The Cheek and Orbit: Part III

17

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17.1 Injuries to the Cheek and Orbit

17.1.1 Cheek Fractures

The zygoma is one of the most important bones of the midface. It plays a key role in the protection of the globe. In addition to providing support to the lateral aspect of the midface, it contributes to the profile to the face. However, due to this prominence, it is more vulnerable to injuries. Fractures are common and comprise a spectrum of severity, from relatively simple 'cracks' in the bone, resulting in minimal cosmetic problems, to complex injuries with gross disfigurement and considerable functional disability. Common causes of zygomatico-maxillary complex fractures in the walking wounded are assault, falls and sports injury. Zygomatic fractures in the young are more related to sports or assaults whist in the elderly they are more related to falls (Figs. 17.1 and 17.2).

Fractures may occur in isolation, or in combination with other injuries. The terminology can be a little confusing as these fractures often go by a variety of other names (Cheek, Zygoma, Malar, Zygomaticomaxillary, Tripod—to name a few). The typical fracture pattern is that of a tetrapod, although these are sometimes referred to as 'tripod' fractures, an erroneous term. There are in fact four "feet" or "pods". These are described in relation to the four main sites of fracture displacement that can be identified clinically or radiographically—i) frontozygomatic (FZ) suture, ii) maxillary buttress, iii) infra-orbital rim and v) zygomatic arch. In reality these fracture sites (except the zygomatic arch) are just part of a more complex, irregular fracture pattern, essentially an oblique ring-like fracture. This allows separation of the entire cheek from the rest of the facial skeleton. Displacement can then

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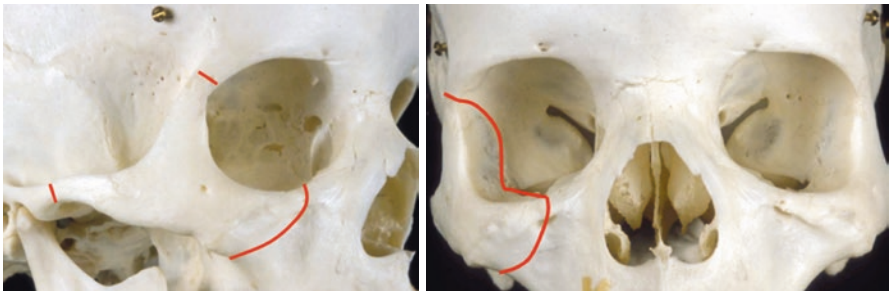

Fig. 17.1 The zygomaticomaxillary complex forms the prominence of the cheek

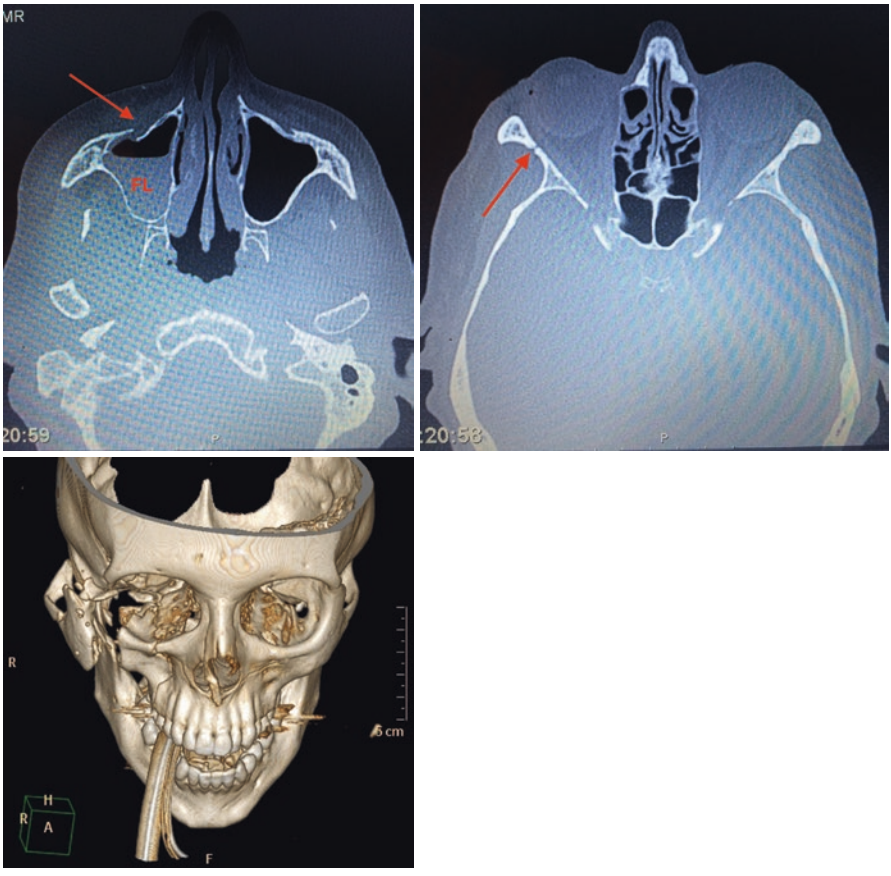

Fig. 17.2 Fractures can vary from minor cracks to collapse of the entire cheek

occur horizontally, vertically and anterio-posteriorly. Although commonly seen as "en bloc" type fractures, as the energy transfer increases from moderate to severe, fracture complexity increases correspondingly, with increasing comminution. Management of these fractures can therefore vary widely. Various classification systems have been described.

- Knight and North classified zygomatic fractures by the direction of displacement on radiography.
- Manson and colleagues proposed a classification based on the pattern of segmentation and displacement—'low-energy' fractures (very minimally displaced), 'medium-energy' (moderate displacement) and 'high-energy' (with comminution and segmentation).
- Gruss classification recognises the importance of specific sites (such as the zygomatic arch).
- Zingg classification related to fracture site and displacement
- Dingman classification related to direction of displacement and stability.
- Rowe and Killey's classification indicates stable and unstable fractures.

Generally speaking as the amount of displacement and comminution increases, the fractures are increasingly unstable and therefore likely to need surgical repair. From a practical viewpoint fractures can be considered as:

- Isolated fractures of the zygomatic arch
- Isolated fractures of the infra-orbital rim (uncommon)
- Minimally displaced "en bloc" fractures
- Significantly displaced "en bloc" fractures
- Comminuted fractures
- Fractures with associated midface or complex orbital floor/wall injuries (Figs. 17.3 and 17.4).

The mechanism of injury is helpful in diagnosis and planning. Direct blows can result in an isolated zygomatic arch or infero-medially displaced fractures. Frontal

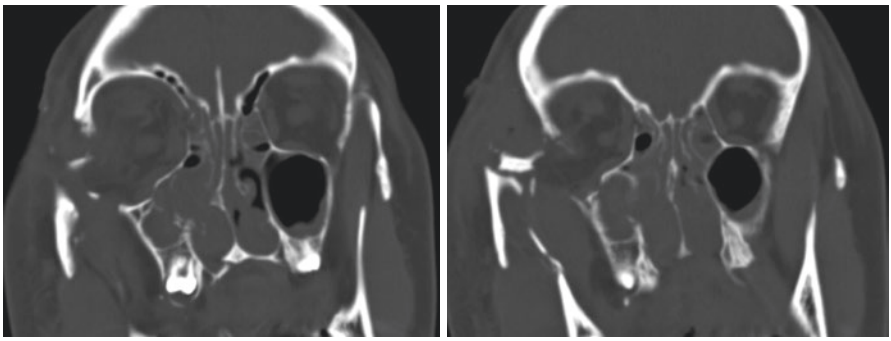

Fig. 17.3 Extensive fracture with orbital involvement

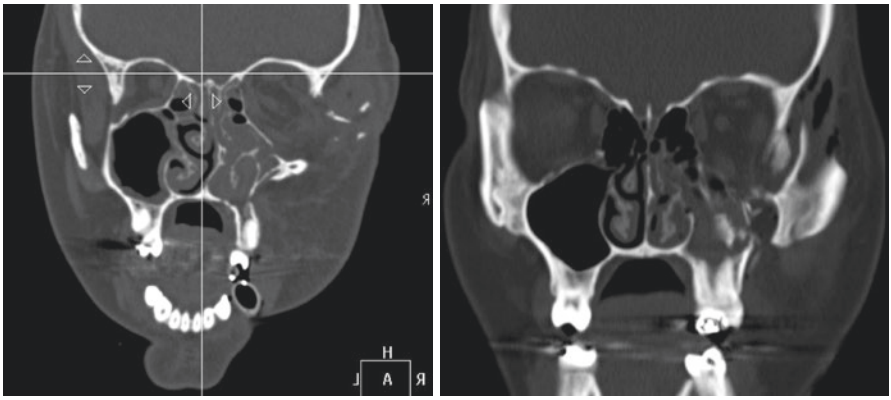

Fig. 17.4 Extensive orbital disruption in association

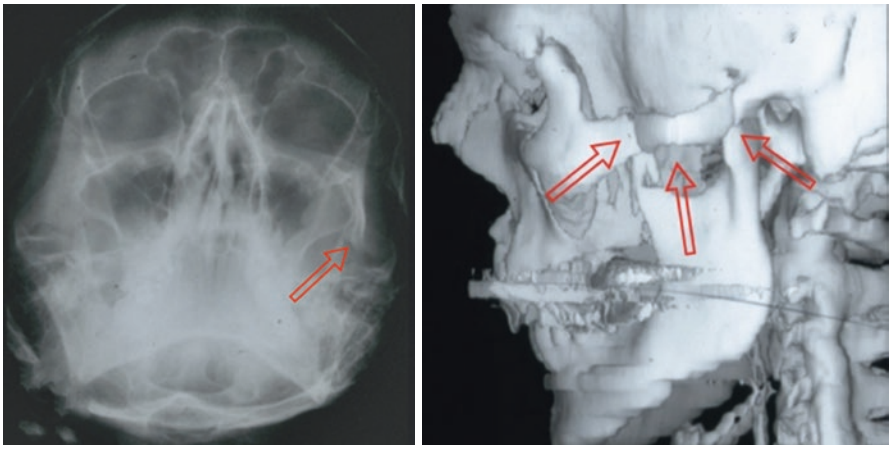

Fig. 17.5 Isolated arch fractures

impacts can displace fractures posteriorly and inferiorly. Irrespective of displacement all zygomatic fractures, by definition, extend into the orbit. This can replace the orbital contents at risk of entrapment, swelling, bleeding or injury. Patients should therefore be carefully assessed for ocular injury and diplopia. The eye always takes priority. Common ocular problems include i) Globe/muscle injuries, ii) Severe swelling or bleeding behind the eye, resulting in orbital compartment syndrome, or retrobulbar haemorrhage, iii) Superior orbital fissure syndrome and iv) Orbital apex syndrome. In some cases, the zygoma itself can be displaced into the orbit, increasing orbital pressures (Fig. 17.5).

Clinical features can vary depending on the force applied and the direction of displacement of the fracture. It is important to remember that a well-defined 'black eye' or a subconjunctival haematoma with no posterior limit, are reliable signs of

a fracture involving the orbit. Other eye signs include, diplopia, dystopia, enophthalmos, disruption of pupillary response and visual disturbances. Globe injuries are discussed in the chapter on the eye, but should always be considered and excluded. The cheek is often tender, swollen and bruised. Surgical emphysema may sometimes be palpable. Flattening of the malar prominence however may be masked by swelling if the patient presents soon after injury. Careful palpation should include the zygomatico-frontal suture, the orbital rim, zygomatic arch and the zygomatic buttress intra-orally, for tenderness and bony steps. Intraoral examination should also include assessment of the occlusion—in some displacements, the hard palate can flex and deform resulting in premature biting on the side of the injury. With isolated arch fractures, a depression is often palpable just in front of the tragus. There may also be pain with decreased mouth opening (Fig. 17.6).

