Chapter 16

Trauma

EYELID TRAUMA

Haematoma

A haematoma (black eye) is the most common result of blunt eyelid injury, which is innocuous. However, in all patients with black eyes it is very important to exclude the following more serious conditions:

1. **Associated trauma to the globe or orbit** (*see later*). It is easier to examine the integrity of the globe before the lids become oedematous (Figure 16.1).
2. **Orbital roof fracture**, if the black eye is associated with a subconjunctival haemorrhage (Figure 16.2) without a visible posterior limit.
3. **Basal skull fracture**, which may give rise to the characteristic bilateral ring haematomas ('panda eyes').

Laceration

The presence of a lid laceration, however insignificant, mandates careful exploration of the wound and examination of the globe. Lacerations can be divided into the following types:

1. **Superficial laceration** parallel to the lid margin without gaping can be sutured with 6-0 black silk and the sutures are removed after 5 days.
2. **Lid margin laceration** must be very carefully aligned (Figure 16.3) to prevent notching. Repair involves the following steps (Figure 16.4).
   (a) Evaluation for possible tissue loss.
   (b) Sparse trimming of any irregular edges or grossly contaminated tissue.
   (c) Initial eyelid margin re-alignment with a 6-0 black silk suture placed through the meibomian gland orifices (Figure 16.4**a**). The bite should extend 2 mm on each side of the wound edge and be about 2 mm in depth.

Figure 16.1 Severe periocular haematoma

Figure 16.2 Periocular haematoma and subconjunctival haemorrhage

Figure 16.3 Laceration involving the eyelid margin. (**a**) Before suturing; (**b**) after suturing

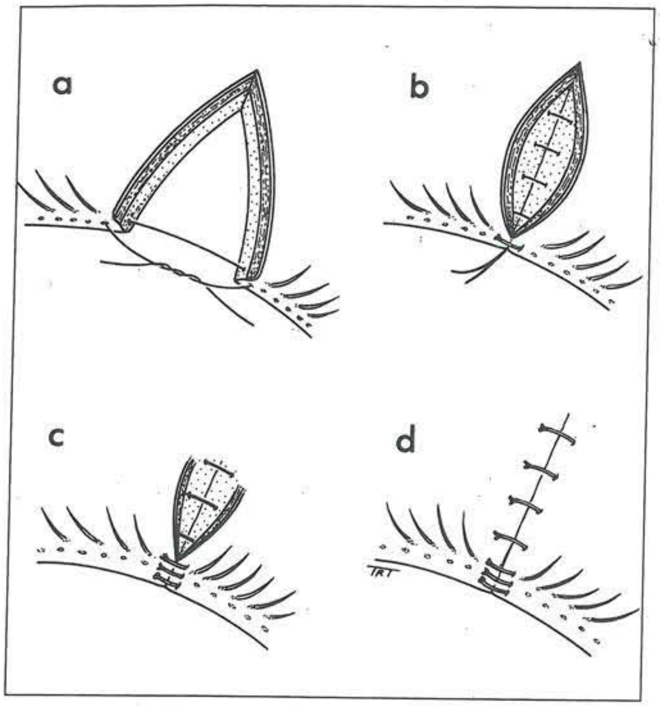

Figure 16.4 Technique of suturing a marginal lid laceration (*see text*)

(d) The tarsal plate is closed using fine absorbable sutures such as 7-0 chromic catgut (Figure 16.4**b**). Polyglactin (Vicryl) or polyglycolic acid (Dexon) may also be used.
(e) The sutures are tied on the anterior surface of the tarsal plate to prevent corneal irritation.
(f) Additional marginal 6-0 silk sutures are placed to achieve precise alignment of the tarsal margin and lashes (Figure 16.4**c**). Their ends are left long and anchored by a skin suture to prevent corneal contact and irritation.
(g) The skin is then closed with multiple interrupted 6-0 black silk sutures (Figure 16.4**d**).

Figure 16.5 Laceration involving the inferior canaliculus

(h) The sutures are left in place for 7–10 days.

3. **Laceration with mild tissue loss** of less than 30% which prevents simple primary closure can usually be managed by performing a lateral cantholysis in order to increase lateral eyelid mobility.
4. **Laceration with severe tissue loss** requires a major reconstruction such as used following lid resection for malignant tumours (*see* Chapter 1).
5. **Canalicular lacerations** (Figure 16.5) should be repaired within 24 hours by locating and approximating the ends of the laceration and bridging the defect with silicone tubing (Figure 16.6), which is left in situ for about 3 months.

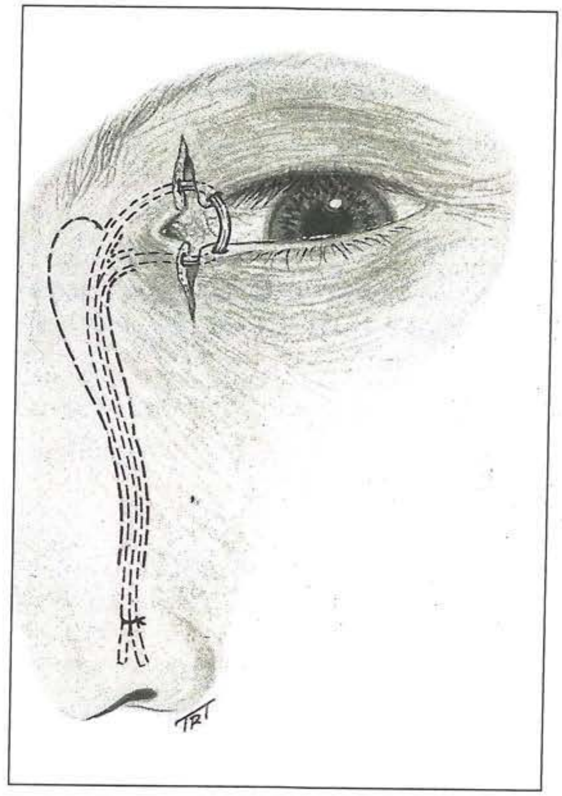

Figure 16.6 Intubation of the lacrimal system following lacerations of upper and lower canaliculi

FRACTURES OF THE ORBIT

Blow-out fracture of the orbital floor

PATHOGENESIS

A 'pure' blow-out fracture of the orbit does not involve the orbital rim whereas an 'impure' fracture involves the orbital rim and adjacent facial bones. A blow-out fracture of the orbital floor is typically caused by a sudden increase in the orbital pressure by a striking object which is >5 cm in dia-

