Hall AJ. Itchy Burning Eyes: Diagnosis and Management. Current Therapeutics 1999; 11:34-36.
20. Class'e JG. Anterior segment disease Update. Optometry Clinics. 1(4):59-70.
21. Wishart PK, James C, Wishart MS, Darougar S. Prevalence of acute conjunctivitis caused by chlamydia, adenovirus, and herpes simplex virus in an ophthalmic casualty department. Br J Ophthalmol 1984; 68:653-5.
22. Fitch CP, Rapoza PA, Owens S et al. Epidemiology and diagnosis of acute conjunctivitis at an inner city hospital. Ophthalmology 1989; 96:1215-20.
23. Seal DV, Barrett SP, McGill JI. Aetiology and treatment of acute bacterial infection of the external eye. Br J Ophthalmol 1982; 66:357-60.
24. Gigliotti F, Williams WT, Hayden FG, et al. Etiology of acute conjunctivitis in children. J Paediatr 1981; 98:531-56.

25. Sheikh A, Hurwitz B, Cave J. Antibiotics for acute bacterial conjunctivitis. In: The Cochrane Library, Issue 4. Oxford, 1999.
26. Bodor FF. Conjunctivitis-otitis media syndrome: more than meets the eye. *Contemp Pediatr* 1989; 6:55-60.
27. Bowman RJC, Sillah A, Van Dehn C, et al. Operational comparison of single-dose azithromycin and topical tetracycline for trachoma. *Investig Ophthalmol Visu Sci* 2000; 41:4074-9.
28. The WHO Western Pacific Region Gonococcal Antimicrobial Surveillance Programme. Surveillance of antibiotic resistance in *Neisseria gonorrhoeae* in the WHO Western Pacific Region, 1999. *Commun Dis Intell* 2000; 24: 269-71.
29. Centers for Disease Control and Prevention. 1998 Guidelines for the treatment of sexually transmitted diseases. *Morb Mortal Wkly Rep* 1998; 47(RR-1):60.
30. The Loteprednol Etabonate US uveitis Study Group. Controlled evaluation of loteprednol etabonate and prednisolone acetate in the treatment of acute anterior uveitis. *Am J Ophthalmol* 1999; 127:537-44.
31. McClellan K. Topical Eye Preparations. *Current Therapeutics* 1995, January; 61-6.
32. Wolfs RC, Grobbee DE, Hofman A, de Jong PT. Risk of acute angle-closure glaucoma after diagnostic mydriasis in nonselected subjects: the Rotterdam Study. *Invest Ophthalmol Vis Sci* 1997; 38(12):2683-7.
33. Patel KH, et al. Incidence of acute Angle-Closure Glaucoma After Pharmacologic Mydriasis. *Am J of Ophtham* 1995; 120:709-17.
34. Stawell RJ, Hall AJ. Eyes signs in Systemic Disease. *Australian Family Physician* 2002. 31(3):217-22.
35. Australian Medicines Handbook 2002. South Australia: Australian Medicine Handbook Pty Ltd; 375-400.
36. Therapeutic guidelines: Antibiotic version 11 ed. Victoria, Australia: Therapeutic Guidelines Limited, 2000; 47-53.
37. Reese RE, Betts RF, Gumustop. *Handbook of Antibiotics*. 3rd ed. Philadelphia, USA: Lippincott Williams & Wilkins, 2000; 383-98 & 440-5.
38. Walker S, et al. Lack of evidence for systemic toxicity following topical chloramphenicol use. *Eye* 1998; 12:875.
39. Lopez AJ, et al. Ceftriaxone-induced cholelithiasis. *Ann Intern Med* 1991; 115:712.
40. Wilhelmus KR, Coster DJ, Jones BR. Acyclovir and debridement in the treatment of ulcerative herpetic keratitis. *Am J of Ophtham* 1981; 91:323-7.
41. Jensen KB, Nissen SH, Jessen F. Aciclovir in the treatment of herpetic keratitis. *Acta Ophthalmol* 1982; 60:557-63.
42. Parlato CJ, Cohen EJ, Sakauye CM, Dreizen NG, Gakentine PG, Laibson PR. Role of debridement and trifluridine (trifluorothymidine) in herpes simplex dendritic keratitis. *Arch Ophthalmol* 1985; 103:673-5.