Subtle signs which may be noted include a palpable infraorbital step, slanting of the palpebral fissure, loss of lower eyelid support and vertical ocular dystopia). Altered sensation in the cheek and upper lip, limitation of mouth opening and change in bite should also be documented. This can occur in up to 80% of patients and has medicolegal implications if surgical repair is being considered.

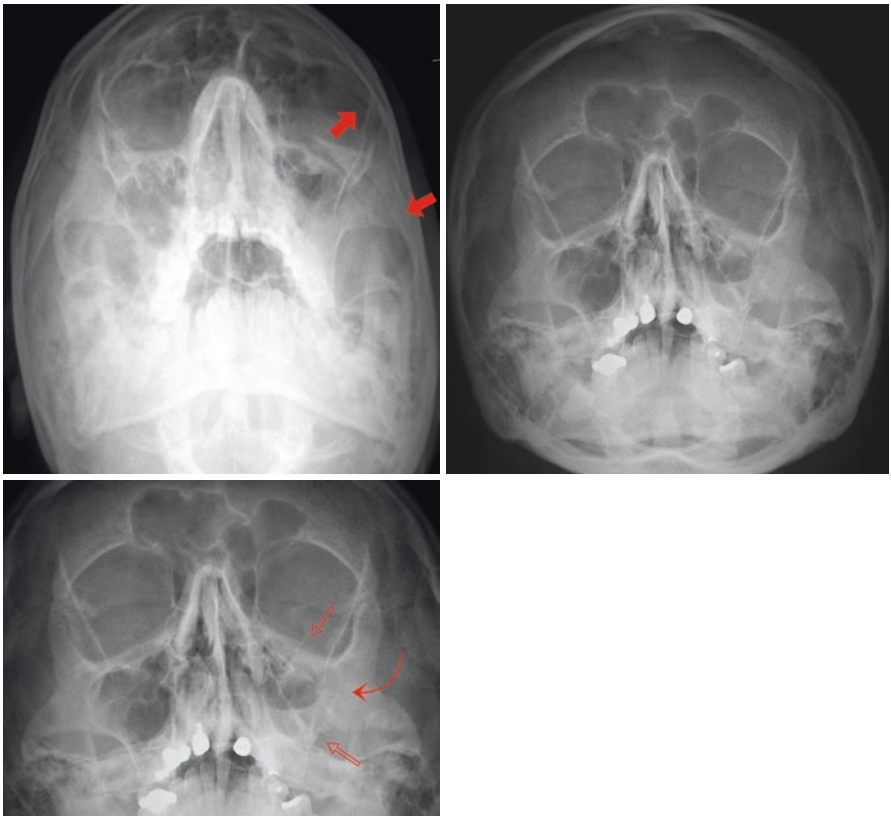

Fig. 17.6 Simples fractures can be evaluated using plain X-ray views

Depending on local protocols and clinical findings, patients with fractures of the zygoma may need further assessment from ophthalmology and orthoptics. This may be indicated to further assess the globe and ensure there are no serious injuries, and to assess the position of the globe and measure ocular movements. This is particularly important if the patient is complaining of double vision, or if surgery to the orbit is anticipated. Further investigations involve imaging. In most cases, plain radiographs will provide sufficient information to diagnose and plan treatment. Waters' view demonstrates the zygomatico-maxillary buttress and the inferior orbital rim. Caldwell's posterior-anterior view shows the zygomatico-frontal suture, the inferior orbital rim and the zygomatico-maxillary buttress. Submentovertex views will show fractures of the zygomatic arch. With more complex fractures, or if there is suspicion that the orbit has been significantly damaged (i.e. there is hypoglobus, enophthalmos, or obvious restriction of eye movements), a CT will probably be required. Axial, coronal and sagittal views with 3D reconstructions can now define these fractures in excellent detail. Ultrasound and maxillary sinus endoscopy (looking for orbital floor fractures) have also been reported as useful techniques, but these usually have no role in the emergency department setting. They are rarely undertaken routinely (Fig. 17.7).

Interpreting occipitomental views

To the inexperienced, interpreting occipitomental (OM) images can be difficult and sometimes confusing. This is due to a combination of complex anatomy, superimposition of the skull (notably the vascular markings and sutures), and the relatively oddly angled views compared with images taken elsewhere in the body. The best way to learn is to see plenty of examples. A number of useful approaches and signs have been described to help in interpretation (McGrigor-Campbell lines, Dolan Lines, "Black-Eyebrow" sign). Knowledge of the geometry of the "tetrapod" fracture configuration enables one to inspect the key areas (or "pods") on an OM view. These are the sites where displacement is most noticeable. Alternatively the "baby elephant" interpretation involves checking these sites and looking for a broken "trunk" (Figs. 17.8, 17.9, 17.10 and 17.11).

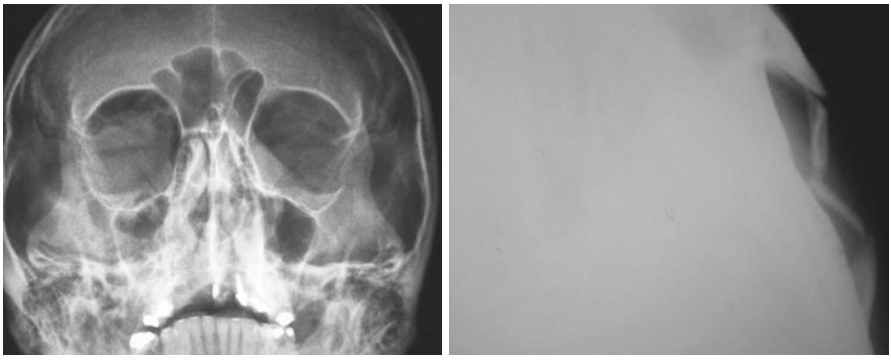

Fig. 17.7 Plain films can be difficult to interpret without clinical information

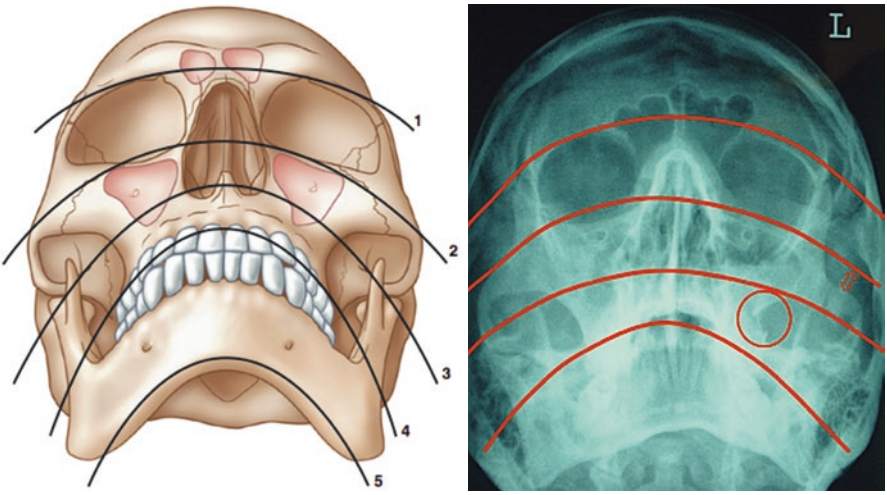

Fig. 17.8 Campbell's lines

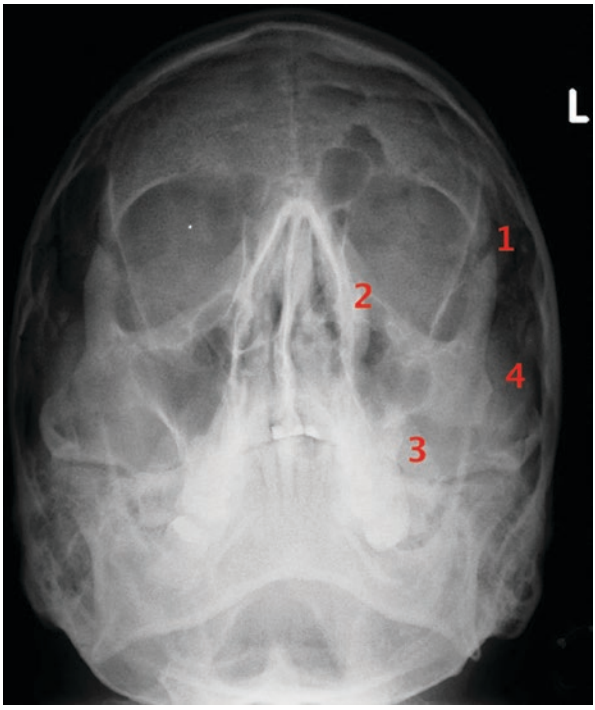

Fig. 17.9 Common sites of displacement visible on X-ray

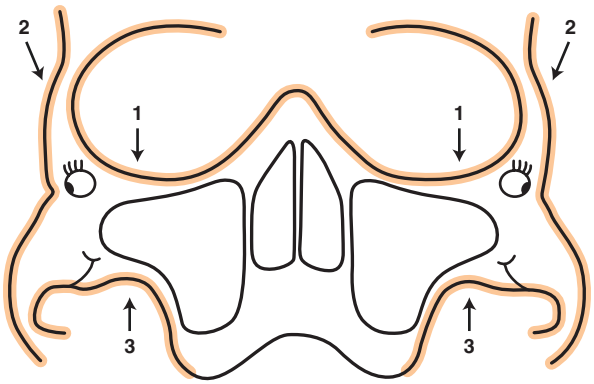

Fig. 17.10 "Baby elephant" interpretation

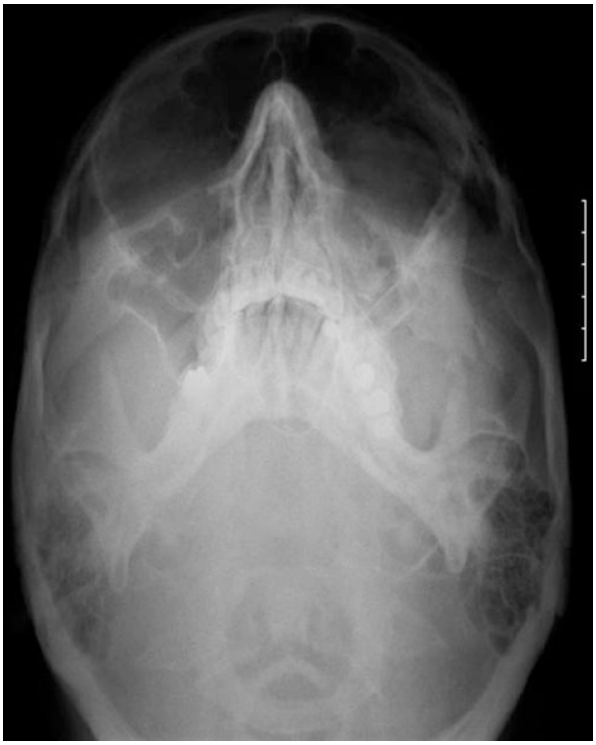

Fig. 17.11 Can you see the fractures?