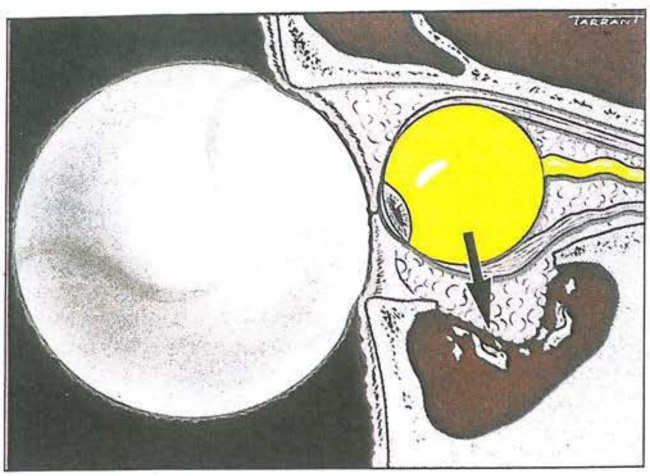

Figure 16.7 Mechanism of floor blow-out fracture

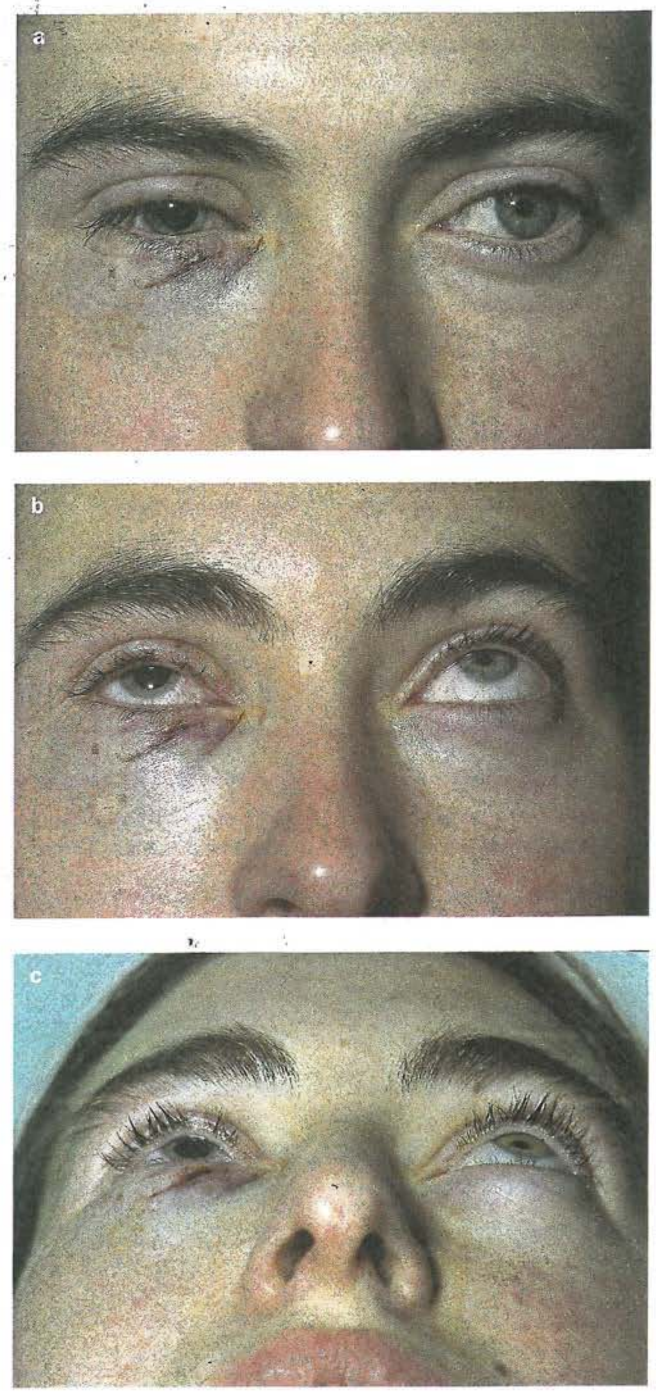

Figure 16.8 Right floor blow-out fracture. (**a**) Mild bruising of eyelids and dilated pupil following examination of the fundus; (**b**) defective elevation of the right eye; (**c**) mild right enophthalmos

meter, such as a fist or tennis ball (Figure 16.7). Because the bones of the lateral wall and the roof are usually able to withstand a sudden and severe raised intraorbital pressure, the fracture most frequently involves the floor of the orbit along the thin bone covering the infraorbital canal. Occasionally, the medial orbital wall may also be fractured. The clinical features vary according to the severity of trauma and the time interval between the fracture and the examination.

SIGNS

1. **Periocular signs** include variable ecchymosis and oedema (Figure 16.8**a**).
2. **Infraorbital nerve anaesthesia** involving the lower lid, cheek, side of nose, upper lip, upper teeth and gums is very common because a blow-out fracture frequently involves the infraorbital canal.
3. **Diplopia** may be caused by one of the following mechanisms:
   (a) Haemorrhage and oedema of the orbital fat may cause the septa, which connect the inferior rectus and inferior oblique muscles to the periorbita, to become taut and thus restrict movements of the globe. Ocular motility usually improves as the haemorrhage and oedema are absorbed.
   (b) Mechanical entrapment within the fracture of the inferior rectus or inferior oblique muscle, or adjacent connective tissue and fat. The diplopia typically occurs in both upgaze (Figure 16.8**b**) and downgaze (double diplopia). In these cases the forced duction test and the differential intraocular pressure tests are positive. The diplopia may subsequently improve if it is caused mainly by entrapment of connective tissue and fat within the fracture, but it usually persists if there is significant involvement of the muscles themselves.
   (c) Direct injury to an extraocular muscle is associated with a negative forced duction test. The muscle fibres usually regenerate and normal function returns within about 2 months.

4. **Ocular damage** (e.g. hyphaema, angle recession, retinal dialysis), although uncommon, should be excluded by careful slitlamp and fundus examination.
5. **Enophthalmos** (Figure 16.8c) may be present if the fracture is severe. In the absence of surgical intervention, enophthalmos may continue to increase for about 6 months as post-traumatic orbital degeneration and fibrosis develop.

INVESTIGATIONS

1. **CT** with coronal sections (Figure 16.9) is particularly useful in evaluating the extent of the fracture, as well as determining the nature of antral soft-tissue densities which may represent prolapsed orbital fat, extraocular muscles, haematoma or unrelated antral polyps.
2. **Hess test** is useful in assessing and following the progression of diplopia (Figure 16.10).
3. **Field of binocular vision** can be assessed on the Lister or Goldmann perimeter.

TREATMENT

1. **Initial treatment** is conservative with systemic antibiotics if the fracture involves the maxillary sinus.

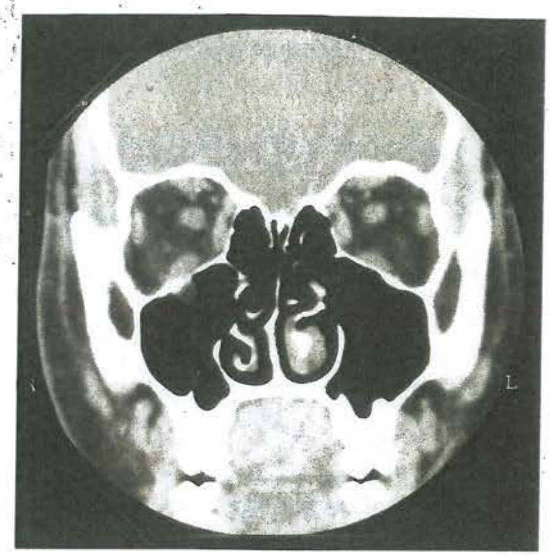

Figure 16.9 Coronal CT scan of a right floor blow-out fracture showing the 'tear drop' sign

The patient should also be instructed not to blow the nose.

2. **Subsequent treatment** is aimed at prevention of permanent vertical diplopia and/or cosmetically unacceptable enophthalmos. The three factors that determine the risk of these late complications are: fracture

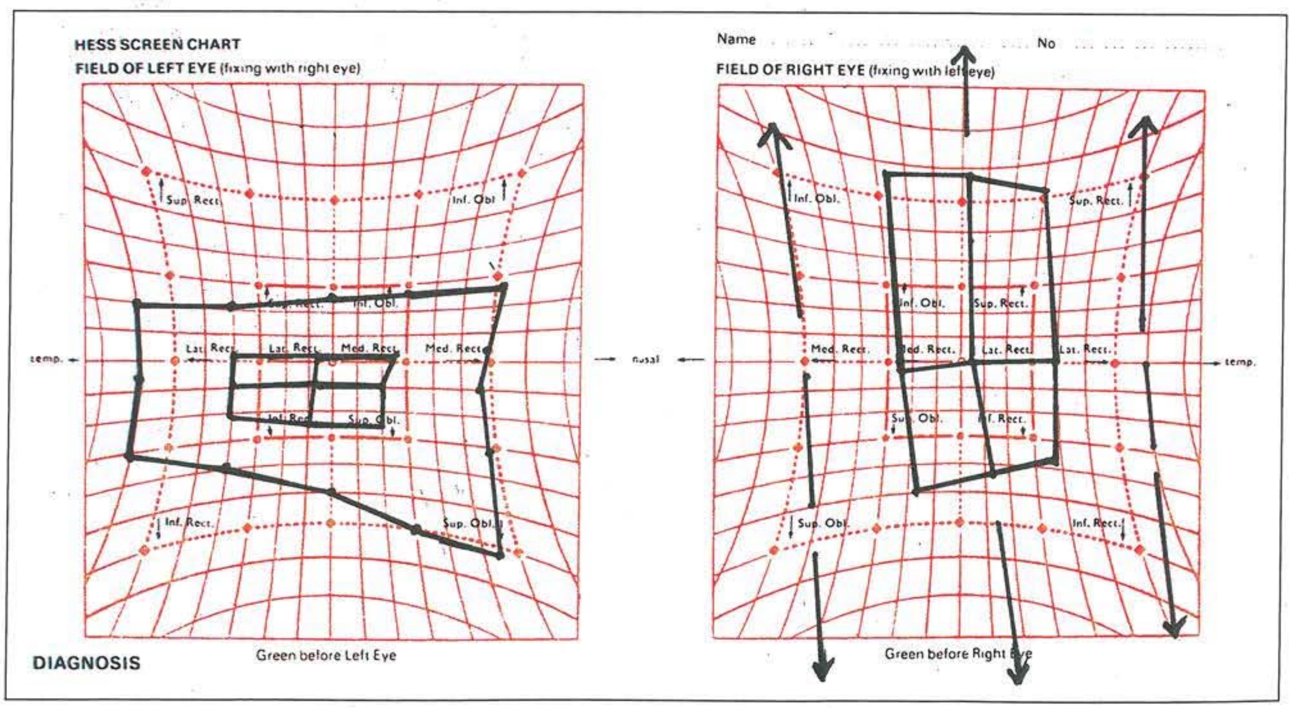

Figure 16.10 Hess chart of patient with a right floor blow-out fracture showing restriction of right upgaze (superior rectus and inferior oblique) and restriction on downgaze (inferior rectus). There is also considerable secondary overaction of the left eye

size, herniation of orbital contents into the maxillary sinus and muscle entrapment. Although there may be some overlap, most fractures will fall into one of the following categories:

(a) Small cracks not associated with herniation. These do not require treatment as the risk of permanent complications is negligible.