Management of zygomatic fractures depends on the age of the patient, degree of displacement and the fracture and any functional deficit. Treatment varies from observation to open reduction and internal fixation. Stable, minimally displaced fractures with no clinical problems can be followed up in outpatients. Surgical

repair, if indicated is generally not urgently required. Over the years, with the development of better implants, there has been an increasing trend towards internal fixation.

In the first instance it is important to advise the patient not to blow their nose for 3 weeks. If they do, pressurised air can pass through the nose and antrum into the orbit via the fracture. This could potentially introduce bacteria resulting in orbital cellulitis. For the same reason, they should also be advised that if they have to sneeze, they should do so with their mouth open. This prevents a rapid buildup of pressure within the sinuses. Some specialists advise prophylactic antibiotics, but this is controversial. Patients should be told to return immediately if they have increasing swelling, pain or change in visual acuity (Fig. 17.12).

Referral to ophthalmology will depend on local policies and any clinical concerns. Not all fractured zygomas need to be referred routinely. However, never allow a patient with a suspected fracture to go home if you have been unable to assess the eye. Inability to open the eyelids is not an acceptable reason. If you cannot assess the eye, discuss this with ophthalmology.

Timing of surgical repair depends on the degree of swelling and other general medical or surgical considerations, notably any coexisting head, cervical or ocular injuries. Surgery is usually carried out either immediately or more commonly 7–10

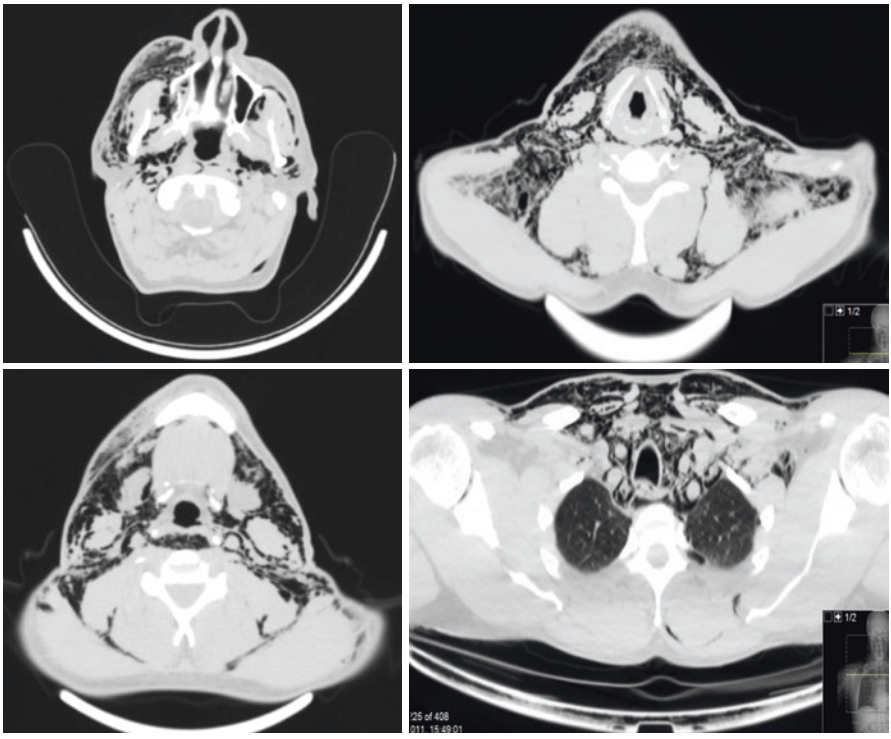

Fig. 17.12 Extensive surgical emphysema following repeated nose blowing

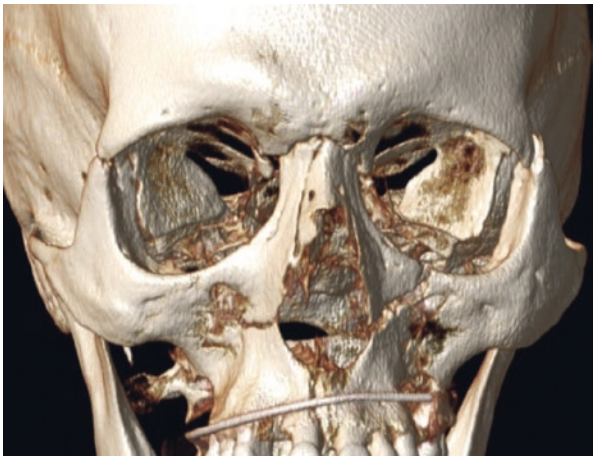

Fig. 17.13 3DCT showing significant displacement

days following injury. Acceptable results can still be obtained at 3–4 weeks, but this is far from ideal as reduction and repair of the fracture becomes more difficult. Many fractures are treated by open reduction and internal fixation (ORIF) with titanium miniplates. Surgical access for reduction and fixation is commonly through the mouth to avoid facial scars. Access to the frontozygomatic suture and infraorbital rim may also be necessary to assist reduction and fixation. The more displaced or comminuted the fracture the more fracture sites need to be exposed. Major complications are rare but include retrobulbar haemorrhage, blindness, traumatic optic neuropathy and superior orbital fissure syndrome (Fig. 17.13).

17.2 Orbital Fractures (Isolated)

Orbital fractures can affect any of the four orbital walls and the orbital margin. They commonly occur in isolation, but they can also be involved as part of a larger fracture, extending into the surrounding bones. These include fractures of the zygoma, nasoethmoid region and anterior cranial fossa). The term 'Blow-out' fracture is a specific term that refers to an isolated injury of one or more orbital walls (commonly the floor or medial wall). In these fractures the surrounding orbital rims and facial bones are intact. They are commonly seen following moderate energy, localised impacts to the prominence of the cheek, or directly on the globe. The classic mechanism described is the 'squash ball in the eye', although this is not commonly seen (Figs. 17.14 and 17.15).

The orbital floor and medial orbital wall are particularly delicate structures and are the two sites most commonly fractured in blow-out fractures. Two proposed mechanisms have been suggested—i) a direct blow to the globe ('squash ball'). This results in the transfer of energy throughout the orbital contents and directly onto the orbital floor and medial wall. This transfer of energy is referred to as the "hydraulic

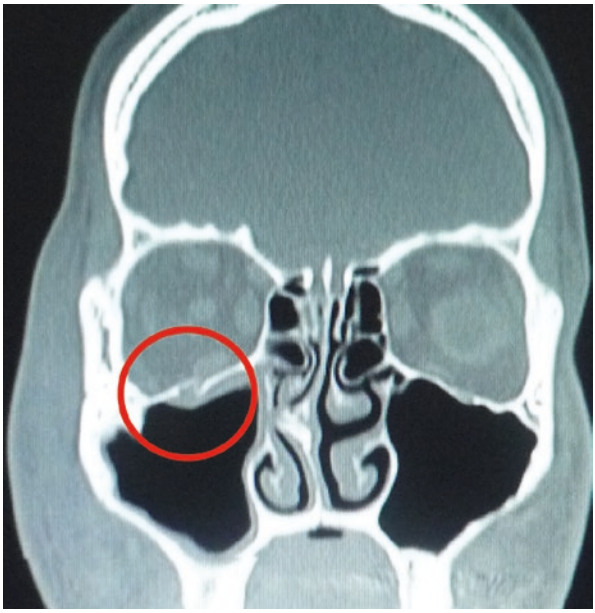

Fig. 17.14 Trap-door type fracture orbital floor

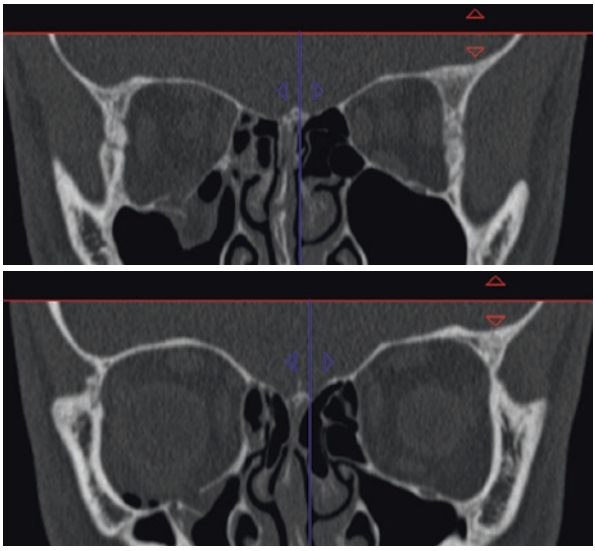

Fig. 17.15 Orbital floor fracture

theory". In these cases the globe is also seriously injured. Alternatively, ii) a localised blow to the prominence of the cheek can deform the bone such that it 'buckles', resulting in fracture propagation within the orbit, but not the cheek itself. Bone is a 'plastic' material and not completely rigid or brittle. It is more like wood than ceramic. Consequently, bone tends to 'bend' a little before it breaks. This is especially seen in children. It is also the reason why, despite our best efforts, it can be difficult to get a perfect anatomical reduction in comminuted fractures. This theory is referred to as the "buckling theory" and may be associated with other facial fractures.

Whatever the mechanism, the displaced (depressed) blow-out bony fragment allows the orbital tissues to sag downwards into the maxillary sinus, or medially into the ethmoid sinuses. Posterior (enophthalmos), inferior (hypoglobus) and sometimes medial displacement of the globe then occurs. If fat becomes trapped within the fracture, movement of the globe may become restricted. Less commonly, the inferior rectus muscle itself may become directly trapped in the fracture, leading to very painful restriction of globe movement. This requires urgent release (Figs. 17.16 and 17.17).

The orbital floor is not flat, but rather 'sinusoidal' in geometry. This is best seen on the sagittal view of an orbital CT. It has an anterior concavity immediately behind the inferior orbital rim, which dips into the maxillary sinus, followed by a convex "bulge" posteriorly. It also slants up (from front to back and from lateral to medial) and blends almost imperceptibly with the medial orbital wall. This complex geometry must be re-created when reconstructing the orbit. The medial wall of the orbit is formed by the thin orbital plate of the ethmoid bone, one of the thinnest bones in the body. Fractures involving the medial orbital rim may displace the bone to which the medial canthal tendon and lacrimal sac are attached. This may result in telecanthus and epiphora (discussed in the chapter on the nose) (Fig. 17.18).