(b) Fractures involving less than half of the orbital floor, with little or no herniation, and rapidly improving diplopia. These also do not require treatment unless more than 2 mm of enophthalmos develops.

(c) Fractures involving half or more of the orbital floor with entrapment of orbital contents, and persistent and significant diplopia in the primary position. These should be repaired within 2 weeks. If surgery is delayed, the results are less satisfactory because secondary fibrotic changes develop in the orbit.

3. **Technique of surgical repair**

(a) A transconjunctival or subciliary incision is made (Figure 16.11**a**).

(b) The periosteum is elevated from the floor of the orbit and all entrapped orbital contents are removed from the antrum (Figure 16.11**b**).

(c) The defect in the floor is repaired by using synthetic material such as Supramid, silicone or Teflon (Figures 16.11**c**).

(d) The periosteum is sutured (Figure 16.11**d**).

Figure 16.12 is a coronal CT scan showing the postoperative appearance following repair of a right blow-out fracture with a plastic implant.

Blow-out fracture of the medial wall

Most medial wall orbital fractures are associated with floor fractures (Figure 16.13). Isolated fractures are less common.

1. **Signs**

(a) ***Periorbital subcutaneous emphysema*** (Figure 16.14), which typically develops when the patient blows the nose. Because of the possibility of forcing infected sinus contents into the orbit, blowing of the nose should be discouraged.

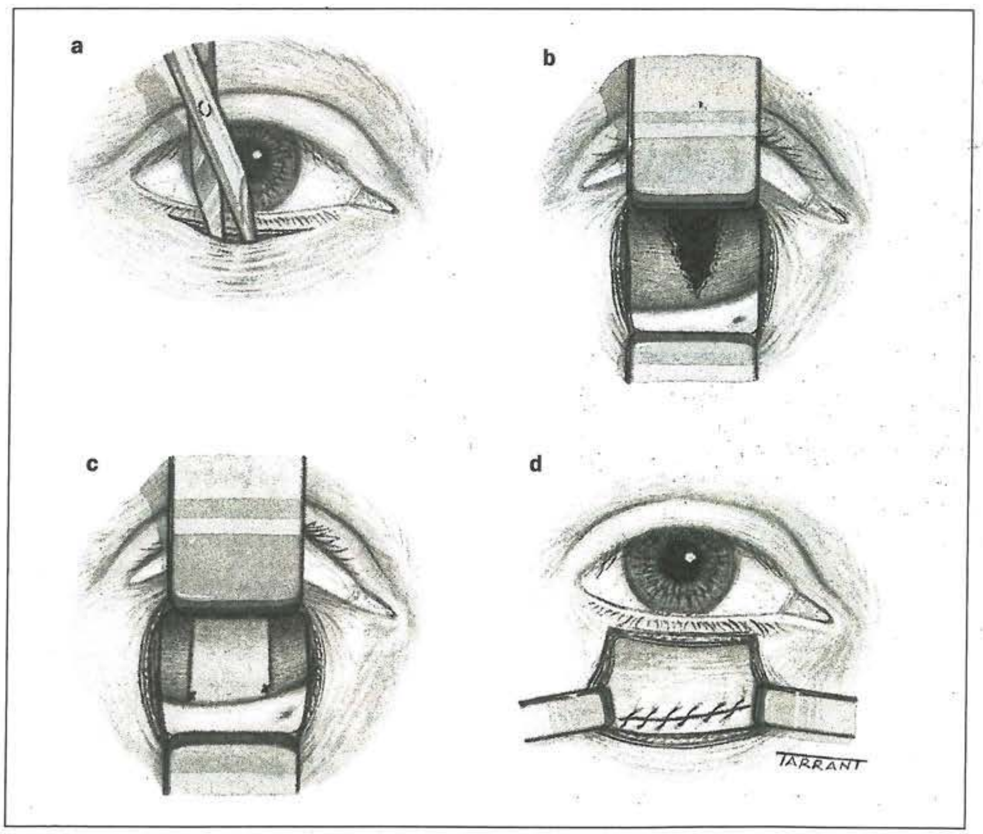

Figure 16.11 Technique of repair of a floor blow-out fracture (*see text*)

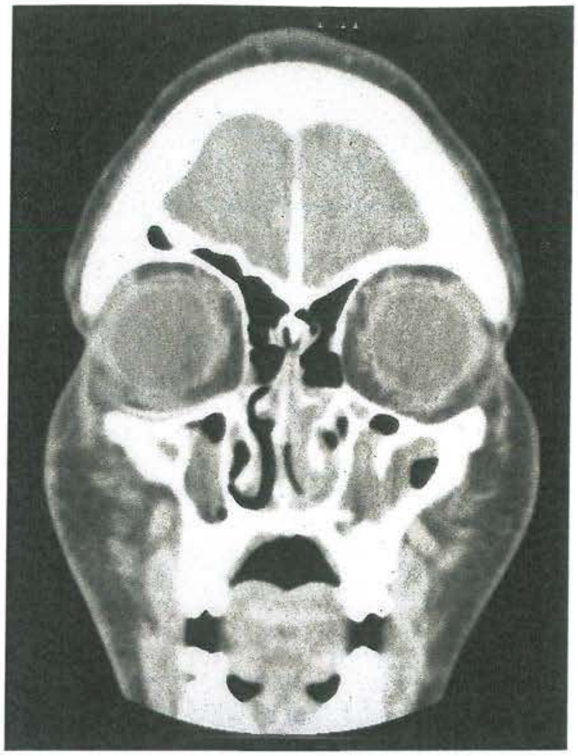

Figure 16.12 Coronal CT scan following repair of a right floor blow-out fracture with synthetic material

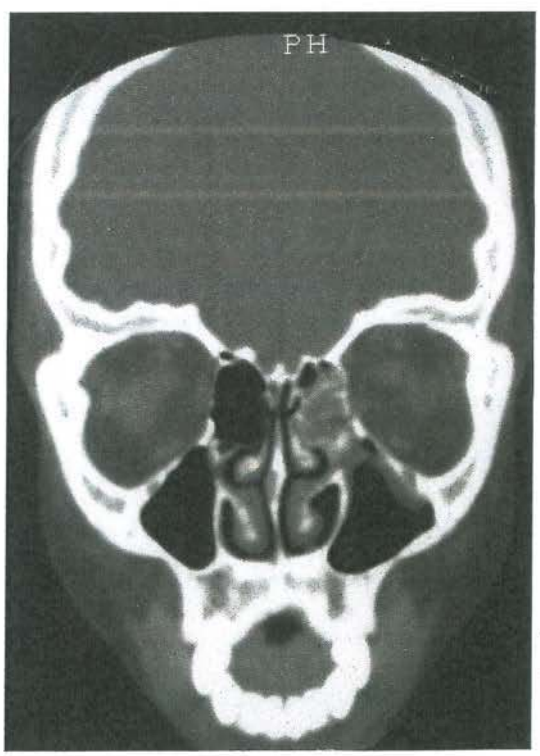

Figure 16.13 Coronal CT scan showing blow-out fractures of the left medial wall and floor

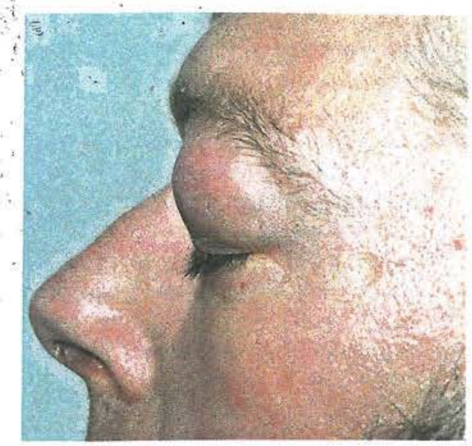

Figure 16.14 Subcutaneous emphysema in a patient with a medial wall blow-out fracture

(b) ***Defective motility*** involving adduction and abduction (Figure 16.15), if the medial rectus muscle is entrapped in the fracture. In some cases associated retraction of the globe and narrowing of the palpebral fissures may resemble Duane syndrome type III.

2. **Treatment** involves release of the entrapped tissue and repair of the bony defect.

Figure 16.15 Limitation of left abduction due to entrapment of the left medial rectus muscle in a medial wall blow-out fracture

Roof fracture

Roof fractures are rarely encountered by ophthalmologists. Isolated fractures, caused by minor trauma such as falling on a sharp object or a blow to the brow or forehead, are most common in young children. Complicated fractures, caused by major trauma with associated displacement of the orbital rim or significant disturbance of other craniofacial bones, most commonly affect adults.

1. **Presentation** is with a haematoma of the upper eyelid and periocular ecchymosis which develops after a few hours and may later spread to the opposite side (Figure 16.16).
2. **Signs**
   (a) Inferior or axial displacement of the globe.
   (b) Large fractures may be associated with pulsation of the globe which is not associated with a bruit.
3. **Treatment**
   (a) Small fractures may not require treatment but it is important to observe the patient for the possibility of a CSF leak which may lead to meningitis.
   (b) Sizeable bony defects with downwardly displaced fragments usually require reconstructive surgery.

Figure 16.16 Bilateral periocular ecchymosis

Lateral wall fracture

Acute lateral wall fractures (Figure 16.17) are rarely encountered by ophthalmologists. The lateral wall of the orbit consists of more solid bone than the other walls, so that a fracture is usually associated with extensive facial damage.

Figure 16.17 Axial CT scan showing a right lateral wall fracture with bony fragments impinging on the optic nerve

TRAUMA TO THE GLOBE

Introduction

DEFINITIONS

The two types of trauma to the globe are blunt and penetrating.

1. **Closed injury** is one in which the cornea and sclera are intact but there is intraocular damage.
2. **Open injury** is associated with a full-thickness wound of the cornea or sclera, or both.
3. **Rupture** is a full-thickness wound caused by blunt trauma.
4. **Laceration** is a full-thickness injury caused by a sharp object.
5. **Penetration** is caused by a single laceration with a sharp object.
6. **Perforation** consists of two lacerations, one entry and one exit.

GENERAL PRINCIPLES OF MANAGEMENT

1. **Initial assessment** should be performed in the following order:
   (a) Determination of the nature and extent of any life-threatening problems.
   (b) History of the injury, including the circumstances, timing and likely object.
   (c) Thorough examination of both eyes and orbits.
2. **Special investigations**
   (a) ***Plain X-rays*** should be performed when a foreign body is suspected (Figures 16.18 and 16.19).
   (b) ***CT scanning*** may be useful in the detection of foreign bodies (Figure 16.20). It should be emphasized that MRI scanning should not be performed if a metallic foreign body is suspected.

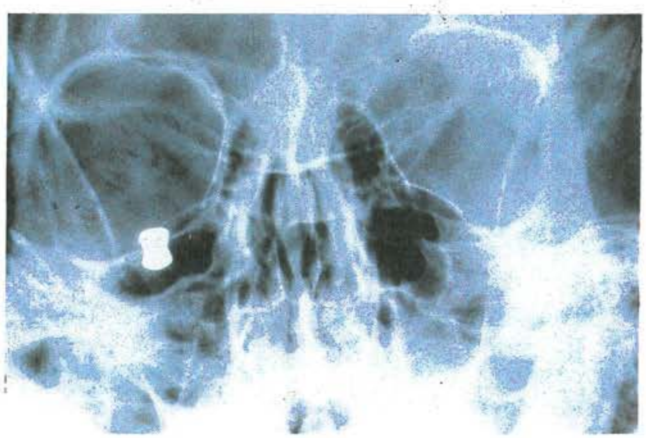

Figure 16.18 Plain radiograph showing a right airgun pellet

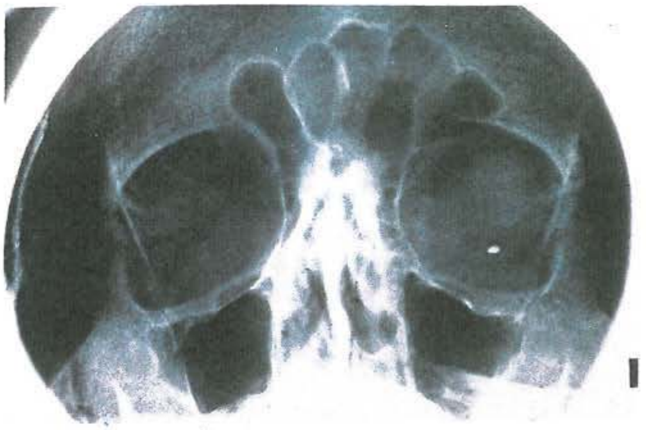

Figure 16.19 Plain radiograph showing a left foreign body

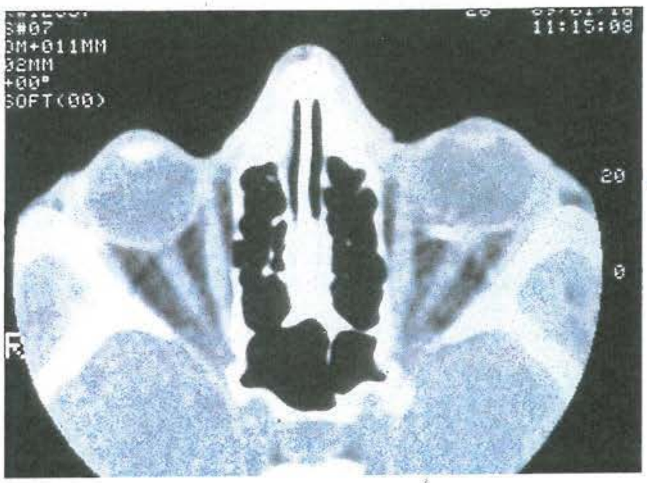

Figure 16.20 Axial CT scan showing a foreign body near the left optic nerve

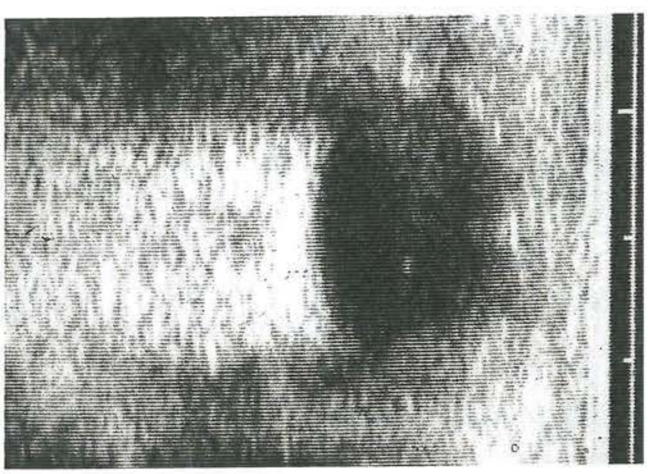

Figure 16.21 B-scan ultrasonograph showing an intraocular foreign body

(c) ***Ultrasonography*** may be useful in the detection of intraocular foreign bodies (Figure 16.21), globe rupture, suprachoroidal haemorrhage (Figure 16.22) and retinal detachment. It is also helpful in planning surgical repair, for example regarding placement of infusion ports during vitrectomy and whether drainage of suprachoroidal haemorrhage is required.

(d) ***Electrophysiological tests*** may be useful in assessing the integrity of the optic nerve and retina, particularly if some time has passed since the original injury and there is suspicion of a retained intraocular foreign body.

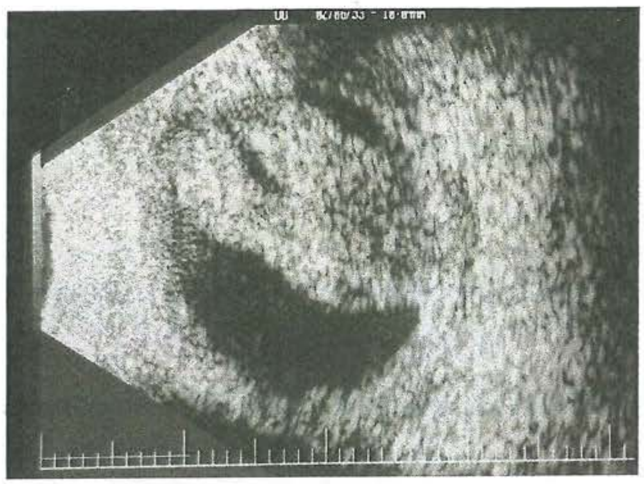

Figure 16.22 B-scan ultrasonograph showing a suprachoroidal haemorrhage

Blunt trauma

CAUSES AND PATHOGENESIS

The most common causes of blunt trauma are squash balls, elastic luggage straps, champagne corks and airgun pellets. Severe blunt trauma to the globe results in a decrease in anteroposterior diameter and a simultaneous expansion at the equatorial plane which is associated with a short-lived increase in intraocular pressure. Although the impact is primarily absorbed by the lens–iris diaphragm and the vitreous base, damage can also occur at a distant site such as the posterior pole. The extent of damage to the globe depends on the severity of trauma.