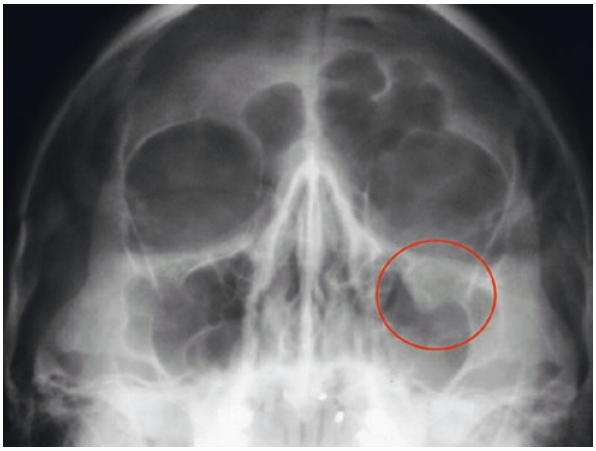

Fig. 17.16 Possible "tear drop" sign

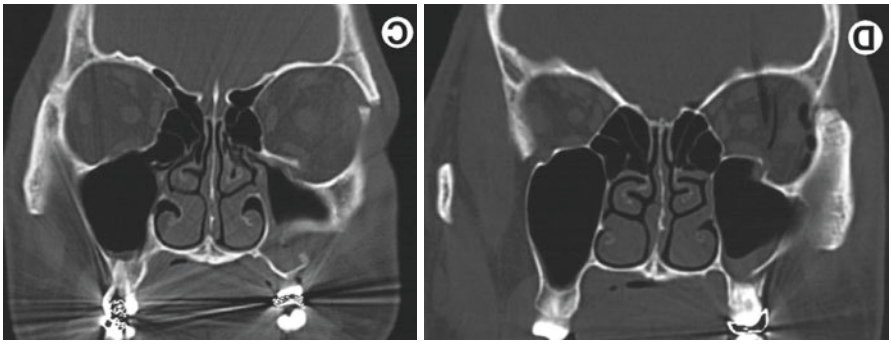

Fig. 17.17 All zygoma fractures involve the orbit to some extent

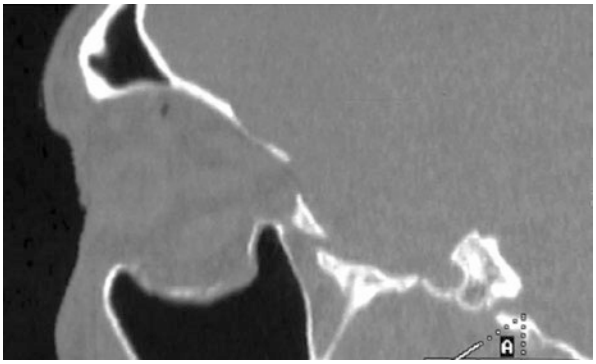

Fig. 17.18 Significant volume change in orbit as a result of loss of orbital floor support

Clinically, patients usually present with findings similar to that of a fractured zygoma. Distinguishing between the two may be difficult. In severe cases there may be significant proptosis. There may be a history of blunt trauma to the globe, cheek or orbit. Periorbital and subconjunctival haematoma are usually present and there may be numbness in the distribution of the infraorbital nerve. Patients may also complain of double vision (usually when looking upwards or downwards). Whilst this may generally be a little uncomfortable for the patient, severe pain should be investigated urgently. The globe should always be carefully examined, especially if it has been directly injured. Depending on the mechanism of injury, damage to the globe can vary from minor abrasions on the cornea, to devastating loss of vision from a globe rupture. About 10–15% of orbital fractures may have an associated globe injury. The possibility of an intraorbital foreign body should also be considered (Figs. 17.19, 17.20, 17.21, 17.22 and 17.23).

When diplopia is present, a "forced-duction" test may be used to confirm entrapment of the soft tissues in the fracture site. This is not often done in the emergency department, but is nevertheless a very useful diagnostic test. Topical local

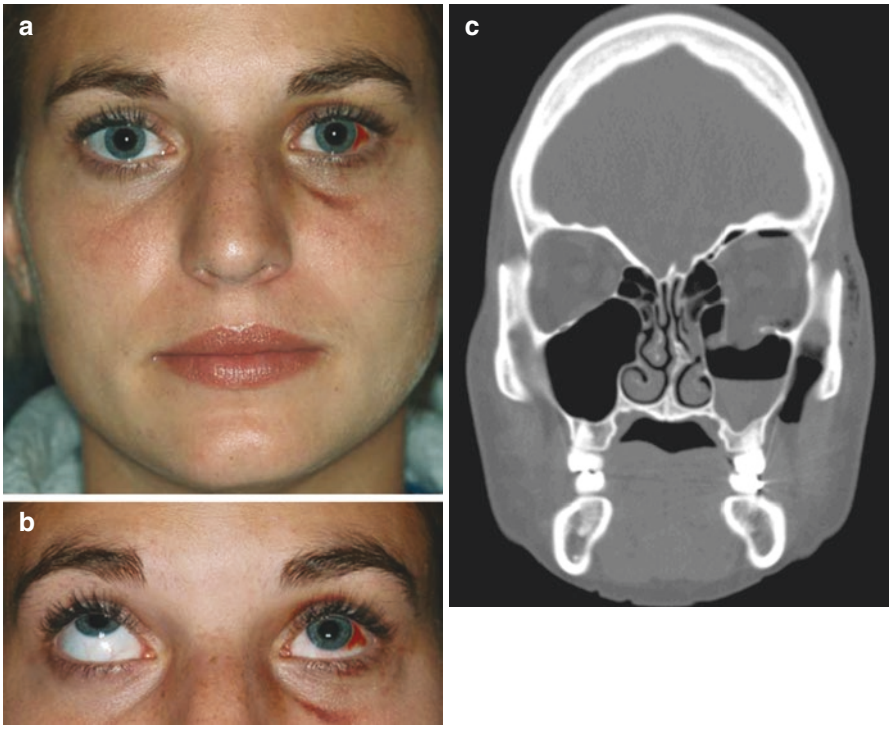

Fig. 17.19 (**a**) 24-year-old female, 1 week after periorbital trauma, resulting in an orbital blow-out fracture on the left side. The patient has 2 mm of enophthalmos. (**b**) Ocular motility shows restriction of upgaze of the left eye. Forced ductions demonstrated a restrictive motility disturbance. (**c**) Coronal CT scan shows an orbital floor fracture with orbital soft tissue prolapsed into the maxillary sinus

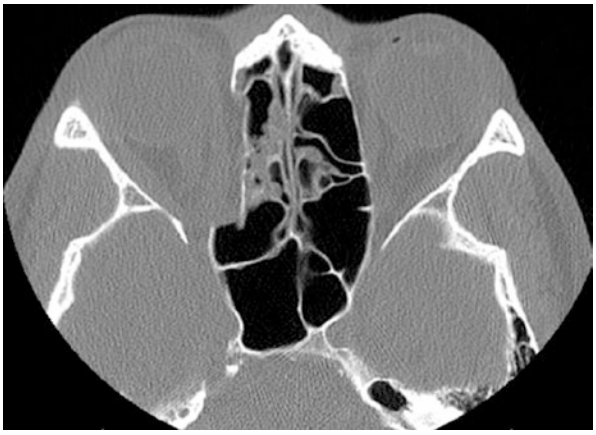

Fig. 17.20 Medial wall fracture

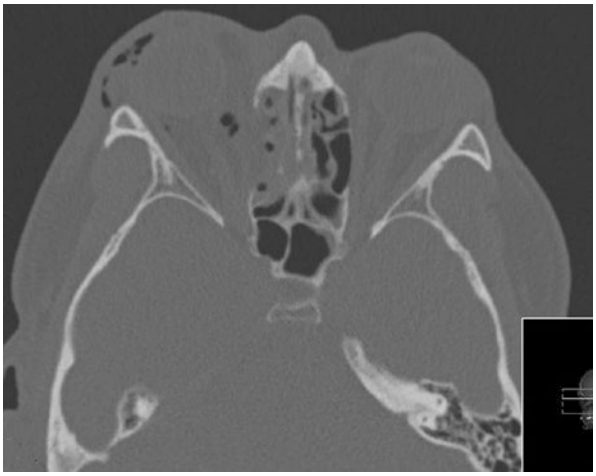

Fig. 17.21 Medial wall fracture

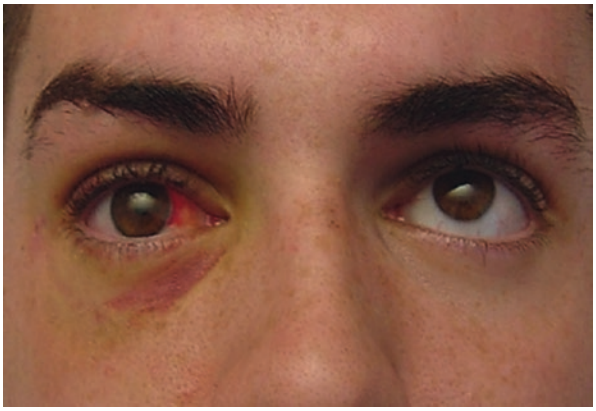

Fig. 17.22 Entrapment on looking up

anaesthesia is placed in the lower fornix of the eye. The inferior rectus muscle is then gently grasped at its point of insertion, using fine forceps and the eye is gently rotated up. If resistance it is felt, this is highly suggestive of fat or muscle entrapment. Entrapment occurs more commonly with small fractures. With large fractures, entrapment is less likely but the enlargement of the orbital volume results in globe dystopia and enophthalmos. This may be accompanied by supratarsal hollowing and ptosis of the upper eyelid. Enophthalmos is best assessed with Hertel exophthalmometry once any swelling has resolved (Figs. 17.24, 17.25 and 17.26).

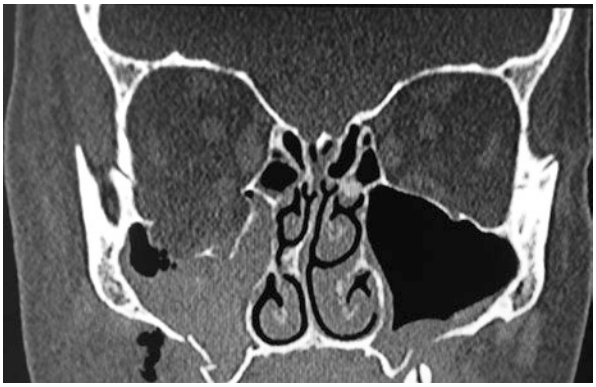

Fig. 17.23 Large floor defect

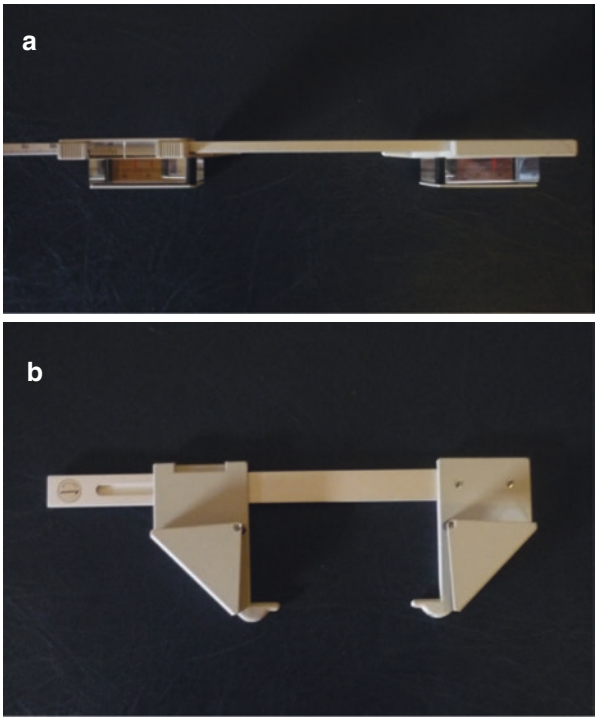

Fig. 17.24 Hertel exophthalmometer

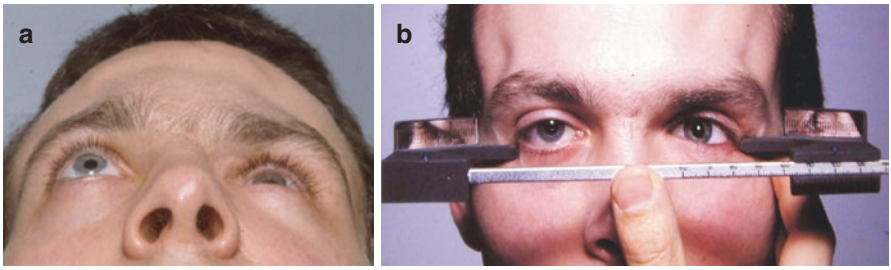

Fig. 17.25 Correct positioning is important. Measurement will be difficult if there is coexisting displacement of the zygoma