ANTERIOR SEGMENT COMPLICATIONS

1. **Hyphaema** (Figure 16.23) is a common complication of blunt trauma. The source of the bleeding is the iris or ciliary body. Eyes with hyphaemas may shows other signs of anterior segment damage such as

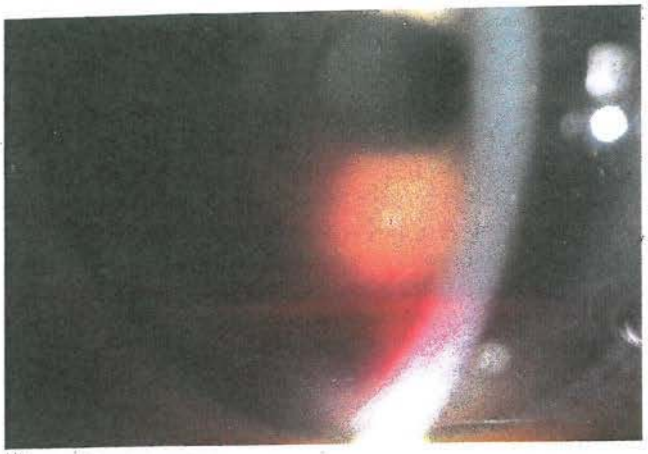

Figure 16.23 Traumatic hyphaema

tears of the sphincter pupillae (Figure 16.24), iridodialysis (Figure 16.25), pigment (Vossius) ring on the anterior lens surface (Figure 16.26), cataract (Figure 16.27), lens subluxation or dislocation (Figure 16.28) and angle recession (Figure 16.29). Although most

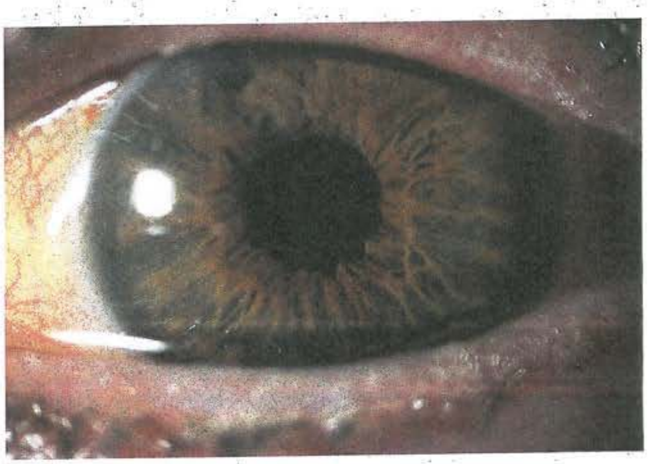

Figure 16.24 Rupture of the iris sphincter

Figure 16.25 Large inferior iridodialysis

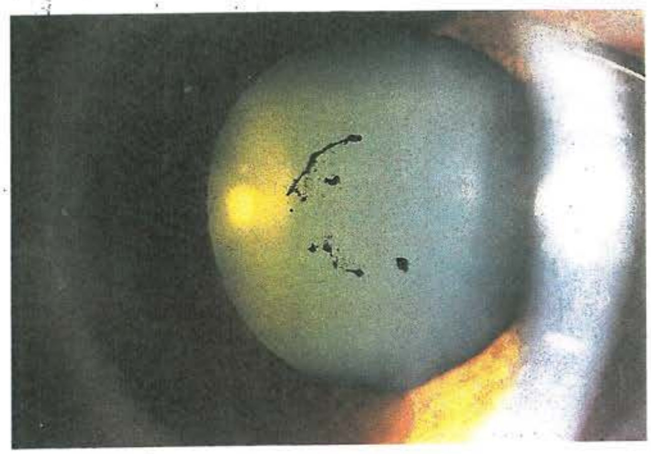

Figure 16.26 Iris pigment on anterior lens capsule

Figure 16.27 Post-traumatic flower-shaped cataract

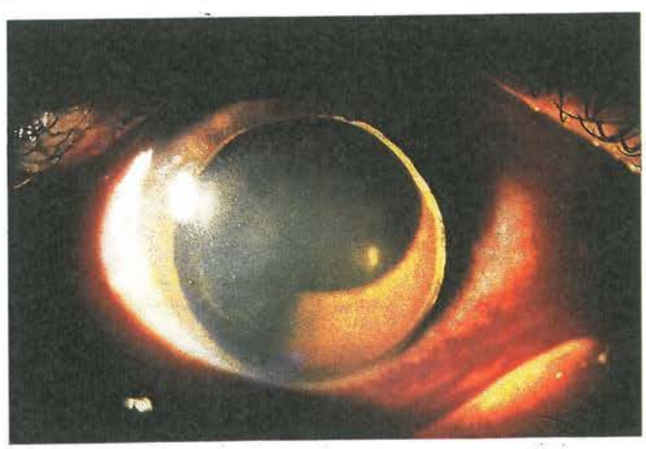

Figure 16.28 Lens dislocation into the anterior chamber

traumatic hyphaemas are relatively innocuous and transient, some may give rise to severe and prolonged elevation of intraocular pressure which may damage the optic nerve head and cause bloodstaining of the

Figure 16.29 Severe angle recession

cornea. The management of acute hyphaema is discussed in Chapter 6.

2. **Rupture of the globe** may result from very severe blunt trauma. The rupture is usually anterior, at or near the limbus with prolapse of uveal tissue. Occasionally, the rupture is posterior (occult) with little damage to the anterior segment. Clinically, occult rupture should be suspected if there is asymmetry of anterior chamber depth and intraocular pressure in the affected eye is low. The principles of repairing scleral ruptures are described below.

POSTERIOR SEGMENT COMPLICATIONS

Severe blunt trauma may result in the following posterior segment complications which may be peripheral or central.

1. **Commotio retinae** is caused by concussion to the neuroretina resulting in cloudy swelling. It is characterized by a grey appearance, which most frequently affects the temporal fundus (Figure 16.30) but may also involve the macula (Figure 16.31). Mild commotio is usually innocuous and resolves without sequelae. More severe involvement may be associated with intraretinal haemorrhage and the subsequent development of atrophic changes including macular hole formation.

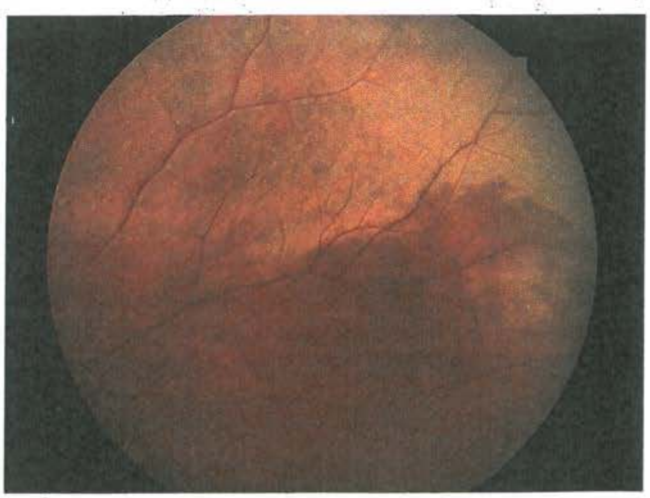

Figure 16.30 Peripheral commotio retinae

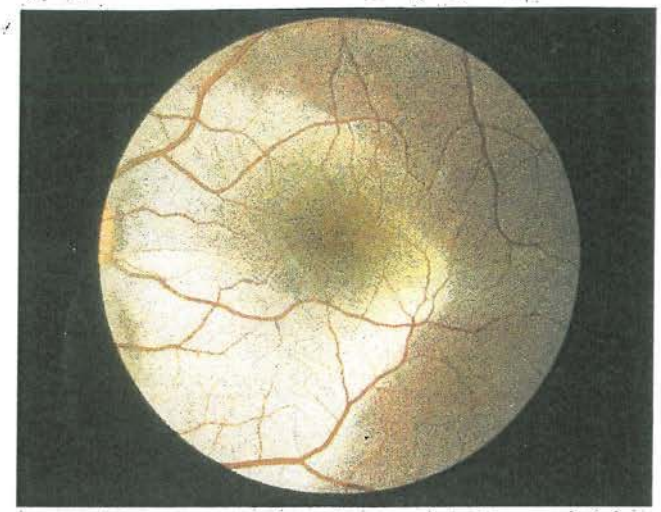

Figure 16.31 Commotio retinae involving the macula

2. **Choroidal rupture**, which may be multiple, is characteristically concentric with the optic disc and frequently involves the fovea (Figure 16.32). Small ruptures may not be associated with haemorrhage but more extensive ruptures are frequently associated with subretinal haemorrhage (Figure 16.33). In some cases the haematoma may rupture through the internal limiting membrane and result in subhyaloid or vitreous haemorrhage. Old choroidal ruptures are

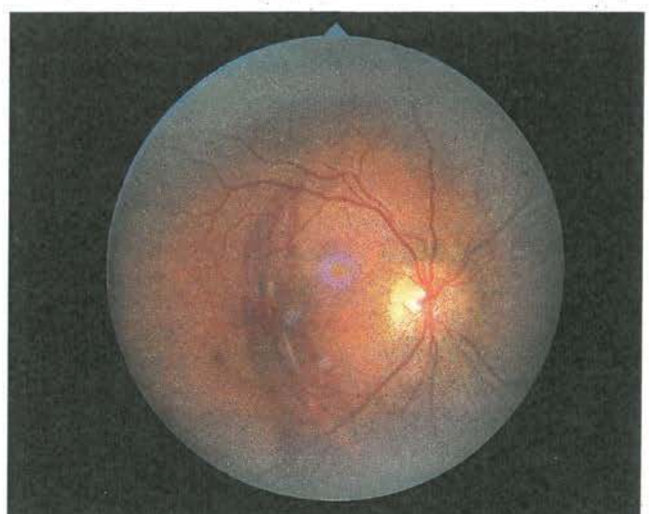

Figure 16.32 Acute choroidal rupture and subretinal haemorrhage involving the fovea

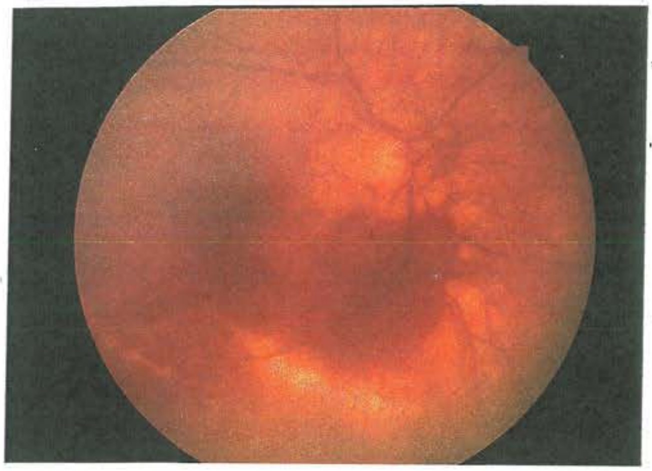

Figure 16.33 Severe acute choroidal rupture with extensive subretinal haemorrhage

characterized by a white crescentic streak of exposed underlying sclera (Figure 16.34). A late complication of choroidal rupture is secondary choroidal neovascularization which may result in bleeding and further visual deterioration.

3. **Retinal breaks,** which may lead to retinal detachment, are of three main types:
   (a) ***Retinal dialyses*** (Figure 16.35), are caused by traction by the relatively inelastic vitreous gel along the posterior aspect of the vitreous base. In some cases the vitreous base becomes avulsed giving rise to a 'bucket-handle' appearance (Figure 16.36) which comprises a strip of ciliary epithelium, ora serrata and immediate post-oral retina into which basal vitreous gel remains inserted. Traumatic dialyses may occur in any quadrant but are most frequent in the upper nasal. Although dialyses occur at the time of injury, any subsequent retinal detachment usually does not develop until several months

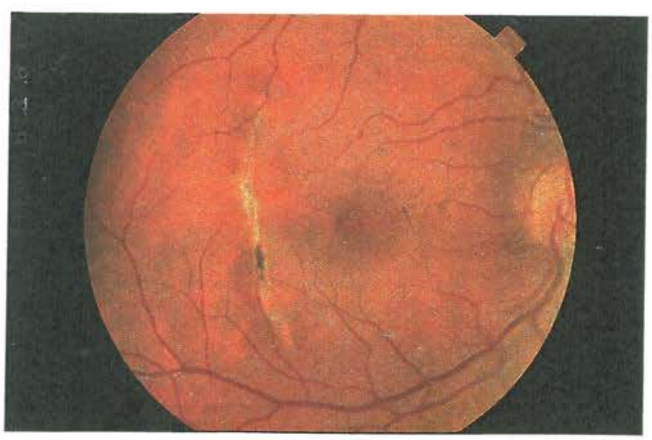

Figure 16.34 Old choroidal rupture

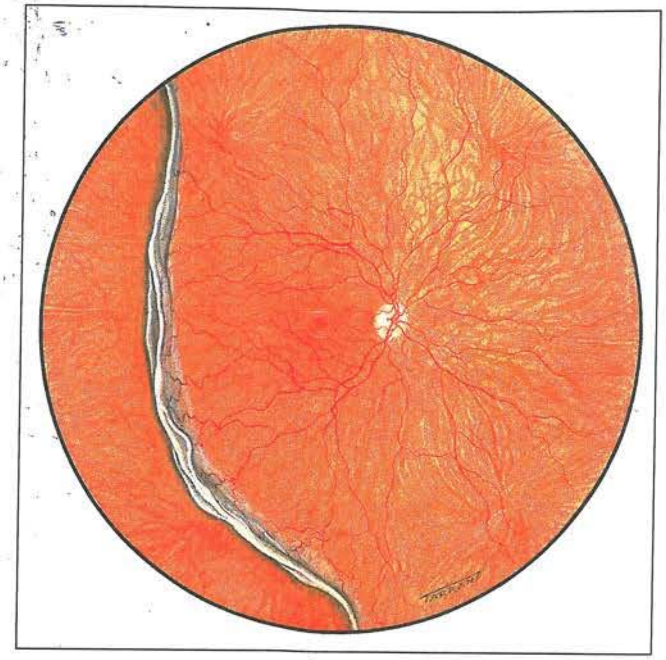

Figure 16.35 Giant traumatic retinal dialysis

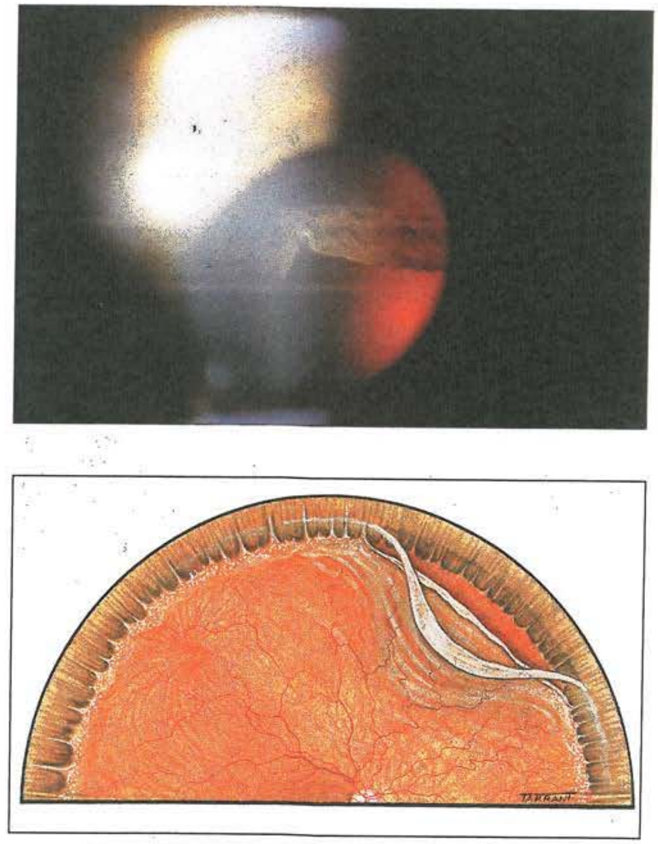

Figure 16.36 Avulsion of the vitreous base giving rise to a 'bucket handle' appearance

later. Progression is usually slow, probably because the vitreous gel is healthy.

(b) ***Equatorial tears*** are less frequent and are caused by direct retinal disruption at the point of scleral impact and may occasionally extend for more than one quadrant (giant tears, Figure 16.37).

(c) ***Macular holes*** (Figure 16.38) may occur either at the time of injury or later following the resolution of commotio retinae.

4. **Optic nerve damage,** contusion or rarely avulsion, may occur after sudden rotation of the globe.

Figure 16.37 Retinal detachment caused by a traumatic giant retinal tear

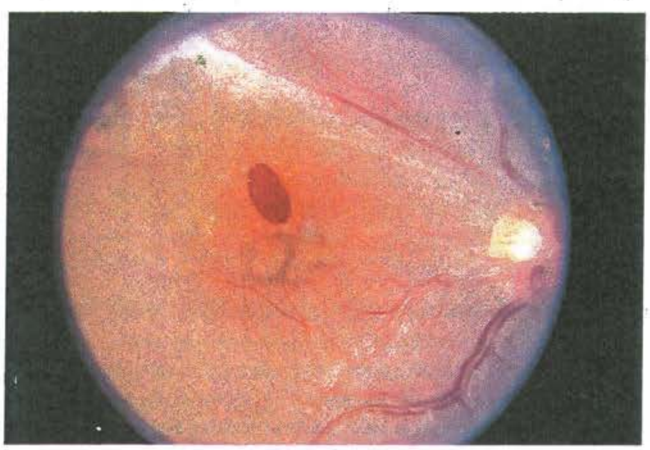

Figure 16.38 Post-traumatic macular hole

Penetrating trauma

CAUSES AND PATHOGENESIS

Penetrating injuries are more common in males than females by a 3:1 ratio, and in the younger age group. The most common causes are assault, domestic accidents and sport. The extent of the injury is determined by the size of the object, its speed at the time of impact and its composition. Sharp objects such as knives will cause well-defined lacerations of the globe. However, the extent of damage caused by flying foreign bodies is determined by their kinetic energy. For example, an airgun pellet is large and although relatively slow moving has a high kinetic energy and can thus cause considerable ocular damage. In contrast, a fast moving fragment of shrapnel has a low mass and therefore will cause a well-defined laceration with relatively less intraocular damage than an air gun pellet. Tractional retinal detachment following penetrating trauma is the result of vitreous incarceration in the wound and the presence of blood within the vitreous gel which acts as a stimulus to fibroblastic proliferation along the planes of incarcerated vitreous (Figure 16.39). The contraction of such membranes leads to shortening and a rolling effect on the peripheral retina in the region of the vitreous base and eventually to tractional retinal detachment.