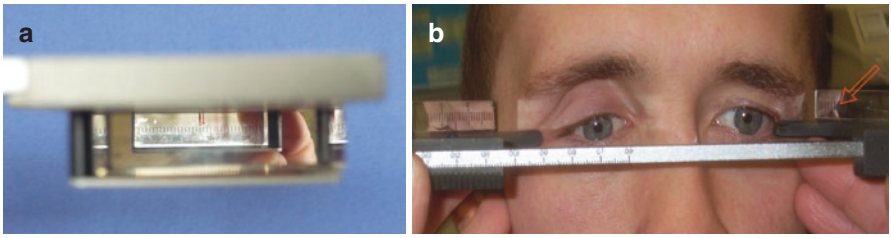

Fig. 17.26 The mirrors are used to take the measurements

17.2.1 WEBOF Fractures

These are a small but very important group of orbital fractures. The term WEBOF refers to 'white-eye blowout fracture', that is a blowout fracture without any obvious external signs of injury (the globe otherwise appears to be healthy). In children, fracture patterns and clinical presentation can differ from adults. In children the orbits are relatively shallow. There is also greater elasticity in the bones which can result in a "trapdoor" type mechanism. At the moment of impact the orbital floor fractures, momentarily gapes open and then closes again. During this brief moment the soft tissues can herniate through the gap, to then become entrapped when it closes. There is also a greater chance of muscle entrapment with ischaemic incarceration. Most 'trapdoor' cases tend to present as "pure" blowout fractures, without involvement of the zygoma or orbital rim. Consequently, there may be few clinical signs (such as subconjunctival haemorrhage and periorbital bruising), hence the name "WEBOF".

Patients often complain of severe (disproportionate) pain. They often display blepharospasm and have autonomic symptoms, such as nausea, vomiting, bradycardia and even syncope (as a result of increased vagal tone). Such symptoms are highly suggestive of WEBOF. Not surprisingly however, these same symptoms may initially be thought of as secondary to a head injury. The mechanism of injury is

therefore a useful clue in diagnosing this condition. Once WEBOF with muscle entrapment is confirmed, immediate surgical intervention is indicated. This is because if left untreated, ischaemic necrosis and subsequent scarring of the trapped soft tissues and muscles may occur (similar to that seen in Volksmann's ischaemic contracture of the extremities).

17.2.2 "Blow-In" Fractures

These comprise a small group of uncommon but potentially sight-threatening injuries. They often occur following a high energy blow to the orbital rims, or temple. All four walls can be affected, resulting in displacement into the orbit, although these usually occur in isolation. As these are high energy impacts there may also be an associated subperiosteal haematoma, adding to any 'mass effect'. These fracture are often associated with globe injury or ocular mobility problems. They can occasionally extend to the orbital apex and result in optic nerve injury. With orbital roof fractures there is a high incidence of intracranial problems, including CSF leaks into the orbit and CSF oculorrhoea. In addition to the signs of a fracture, these fractures can result in extensive orbital haematoma, proptosis, raised IOP, ptosis and optic nerve contusion.

Late complications include orbital mucocele as a result of entrapped sinus mucosa, cholesterol cysts from unresolved subperiosteal haematomas and encephalocoeles (Figs. 17.27 and 17.28).

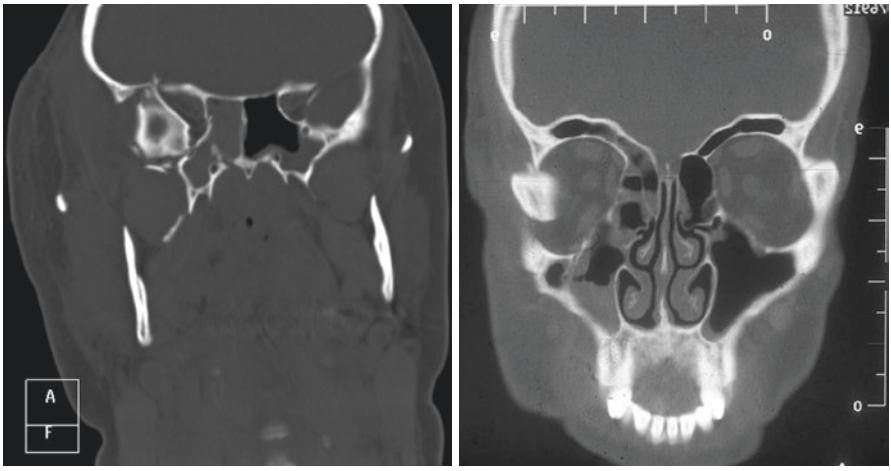

Fig. 17.27 Deep fractures of the orbit should be regarded as skull base fractures. They require a high amount of energy to occur

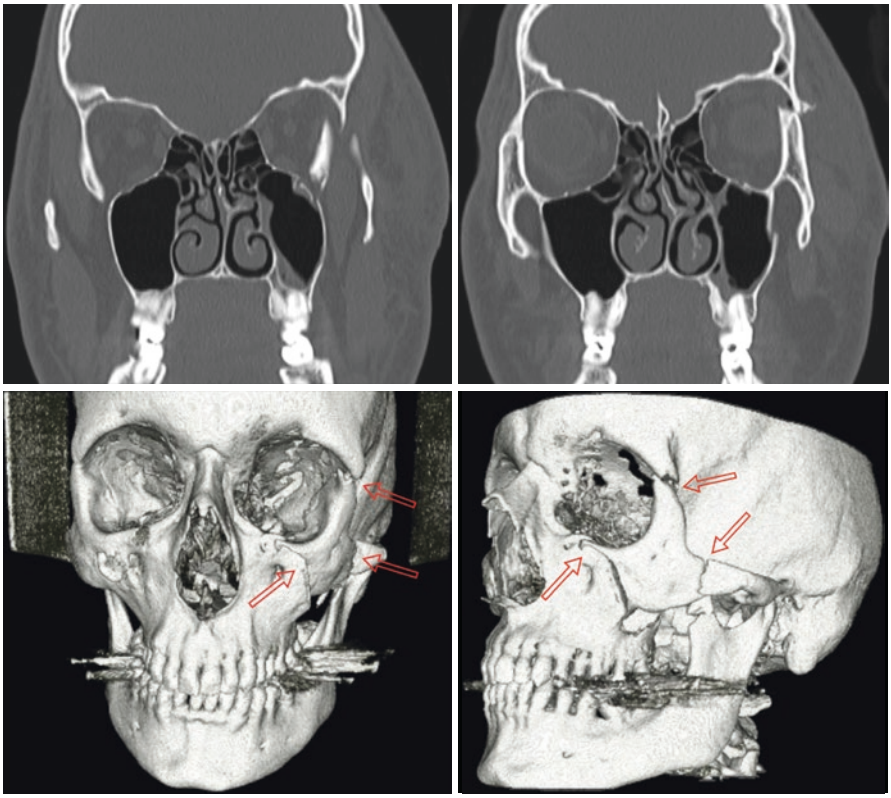

Fig. 17.28 Medial displacement of the lateral orbital wall following side impact

17.2.3 Orbital Haematomas and Haematic Cysts

Haematomas in the orbit usually occur following traumatic, but they can also present as complications of anticoagulation or coagulopathies. Occasionally, a ruptured vascular malformation can produce a haematoma. In the absence of significant trauma haemorrhage into a tumour should always be considered. Penetrating trauma can produce a haematoma anywhere within the orbit, but following blunt trauma, they tend to be subperiosteal. Persistent haematoma may develop into a haematic cyst, in which cholesterol clefts develop within the clot, which together with blood degradation products stimulate a granulomatous reaction. Ongoing inflammation together with small rebleeds can then result in a slowly enlarging mass, proptosis and/or diplopia months to years after the initial injury.

Initial investigations in orbital trauma often commences with occipitomental (OM) and lateral facial views (to assess for cheek or midface fractures). These may show a 'hanging drop' sign. This is a small opacity, seemingly suspended from the roof of the maxillary sinus (the orbital floor). This may represent the herniation of

orbital contents into the maxillary sinus. However it may not be easily discernible and not all 'hanging drops' are herniated contents. Nevertheless it is an important sign and merits further investigation. A fluid level in the maxillary sinus following trauma also suggests the possibility of a fracture somewhere, although this may not necessarily be the orbit. The definitive test is a CT scan of the orbits. Slices should be 1 or 2 mm thick. Conventional head CTs are therefore inadequate. Once the diagnosis is confirmed, patients usually require referral for an ophthalmic and orthoptic assessment—Hess chart, measurement of globe projection and fields of binocular vision (to assess restriction of ocular movement). However swelling may preclude immediate assessment (Figs. 17.29 and 17.30).

Initial management of orbital fractures is similar to fractures of the zygoma. The eye takes priority. Where an injury to the globe or associated nerves is suspected an ophthalmic opinion should always be sought. In all cases it is important to advise the patient not to blow their nose for 3 weeks and sneeze with their mouth open. They should be told to return if they develop any visual problems or increasing pain. Depending on local protocols and clinical findings, antibiotics may be prescribed. Never allow a patient with a suspected blowout fracture to go home if you have been unable to assess the eye. Surgical repair of a blowout fracture is not necessary in every case and is not urgent, except in children with WEBOF, where this should be regarded as a surgical emergency. In many cases, minor symptoms of diplopia associated with small fractures resolve within a short period of time, without the need for surgical repair. In such cases the double vision is secondary to swelling and bruising of the muscles. Surgery is usually indicated if significant diplopia fails to resolve, is involving day-to-day activities, or has been shown (on CT or a forced duction test) to be as a result of entrapment. Common indications for surgery for blow-out fractures are

- Significant diplopia on upward gaze (ideally with a retraction sign)
- Significant diplopia on downward gaze—this can interfere more with day-to-day activities, such as reading, walking, using stairs etc.
- Entrapment confirmed on CT scan and a forced-duction test
- Significant enophthalmos
- Enlargement of the orbital volume sufficient enough to result in future enophthalmos. This latter indication is more controversial in terms of fracture size.