Figure 16.39 Fibrous proliferation following penetrating trauma

PRINCIPLES OF PRIMARY REPAIR

The technique of primary repair depends on the severity of the wound and the presence of complications such as iris incarceration, flat anterior chamber and damage to intraocular contents.

1. **Small corneal laceration** may not require suturing as it may heal spontaneously or with the aid of a soft bandage contact lens.
2. **Medium-sized corneal laceration** usually requires suturing, especially if the anterior chamber is shallow or flat (Figure 16.40). If the laceration involves the limbus it is very important to expose the adjacent sclera to assess the severity of any scleral extension. A shallow anterior chamber may re-form spontaneously once the cornea has been sutured, if not it should be reformed with balanced salt solution. A postoperative bandage contact lens (Figure 16.41)

Figure 16.40 Flat anterior chamber following a corneal laceration

Figure 16.41 Bandage contact lens following repair of a corneal laceration

may also be useful for a few days to ensure that the anterior chamber remains deep.

3. **Corneal laceration with iris incarceration.** The management depends on the duration and extent of incarceration. A small peak of recently incarcerated iris may be replaced by constricting the pupil with intracameral Miochol. Large incarcerations of iris should be abscised, especially if the iris appears non-viable.
4. **Corneal laceration with lens damage** (Figure 16.42) is treated by suturing the laceration and also removing the lens. The latter can be performed by phacoemulsification or with a vitreous cutter. The latter is preferred if there is associated vitreous involvement.
5. **Anterior scleral laceration** that does not pass posterior to the insertions of the extraocular muscles has a better prognosis than a more posterior wound involving the retina. Nevertheless, anterior scleral wounds may be associated with serious complications such as uveal prolapse and vitreous incarceration. The latter, unless appropriately managed, may result in subsequent vitreoretinal traction and retinal detachment.

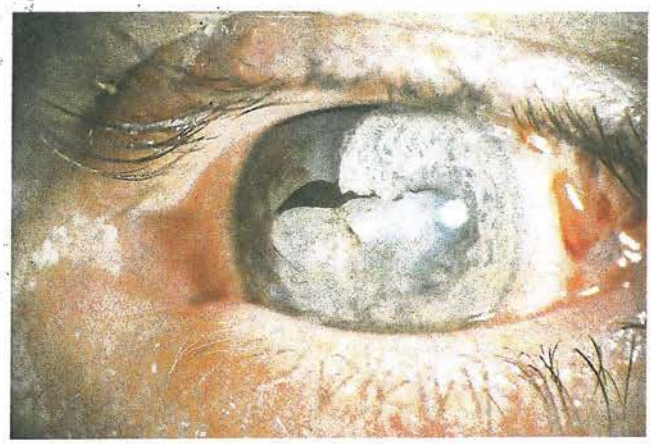

Figure 16.42 Severe corneal laceration associated with a large iridodialysis and cataract

Every attempt should be made to reposit exposed viable uveal tissue and cut prolapsed vitreous flush with the wound. Cellulose sponges should not be used to remove vitreous for fear of inducing vitreous traction. The scleral wound is closed, starting anteriorly and working posteriorly.

6. **Posterior scleral lacerations** are frequently asssociated with retinal breaks unless the laceration is very superficial. During repair it is very important not to exert excessive pressure and traction on the eye to prevent or minimize loss of intraocular contents. It may also be necessary to treat retinal breaks prophylactically.

AIMS OF SECONDARY REPAIR

Secondary repair of posterior segment trauma, if appropriate, is usually carried out 10–14 days after the primary repair. This allows time not only for healing of wounds but also for the development of posterior vitreous separation which facilitates closed intraocular microsurgery. The main aims of secondary repair are:

1. To clear opacities of the media such as cataract and vitreous haemorrhage to improve vision.
2. To stabilize abnormal vitreoretinal interactions and thereby prevent long-term sequelae such as tractional retinal detachment.

Management of intraocular foreign bodies

PREOPERATIVE ASSESSMENT

1. **Accurate history** is vital in determining the origin of the foreign body and it may be helpful for the patient to bring any causative objects such as a chisel. There may be associated signs such as lid laceration and damage to anterior segment structures.

2. **Localization** of the foreign body is performed with reference to a radio-opaque marker. The suction contact lens method which contains an opaque ring is accurate provided centring is correct (Figure 16.43).

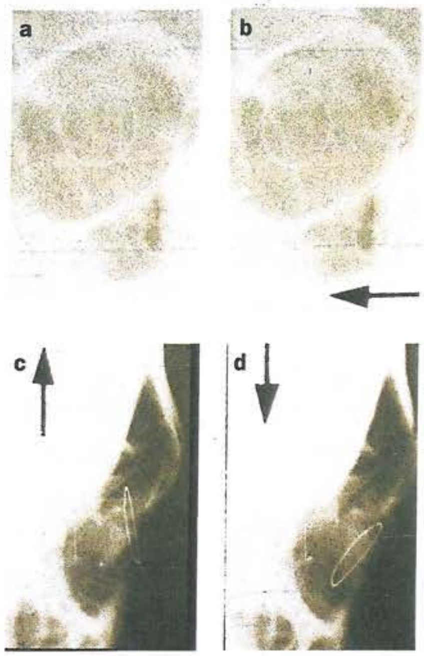

Figure 16.43 Method of localization of an intraocular foreign body using a ring marker. (**a-b**) Postero-anterior views; (**c-d**) lateral views with the patient looking up and down to assess the position of the foreign body in relation to the marker

TECHNIQUE OF REMOVAL

The technique of removing an intraocular foreign body depends on its location as follows:

1. Magnetic foreign bodies within the vitreous cavity that are not adjacent to or on the retina are removed as follows. A sclerotomy is performed approximately 2 clock hours away from the position of the foreign body which is then removed with a magnet. The spacing allows alignment or spinning of the foreign body within the magnetic field without the risk of causing damage to the retina.
2. Foreign bodies adjacent to or on the retina are removed by performing a pars plana vitrectomy, freeing the foreign body from the retina and removing it with forceps. During vitrectomy it is important to make a careful search for any ricochet sites which may have resulted in vitreoretinal incarceration and retinal break formation.
3. Subretinal foreign bodies may be removed either externally through a scleral trap-door incision or internally by gaining access to the foreign body by performing a retinotomy and removing it with forceps.

Enucleation and sympathetic ophthalmitis

ENUCLEATION

Primary enucleation should be performed only in very severe injuries when it is impossible to repair the sclera (Figure 16.44). Secondary enucleation should be considered following primary repair if it is considered that the eye is severely and irreversibly damaged, particularly if it is also unsightly and uncomfortable. Theoretically, enucleation should be performed within 10 days of the original injury in order to prevent the very remote possibility of sympathetic ophthalmitis.

Figure 16.44 Extremely damaged globe where primary enucleation is justifiable

SYMPATHETIC OPHTHALMITIS

Sympathetic ophthalmitis is a very rare, *bilateral*, granulomatous panuveitis which occurs after penetrating ocular trauma, which is usually associated with uveal prolapse or, less frequently, following intraocular surgery. The traumatized eye is referred to as the *exciting* eye and the fellow eye, which also develops uveitis, is called the *sympathizing* eye. Sixty-five per cent of cases of sympathetic uveitis develop between 2 weeks and 3 months after the initial injury and 90% of all cases occur within the first year.

1. **Clinical features**
   (a) ***Anterior segment.*** The exciting eye shows evidence of the initial trauma and is frequently excessively red and irritable. The sympathizing eye becomes photophobic and irritable and then both eyes develop a chronic granulomatous anterior uveitis with iris nodules and mutton fat keratic precipitates (Figure 16.45).
   (b) ***Posterior segment*** involvement is characterized by optic disc swelling and multifocal choroiditis (Figure 16.46) involving the entire fundus. In a

Figure 16.45 Mutton fat keratic precipitates

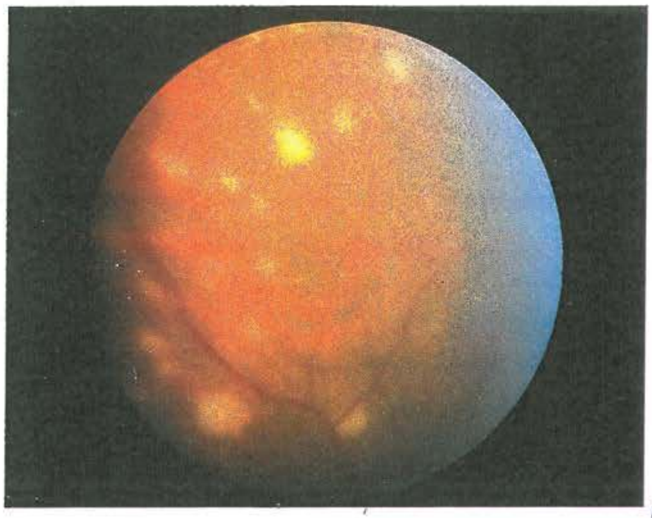

Figure 16.46 Multifocal choroiditis in sympathetic ophthalmitis

few cases the uveitis has a relatively mild and self-limiting course. In most, however, the intraocular inflammation becomes chronic and, unless appropriately treated, it may lead to cataract, glaucoma and phthisis bulbi.