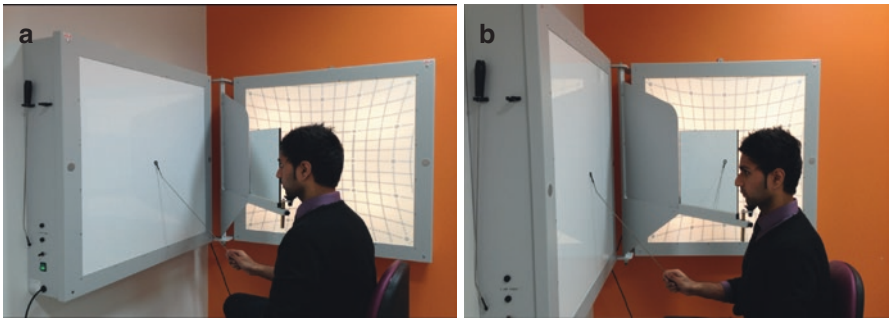

Fig. 17.29 Example of a patient performing a Hess charting with the Lees screen

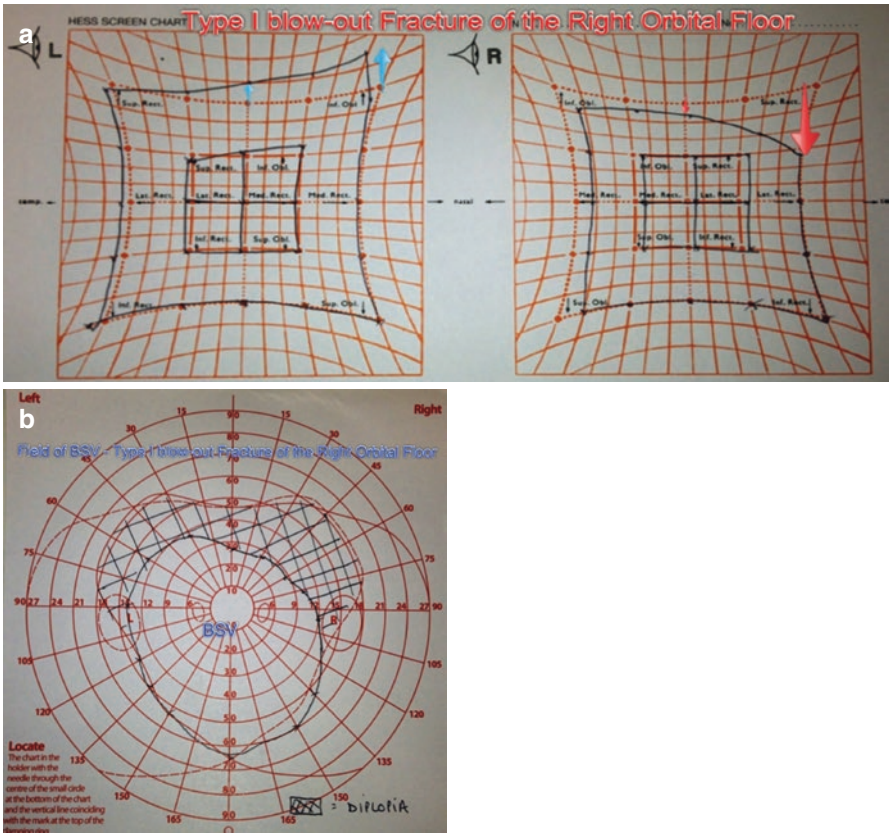

Fig. 17.30 (**a**) Looking at the Hess chart of the right field, you can see the limited elevation mainly affecting abducted elevation (red arrows). This also demonstrates Herring's law by the overaction of the contralateral synergist (blue arrows). Forced duction test is positive with the greatest limitation in abducted elevation. (**b**) Patients often adapt well to a reduced field of BSV as the defect is in the superior field, which is not as commonly used as the primary position and down gaze (e.g., reading)

The aim of repair is to release any herniated or trapped tissues and restore orbital geometry. This should release any restrictions to eye movement and restore globe position. Timing of surgery is controversial and dependent on multiple factors. If the tissues are very swollen or there are minimal signs it is common practice to delay surgery for up to 10–14 days post-injury. This allows swelling to settle and gives a better idea of any continuing disability.

17.2.4 Orbital Roof Fractures

Orbital roof fractures are commonly the result of high impact trauma (such as motor vehicle collisions, falls and assaults). They are found in approximately 10% of all

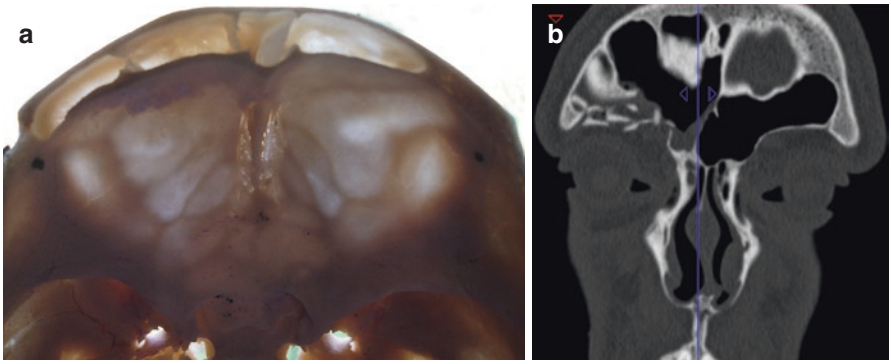

Fig. 17.31 The anterior cranial fossa is "*wafer-thin*" and can be fractured following relatively minor impacts

upper facial fractures. These fractures rarely present in isolation and are more often associated with craniofacial fractures and concomitant injuries to the eye, extra ocular muscles, dura and brain. The orbital roof slopes backward and downward towards the orbital apex. It is composed of the orbital plate of the frontal bone and greater and lesser wings of the sphenoid. These separate the anterior cranial fossa from the orbital contents. However, the bones here are very thin. In fact they are almost transparent, especially medially near the frontal sinus. This can be verified simply by holding a real skull up to sunlight. Medially is the attachment of the superior oblique tendon. Diplopia secondary to disruption of the superior oblique muscle's function (Brown's syndrome) can be very difficult to treat (Fig. 17.31).

Orbital roof fractures are difficult to manage because of the risks to the eye, extra ocular muscles, frontal sinus, dura and brain. Fractures may be classified as

i) Undisplaced or minimally displaced. These generally require no specific treatment.
ii) "Blow-in" (inferior displacement of the orbital roof). This is due to an abnormal increase in intracranial pressure. Fracture fragments are seen within the orbital cavity.
iii) "Blow-out" (superior displacement of the roof into the anterior cranial fossa). This is due to increased intraorbital pressure.
iv) Supraorbital rim fracture. These are indicative of a significant impact. The frontal bone is usually thick and strong.
v) Frontal sinus fractures involving the orbital roof. These may require complex management of both the frontal sinus and the orbit.

Important structures that lie within, or in close proximity to the orbital roof are the lacrimal gland, superior oblique, levator palpebrae superioris and superior rectus muscles. The frontal nerve, the superior division of the oculomotor nerve and the eye are also nearby. In some cases the loss of separation between the orbit and

anterior cranial fossa may result in 'pulsatile exophthalmos'. This occurs when cerebral pulsations are transmitted to the globe. It can result in painful diplopia and autonomic symptoms. Fractures which extend beyond the orbital roof to involve the middle cranial fossa can also result in carotico-cavernous sinus fistula. The patient quickly develops chemosis, proptosis, visual impairment and cranial nerve palsies. This can become a vision threatening problem and requires urgent diagnosis and treatment. The fistula is confirmed by CT angiography or arteriography.

Patients may thus present with varying symptoms. In addition to periorbital oedema and bruising, the patient may complain of diplopia. This can arise if there is involvement of the superior oblique or rectus muscles, with limitation of vertical or inward gaze. Forehead numbness is an useful clue. Depending on the amount of fracture displacement, exophthalmos, enophthalmos, hypoglobus, or proptosis may occur. Surgical emphysema suggests involvement of the frontal sinus. If there is an overlying laceration, this needs to be assessed carefully. Fractures in this area can be associated with dural tears and the leakage of CSF. This can sometimes be seen leaking from the wound. These fractures tend to follow high energy localised impacts to the forehead. Diagnosis is confirmed by computed tomography (CT). It is important to also consider associated injuries and complications which may have implications on treatment. These include i) fractures of the frontal sinus, orbital rim, and NOE region, ii) intracranial injuries, such as cerebrospinal fluid leaks (CSF rhinorrhoea), dural tears, tension pneumocephalus, encephalocoele, frontal lobe contusions, intracranial haemorrhage, iii) orbital injuries involving the eye, optic and other nerves, (especially those passing through the superior orbital fissure), extra ocular muscles and vessels.

In the first instance, these injuries should be regarded as a head injuries (skull fractures) and managed as such. Surgical repair of orbital roof fractures is rarely indicated urgently, unless associated with open, contaminated fractures. In some cases surgery is not required at all. Surgical repair of an orbital roof fracture may be considered if the patient has symptoms, or is at risk of significant complications. Bone fragments displaced intracranially can result in structural damage to the brain, risks of infection and encephalocoele. Large defects in the orbital roof can contribute to diplopia and result in pulsatile exophthalmos. However this does not occur in every case and the decision to repair these injuries is made on a case by case basis. Undisplaced fractures are usually managed conservatively, with head injury and eye observations for the first 24 h. Significantly displaced fractures associated with symptomatic involvement of the eye, extra ocular muscles and nerves, proptosis or CSF leaks may require surgical intervention. If the supraorbital rim is significantly displaced, the patient may also be offered surgery. This is usually undertaken when swelling has settled.

17.2.5 Lateral Orbital Wall Fractures

Isolated lateral orbital wall fractures are rare, as this is the strongest of the four orbital walls. Lateral wall fractures are therefore more commonly seen following

significant maxillofacial trauma involving the malar complex. These are assessed and managed as with all other orbital fractures. “Blow-in” fractures are unusual, but because of the orientation of the lateral orbital wall, can result in significant proptosis. They can also be associated with superior orbital fissure syndrome. If fractures are seen to extend to the superior orbital fissure, this is essentially a middle cranial fossa fracture and should be managed as such.

17.2.6 Penetrating (Transorbital) Roof Injuries

Penetrating orbital-brain injuries, although uncommon, can result in significant brain damage, disability and even death if not promptly treated. Although these injuries may seem trivial at first glance, they can extend deep, through the orbit and dura, to pass intracranially. In some cases, patients may initially present with relatively minimal symptoms. These injuries can therefore be easily overlooked. However they are often associated with serious complications, including brain abscess, meningitis, cerebrospinal fluid leakage, haemorrhage and neurological deficit. Cerebral infection is the most common cause of mortality.

Foreign bodies usually penetrate the orbit via the medial canthus. These can continue through the optic canal, superior orbital fissure or through the fragile orbital roof to enter the cranial cavity. In the latter instance this often results in frontal lobe contusion or laceration. Intracranial passage via the superior orbital fissure can result in injury to the brain stem or the cavernous sinus. Cerebrospinal fluid fistulas, pneumocephalus, orbital cellulitis, carotid-cavernous sinus fistula, central nervous system (CNS) infections, traumatic aneurysm, progressive intravascular thrombosis and intracranial haemorrhage can therefore all occur. Occult penetrating injuries are frequently overlooked as there is often just a small laceration without any other clinical signs. This results in delayed diagnosis. The mechanism of injury is therefore important to maintain a high index of suspicion. In other patients, the penetrating object passes through the globe and then intracranially. These are more obvious injuries, in that the patient will present with obvious and often catastrophic globe injury. Persistent leakage of clear or blood stained fluid should immediately raise suspicion for intracranial injury (CSF). Penetrating eye injuries are discussed in a chapter on the eye.

Initial management of these injuries is to leave any obvious penetrating object in situ until adequate imaging and preoperative planning have been undertaken. The object should only be removed under direct visualisation in the operating theatre. This may require craniotomy. The precise course of the object should therefore be determined, so that the structures that may have been penetrated or lacerated can be anticipated and appropriate surgical access planned. Premature removal without adequate preparation can risk further injury or fatal haemorrhage if the object is tamponading bleeding.

Immediate CT is usually indicated although MRI may be preferable if the object is wooden. CT provides good quality three-dimensional visualisation of the foreign body’s trajectory, any associated fractures, brain swelling and parenchymal

haematoma. Unfortunately, scanning artefacts from metal objects often limit visualisation of the brain. Angiography may also be required. Deeply sited bullets in the brain, may sometimes be left because of the risk for further damage during an attempted retrieval and a relatively low incidence of infection. However, metal bars and other missiles should be removed and prophylactic antibiotics given. Tetanus toxoid should also be considered. Commonly reported pathogens in abscesses and meningitis are Streptococcus and Staphylococcus. Despite the risk of major complications, many of these injuries do have the potential for reasonable recovery. There are numerous documented cases of patients returning to full function with normal vision following timely management.

17.3 Retrobulbar Haemorrhage and Orbital Compartment Syndrome (OCS)

Retrobulbar haemorrhage (RBH—bleeding behind the globe) is an uncommon but potentially blinding orbital emergency which needs immediate diagnosis and treatment. It usually occurs following trauma but is reported to be the most common risk of peri- and retrobulbar anaesthesia and the main cause of loss of vision after blepharoplasty. Drug therapy (anticoagulation), hemorrhagic diseases, vessel malformations and spontaneously bleeding are all examples of non-trauma related cases. In most cases, diagnosis is relatively simple. In essence this is an acute compartment syndrome within the orbit with the symptoms one would expect to see in any compartment syndrome (notably pain). Since the orbit lacks any significant lymphatic drainage the only drainage possible is via the delicate ophthalmic veins. Raised intra-orbital pressure can quickly occlude these vessels. As little as 7 mL of additional fluid behind the eye can produce ischaemic changes. This increased volume is contained within the bony orbit, behind the relatively unyielding orbital septum. Rarely tumours and other slow growing lesions can reach such a size, that OCS develops (Fig. 17.32).

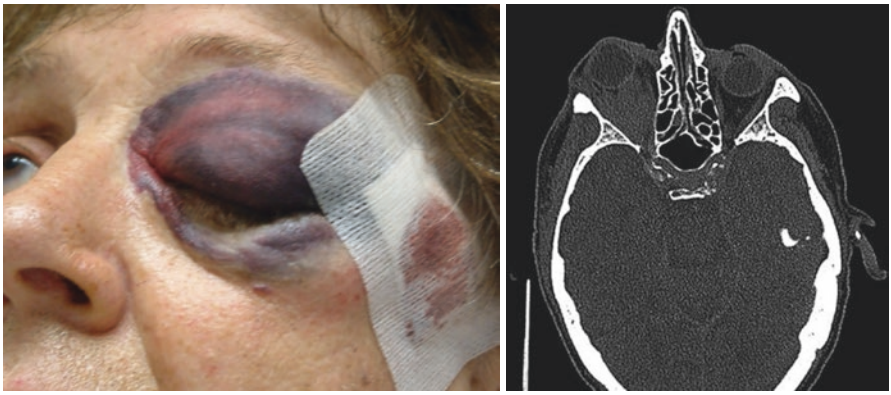

Fig. 17.32 Retrobulbar haemorrhage—one type of orbital compartment syndrome

Causes of orbital compartment syndrome include both traumatic and non traumatic conditions. The most common causes are trauma, surgical procedures and local anaesthetic injections in the periorbital region. RBH has been reported to be a common risk in peri- and retrobulbar anaesthesia and is reported to be one of the main causes of loss of vision following blepharoplasty. Orbital surgery and other procedures performed nearby (to the sinuses, nose etc) can also result in intraorbital haemorrhage. Drug therapy and haemorrhage secondary to congenital or systemic conditions are rare causes of non-traumatic RBH. Very rare causes include orbital vascular malformations (such as orbital varix, lymphangioma, or arteriovenous malformation). Bleeding can occur within or outside the muscle 'cone', intra-conal bleeding being more significant. As the pressure rises, it compresses the ophthalmic and retinal vessels, resulting in retinal and optic ischaemia. However, in many cases of acute proptosis there is no bleeding, but rather extensive oedema. When severe enough this presents in exactly the same way, hence the preferred termed of Orbital Compartment Syndrome. Ischaemia of the optic nerve is the main concern. Any rapidly expanding orbital lesion can results in elevated pressure in the orbital compartment. This then occludes the central retinal artery, with mechanical compression of both the optical nerve and its blood supply. Failure to recognise this phenomenon can quickly result in blindness. Irreversible ischaemic damage to the optic nerve and retina has been estimated to occur within 90 min of ischaemia.

The key to diagnosis is awareness. Proptosis is a common complication following trauma and orbital procedures, but is usually minor and resolves without treatment. However, vision-threatening retrobulbar haemorrhage or orbital compartment syndrome should be considered if the patient develops i) severe pain, ii) worsening proptosis, iii) visual loss or a non-reacting pupil or iv) severely decreased eye movements in all directions (ophthalmoplegia). Intense blepharospasm with inability to open the eyelids and inappropriate pain is highly suspicious for RBH/OCS. Pain is often disproportionately severe for the circumstances and this is an important clue. Just like compartment syndromes elsewhere (ie the limbs), the pain is secondary to severe ischaemia of the orbital contents, notably the muscles. Diagnosis is usually clinical, but when IOP measurements are available, a value of 40 mm Hg is considered the threshold above which intervention is indicated. Intraocular pressure can be assessed with a Tono pen®. However not all emergency departments, clinical departments and wards will have this specialist equipment available. Therefore if suspected it is better to err on the side of caution and assume it is present. Unfortunately clinical diagnosis may be difficult in the uncooperative or unconscious patient, or if morphine has been given. In these cases a tense proptosis and fixed dilated pupil may be the only obvious signs. Electrophysiological testing of the visual pathway, such as with flash-evoked visual potentials (VEPs) and electroretinograms (ERGs), have been reported, but these are generally too cumbersome and not available on an emergent basis.

Time is of the essence. Current evidence suggests that irreversible changes occur after approximately 90 min of ischaemia. Therefore in any patient with deteriorating vision and increasing pain treatment is more likely to be successful if performed when vision is still present. Once they are blind the damage is often done. If

possible, perform an immediate lateral canthotomy and cantholysis. If this is not possible, seek immediate help. Like a surgical airway, this needs to be done immediately and any delay waiting for a 'specialist' to arrive will likely worsen the prognosis. Urgent referral to ophthalmology and other specialists is of course required (depending on the cause and local referral pathways), but do not delay treatment waiting for the on call team to arrive. However, a common error is to assume lateral canthotomy is definitive treatment. This is usually not the case and canthotomies only buy time. Formal decompression and drainage may then be required, depending on the overall clinical picture and likelihood of achieving decompression in a timely fashion.

17.3.1 Lateral Canthotomy and Cantholysis

The lateral and medial canthal tendons attach the eyelids to the orbital rim and to see extent, limit anterior displacement of the globe. The tarsal plates fuse medially and laterally to form the canthal tendons (palpebral ligaments). The lateral canthal tendon has a superior and inferior crus that inserts at the lateral orbital tubercle attached to Whitnall tubercle, which is about 3 mm deep to the lateral orbital rim. Lateral canthotomy with cantholysis is an emergency procedure which may be necessary in patients with acute vision-threatening proptosis. Whilst medical treatments with steroids, mannitol, and acetazolamide may be initiated, in many cases these are only an adjuvant. Canthotomy with cantholysis quickly releases some of the intraorbital pressure. However no drainage of the orbital compartment occurs. This may also be required.

Lateral canthotomy with lateral canthal tendon division can be performed under local anaesthesia in the emergency department, or on the ward. The essential component that relieves the orbital pressure is the cantholysis, not the canthotomy. Equipment needed for the procedure includes:

i) Sterile gloves and drapes
ii) Lignocaine 1% with adrenaline (1 in 200,000)
iii) Syringe with 27- to 30-gauge needle and normal saline for irrigation
iv) Straight haemostat
v) Sterile iris or suture scissors
vi) Forceps

The patient should be positioned so comfortable, ideally supine with the head of the bed elevated 10–15°. Lignocaine with adrenaline is injected into the lateral canthal area of the affected eye and the lateral canthus (skin and deeper tissues) is incised fully, down to the orbital rim. The canthal tendon is then cut and separated from Whitnall's tubercle. This tendon is identified by "strumming" the tissues, while the eyelid is pulled away from its attachment. Great care must be taken to avoid damage to the globe. Some authorities recommend division of the upper lid attachment as well. Division of the canthal tendon allows the globe to translate

forward, partially relieving the pressure behind it by effectively increasing the orbital volume. The key steps in this procedure are as follows

i) Clean the site with sterile saline
ii) Inject local anaesthetic into the lateral canthus. Direct the tip of the needle towards the lateral orbital rim, injecting when the needle touches bone.
iii) While waiting for the local to work, quickly irrigate the eye with normal saline to clear away any debris.
iv) Clamp the skin of the lateral canthus all the way down to the orbital rim for 1–2 min. Advance the tip of the clip into the lateral fornix until the rim of the bony orbit is felt. This will facilitate haemostasis and mark the location of the incision.
v) Use forceps to pick up the skin around the lateral orbit. Then make a 1 cm long horizontal cut through the crush mark and skin. This will divide the skin, fascial septum, orbicularis oculi muscle and conjunctiva. This step only serves to expose the canthal tendon. Whilst it my decrease some pressure, it is often insufficient alone. Therefore, proceed to cantholysis.
vi) Grasp the lower eyelid with the forceps, and gently pull it away from the face. This pulls the inferior crus of the lateral canthal tendon taught so that it can be identified and cut. It will have a "violin string" feel to it
vii) Place the inner blade the scissor just anterior to the conjunctiva and the outer blade just deep to the skin. Then cut
viii) The globe should pull freely away. It may also 'pop' forward, relieving the pressure. Cut any residual lateral attachments of the lower eyelid if it does not move freely. The eyelid wound may bleed profusely, but there is very little drainage from the retrobulbar haematoma itself.
ix) If inferior cantholysis does not decrease the orbital pressure, a superior cantholysis should then be performed.
x) Do not worry about cutting 1 cm of conjunctiva or skin.
xi) If the intact cornea is exposed apply ointment or lubricant to prevent corneal desiccation and infection.
xii) Do NOT apply absorbent gauze dressing to the exposed cornea.
xiii) Medical measures should also be commenced (Mannitol 20% 2 g/kg IV over 5 min, Dexamethasone 8 mg IV and Acetazolamide (a carbonic anhydrase inhibitor which reduces production of aqueous humour), 500 mg IV then 250 mg 6 hourly for 24 h) (Fig. 17.33)

Visual acuity is the key to urgency. If the vision is normal, patients can be investigated or referred. But if the vision is rapidly deteriorating, or has already significantly decreased, this requires urgent action. Think of the vision as the "GCS of the eye". The most common pitfall with this technique is failure to fully expose the canthal tendon and fully divide it. As long as the instruments are directed towards the orbital rim, tangential to the globe surface, injury to globe or extra ocular muscles is unlikely. Delayed complications, such as infection, functional impairment and cosmetic deformity are uncommon and managed later. Spontaneous healing is