2. **Treatment** is initially with topical and systemic steroids. Immunosuppressive therapy may be required in severe steroid-resistant cases.

CHEMICAL INJURIES

Introduction

CAUSES

Although chemical injuries are frequently relatively trivial, some are potentially blinding. The majority are accidental and a few the result of assault. About two-thirds of accidental burns occur at work and the remainder at home. Alkali burns are twice as common as acid burns since alkalis are more widely used at home and in industry. The most common involved alkalis are ammonia, sodium hydroxide and lime. The commonest acids implicated are sulphuric, sulphurous, hydrofluoric, acetic, chromic and hydrochloric.

PATHOPHYSIOLOGY

The severity of a chemical injury is related to the properties of the chemical, the area of affected ocular surface, retention of particulate chemical on the surface of the globe and related effects such as thermal burning. Ammonia and sodium hydroxide may produce severe damage because of rapid penetration. Hydrofluoric acid used in glass etching and cleaning also tends to rapidly penetrate the eye whilst sulphuric acid may be complicated by thermal and high velocity impact after car battery explosions.

1. **Damage** to the ocular structures by severe chemical injuries occurs in the following order:
   (a) Necrosis of the conjunctival and corneal epithelial cells with disruption and occlusion of the limbal vasculature. Loss of limbal stem cells may subsequently result in conjunctivalization and vascularization of the corneal surface or persistent epithelial defects with sterile corneal ulceration and corneal perforation. Other long-term effects are ocular surface wetting disorders, symblepharon formation and cicatricial entropion.
   (b) Deeper penetration causes breakdown and precipitation of glycosaminoglycans and stromal corneal opacification.
   (c) Anterior chamber penetration, which results in iris and lens damage.
   (d) Ciliary epithelial damage impairs secretion of ascorbate which is required for collagen production and corneal repair.
   (e) Hypotony and phthisis bulbi may follow.
2. **Healing** occurs in the corneal epithelium and stroma as follows:
   (a) The epithelium heals by migration of epithelial cells which originate from limbal stem cells.
   (b) Damaged stromal collagen is phagocytosed and new collagen is synthesized.

Management

EMERGENCY TREATMENT

A chemical burn is the only type of eye injury that requires immediate treatment without first taking a history and performing a careful examination. Immediate treatment is as follows:

1. Irrigation is crucially important in minimizing duration of contact of the chemical with the eye and normalizing the pH as soon as possible. Normal saline (or equivalent) should be used to irrigate the eye for 15–30 minutes or until pH is normalized.
2. The eyelids should be double-everted so that any retained particulate matter trapped in the fornices, such as lime or cement, is removed.
3. Necrotic areas of corneal epithelium should be debrided to allow for proper re-epithelialization.

GRADING OF SEVERITY

Acute chemical injuries are then graded to allow appropriate subsequent treatment and an indication of likely ultimate prognosis. This is done by observing the clarity of the cornea and severity of limbal ischaemia. The latter is assessed by observing the patency of the deep and superficial vessels supplying the limbus. Limbal ischaemia is characterized by blanching and stasis of blood cells (Figure 16.47). The severity of damage is then graded as follows:

1. **Grade I**: clear cornea and no limbal ischaemia (excellent prognosis).
2. **Grade II**: hazy cornea but visible iris details and less than one-third of limbal ischaemia (good prognosis).
3. **Grade III**: no iris details and between one-third and half of limbal ischaemia (guarded prognosis).
4. **Grade IV**: opaque cornea and more than half of limbal ischaemia (very poor prognosis).

Figure 16.47 Severe limbal ischaemia following a recent chemical injury

Other features that should be noted at initial assessment are the extent of corneal and conjunctival epithelial loss, iris changes, status of the lens and intraocular pressure.

MEDICAL TREATMENT

Mild (grade I) injuries are treated with a short course of topical steroids, cycloplegics and prophylactic antibiotics for about 7 days. The main aims of treatment of more severe burns are to reduce inflammation, promote epithelial regeneration and prevent corneal ulceration.

1. **Steroids** reduce inflammation and neutrophil infiltration. However, they also impair stromal healing by reducing collagen synthesis and inhibiting fibroblasts migration. For this reason topical steroids are used initially but must be tailed off after 7–10 days when sterile corneal ulceration is likely to occur. After this period they may be replaced by topical non-steroidal anti-inflammatory agents which do not affect keratocyte function.
2. **Ascorbic acid** is essential for collagen production and is normally secreted by the ciliary epithelium which may be damaged in severe cases. It is therefore important to administer ascorbate both topically and systemically. Topical sodium ascorbate 10% is given 2-hourly and the systemic dose is 2 g 4 times daily.
3. **Citric acid** is a powerful inhibitor of neutrophil activity. Topical sodium citrate 10% is given 2-hourly for about 10 days. Its aim is to eliminate the second wave of phagocytes, which normally occurs at 7 days after the injury.
4. **Tetracycline** derivatives are effective collagenase inhibitors. They also inhibit neutrophil activity and reduce sterile ulceration. They are administered both topically and systemically (doxycycline 100 mg twice daily).

SURGICAL TREATMENT

1. **Early surgery** may be necessary to revascularize the limbus, restore the limbal cell population and re-establish the fornices. One or more of the following procedures may be used:

Figure 16.48 Conjunctival adhesions following a chemical injury

(a) Advancement of Tenon capsule and suturing to the limbus is aimed at re-establishing limbal vascularity thus preventing the development of sterile corneal ulceration.
(b) Limbal stem cell transplantation either from the patient's other eye or from a donor is aimed at restoring a normal corneal epithelium.
(c) Amniotic membrane grafting may also assist in epithelial healing.

2. **Late surgery** may involve the following procedures:
(a) Division of conjunctival bands (Figure 16.48) and symblepharon (Figure 16.49).

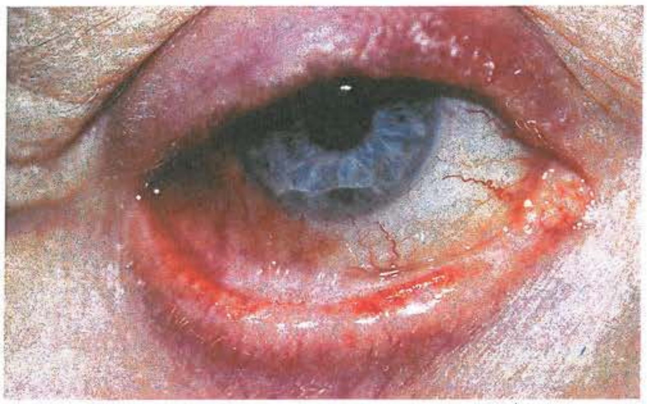

Figure 16.49 Symblepharon following a chemical injury

(b) Conjunctival or mucous membrane grafts.
(c) Correction of eyelid deformities.
(d) Keratoplasty should be delayed for at least 6 months and preferably longer to allow maximal resolution of inflammation.
(e) Keratoprostheses may (Figure 16.50) be required in very severely damaged cases because the results of conventional grafting are poor.

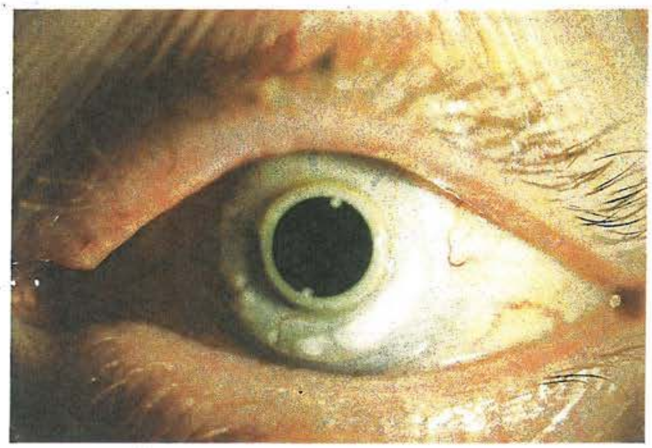

Figure 16.50 Keratoprosthesis in a grossly scarred cornea

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