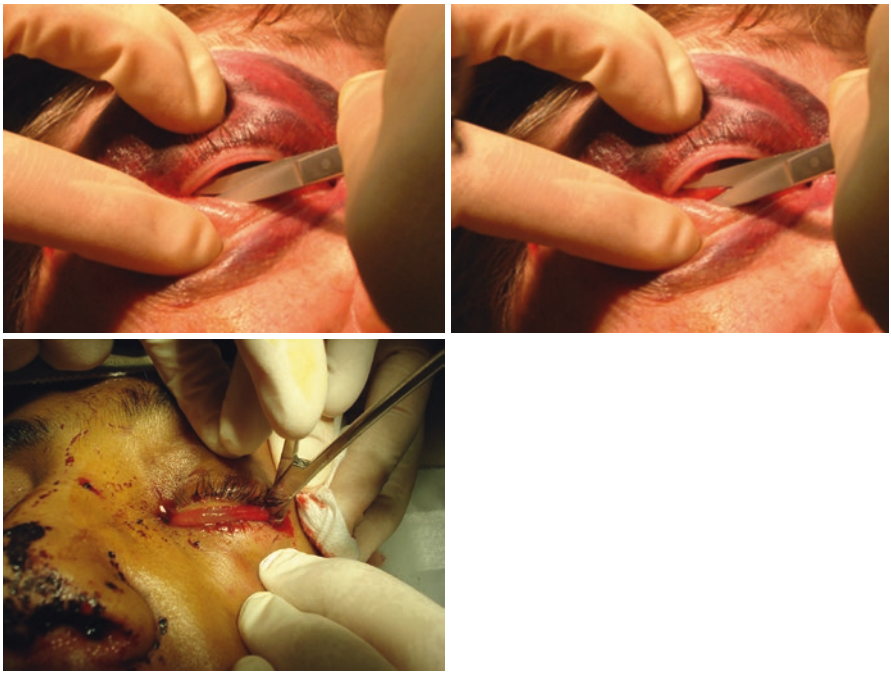

Fig. 17.33 Lateral canthotomy and cantholysis

usual and late repair of the canthotomy is not usually required. Depending on the mechanism of injury, remember also the possibility of globe rupture (suggested by enophthalmos rather than proptosis). This is a contraindication to surgical decompression.

17.4 Orbital Apex Injuries and Orbital Apex Syndrome

A common cause of visual impairment following trauma is a localised injury to the optic nerve within the optic canal. This is referred to as traumatic optic neuropathy—TON. Shearing, contusion, or compression of the nerve as it passes through the canal may all be involved to varying extents. These injuries may occur with or without associated fractures of the optic canal, or skull. In fact traumatic optic neuropathy can occur following a blow to the forehead, without any fractures occurring at all. Orbital apex syndrome is another well-known but rarer complication of high energy trauma, which combines the clinical features of superior orbital fissure syndrome with traumatic optic neuropathy. Symptoms thus include visual impairment (or blindness), ophthalmoplegia (oculomotor, trochlear, and abducens nerve injury) and forehead numbness (ophthalmic branch of the trigeminal nerve) (Figs. 17.34 and 17.35).

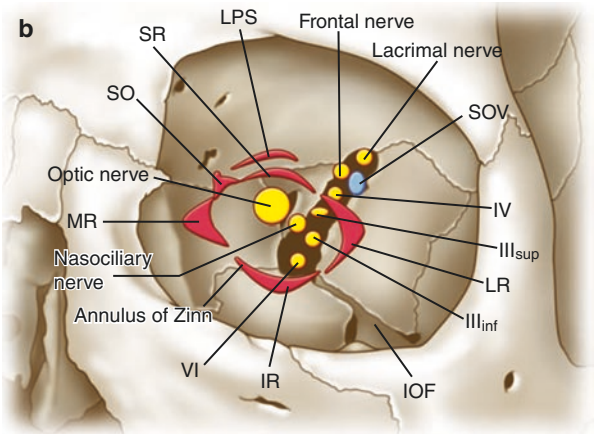

Fig. 17.34 The orbital apex is an important anatomical site for ocular function

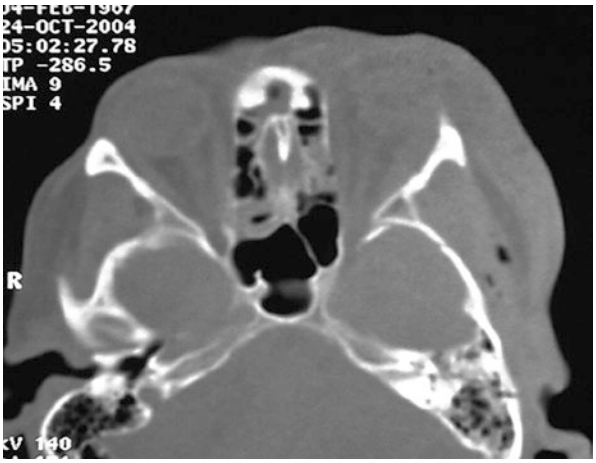

Fig. 17.35 High energy impact. Note fractures around orbital apex

In both traumatic optic neuropathy and orbital apex syndrome, injuries may be classification as direct or indirect. Direct injuries arise from radiologically identifiable compression of the nerves within the optic canal or superior orbital fissure. This is usually from a fracture fragment, although localised haematoma can also give rise to this. With indirect neuropathy there is no radiographic evidence of a fracture or abnormality around the nerves. If vision is lost at the moment of impact, even with CT evidence of compression, decompression of any bony fragments is unlikely to increase the chance of visual recovery—the damage has already been done. Steroids may be given but their efficacy has been questioned. However, when

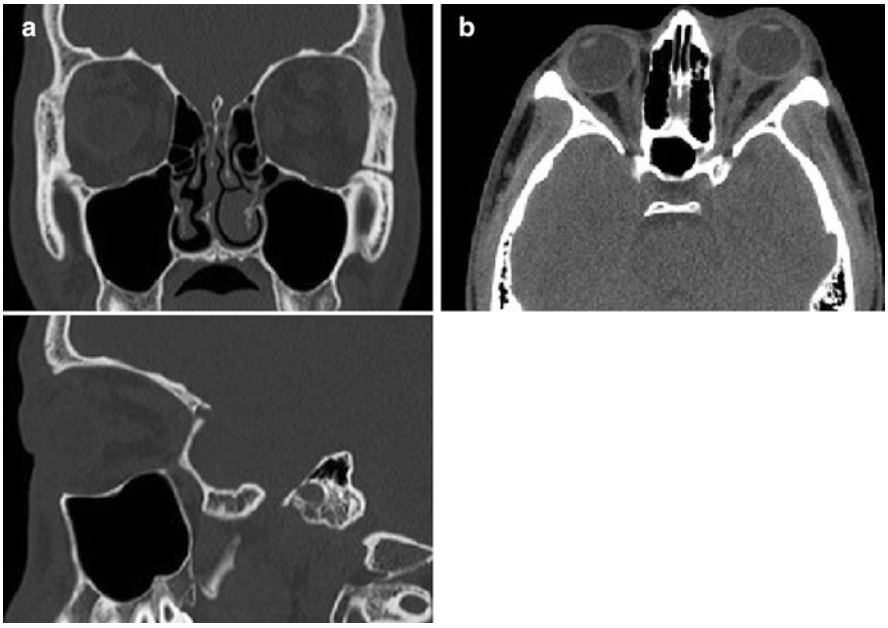

Fig. 17.36 Case study. Left optic nerve sheath hemorrhage following a forceful poke to the left eye while playing basketball. On presentation, he was NLP OS, pupils were 4 mm OD and 5 mm OS with a left RAPD, and he was unable to supraduct the left eye. (**a**) Coronal CT scan of the orbit with increased size of the left optic nerve, from hematoma. (**b**) Axial CT scan of orbit shows enlarged left optic nerve and left proptosis. He underwent emergent optic nerve sheath decompression without subsequent improvement of vision

a bone fragment results in fluctuating or deteriorating vision, urgent optic canal decompression should be considered. Whilst timing is controversial, it has been reported that irreversible ischaemic damage to the optic nerve occurs as soon as 90 min after injury, and certainly after 2 h. If vision is initially present after injury but then begins to deteriorates, swelling from haemorrhage and oedema may be compressing the optic nerve. Surgical decompression or high-dose steroids may then be indicated. Some specialists argue that optic nerve injury, even with no light perception should still be treated with decompression, but this is controversial and the prognosis generally remains poor no matter what is done (Fig. 17.36).

17.5 Injuries to the Lacrimal Gland

The lacrimal gland is located and protected in a recess in the lateral part of the orbital roof. Nevertheless it can be injured following localised head trauma. There have also been reports of lacrimal gland fistulae arising following surgery to the area (cranial/orbital repair and upper eyelid surgery). Congenital fistula can also occur resulting in misdirected lacrimal ductules. Following trauma the watery

discharge may be confused with CSF. Skin wounds in this region should be carefully explored and closed. Prolapse of lacrimal gland is an unusual event which can also occur following trauma. It can also occur secondary to thyroid eye disease, orbital tumours or even spontaneously. Traumatic dislocation occurs exclusively in children since the upper and lateral orbital margins are not well developed.

17.6 Traumatic Globe Subluxation

This is a rare condition and is usually seen following very high energy impacts such as motor vehicle collisions. Other causes include brake handle injuries, eye gouging during an assault and a finger poke into the orbit during sports. These are very specific mechanisms in which there is either a very heavy impact applied to the forehead or cheek, or an object is forcibly positioned behind the globe, pushing it forwards. Because such injuries are usually high energy, orbital, eyelid and intracranial injuries are frequently associated. The globe itself may also be severely damaged internally. Not surprisingly there is usually irreversible visual loss. Spontaneous luxation also has been reported in conditions such as craniofacial dysostosis, intra osseous meningioma invading the orbit, Grave's disease, orbital tumours and floppy eye-lid syndrome. In these conditions the patient is predisposed as a result of a 'shallow' orbit, space occupying pathology, or lack of anterior globe support. In their normal positions, the eyelids play and important role in supporting the globe and preventing it from dislocating anteriorly. However, their protective effects are easily overcome following high-energy impacts. Soft tissue laxity, shallow orbits, exophthalmos and eyelid manipulation are all additional risk factors. In some cases the extra ocular muscles and optic nerve may remain intact in patients with spontaneous globe luxation. They can also be severely damaged, resulting in partial or complete avulsion of the optic nerve, as seen in patients with traumatic globe luxation. Avulsion of the nerve can result in CSF oculorrhea.

In the first instance management is directed towards protecting the globe from further damage and salvaging whatever vision remains. This includes the liberal application of an antiseptic eye ointment and mechanical protection using a hard plastic cover, such as a gallipot. Urgent referral is required, although the possibility of coexisting intracranial injury must not be forgotten. Urgent CT is therefore indicated. Definitive management involves repositioning the eye back into the orbit, if this is possible. This may require removal of a foreign body, or reduction of surrounding fractures. To reduce the globe itself the upper eyelid is pulled upward and the globe is simultaneously pressed backwards. In order to do this rupture of the globe must have been confidently excluded beforehand. This technique may also be difficult if there is any haemorrhage or oedema within the orbit. These will need to be drained before reduction is possible. Ocular complications include anterior segment ischaemia, phthisis bulbi and glaucoma.