Orbital and Ocular Trauma

Hany A. Emam and Deepak G. Krishnan

Contents

M. Miloro et al. (eds.), *Peterson's Principles of Oral and Maxillofacial Surgery*,
https://doi.org/10.1007/978-3-030-91920-7_24

Learning Aims

1. Understand the patterns of orbital and ocular injuries
2. Learn to properly examine and diagnose orbital and ocular injuries
3. Understand principles of surgical and non-surgical management of orbital and ocular trauma
4. Appreciate the technical aspects of surgical approaches to the orbit
5. Acknowledge the technological advancements that might aid in the management of these injuries.

24.1 Introduction

Orbital and ocular injuries are commonly associated with functional and esthetic deformities. These injuries can be the result of assaults, motor vehicle accidents, sport related, and falls. Motor vehicle accidents are the ones usually associated with the most complex fracture patterns with involvement of other midface fractures (ZMC or NOE fractures). Management of such injuries remain challenging since the anatomy of the orbital region is complex with various vital structures and highly specialized organs bundled in a small space. A multitude of approaches exist, and numerous materials are available for reconstruction. No individual approach and no single material is best suited for all patients. Each individual case should be thoroughly evaluated with the help of other services as necessary in order to formulate the appropriate treatment plan. In the acute setting, posttraumatic complications either related to the injury itself or to the repair are difficult to predict and present a very challenging situation for repair. However, advances in maxillofacial and orbital imaging and the introduction of computer virtual surgical planning, intraoperative navigation systems, and improved implant designs have led to more accurate and satisfactory post-operative outcomes.

24.2 Orbital Fractures

24.2.1 Anatomy

The orbit is the bony vault that houses the eyeball or globe. It is a quadrangular-based pyramid that has its peak at the orbital apex. The average adult orbit has a volume of 30 cc; the globe averages 7 cc (Fig. 24.1). Even a modest change in the position of one of the bony walls can have a significant impact on the orbital volume and, thus, globe position. The orbit serves to house and protect the globe. By age 5 years orbital growth is 85% complete, and it is finalized between 7 years of age and puberty [1, 2].

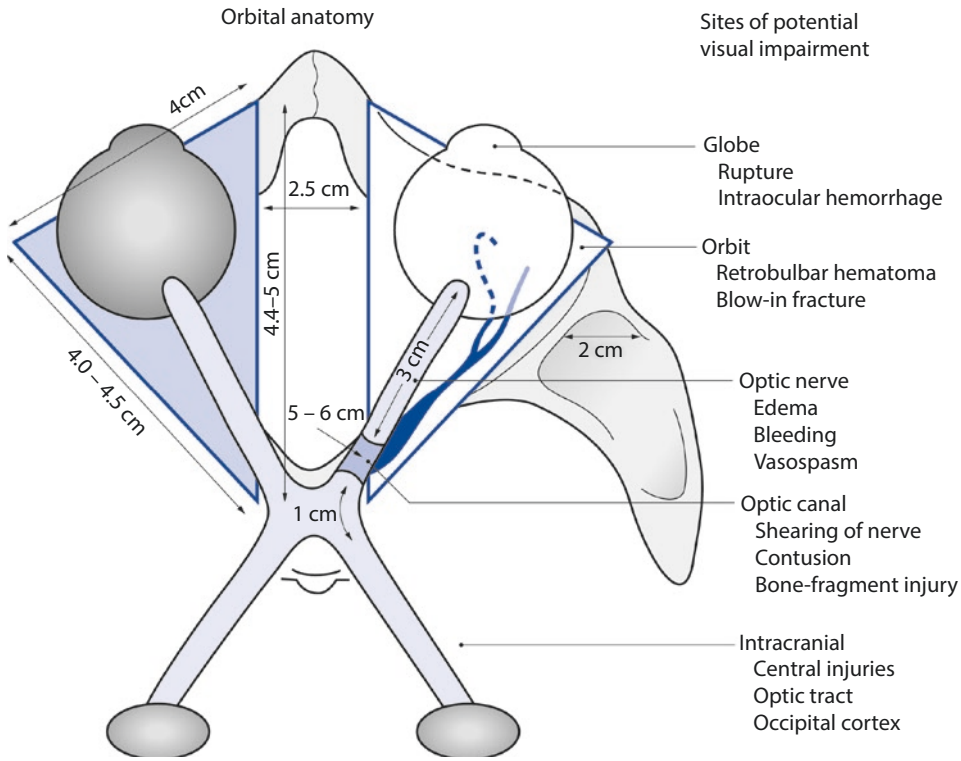


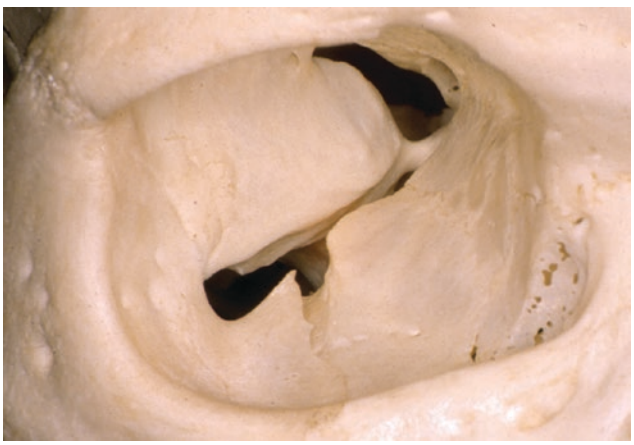
Fig. 24.1 Orbital configuration with potential sites for traumatic injuries leading to visual impairment. (Adapted from Ochs and Johns [96])

The orbital rim is composed of dense cortical bone that generally protects the orbital contents and globe from direct blunt trauma. Seven bones form the orbit: maxillary, zygomatic, frontal, ethmoidal, lacrimal, palatine, and sphenoid. Besides forming a protective socket for the globe, these bones also provide origins for the extraocular muscles, and foramina and fissures for cranial nerves and blood vessels [3].

The orbital walls vary considerably in their thickness. Whereas the superior lateral and inferior rims tend to be rather thick, the bones just posterior to these and the medial rim are usually fairly thin (<1 mm). Fractures of the anterior and middle thirds of the bony orbit are fairly common. The orbital floor and medial wall are most frequently fractured outward owing to their thinness and lack of support. This is a fortunate design since inward or medial displacement of midfacial or zygomatic bones can reduce the orbital volume and be accompanied by direct globe injury resulting in hemorrhage/rupture. The subsequent increased intraorbital pressure is most often relieved by traumatic expansion of the walls with herniation of orbital tissue into the maxillary sinus and/or ethmoid air cells adjacent to these walls. In essence, the paranasal sinuses and ethmoid air cells serve as air bags or shock absorbers to the globe and orbital contents. This protective mechanism explains why globe perforation is relatively uncommon following midfacial trauma. Orbital fractures that involve the frontal sinuses more commonly result in serious eye injuries [4, 5]. These fractures, following blunt trauma, and the associated blindness are probably not seen as often owing to the severity of forces and concomitant neurologic, cervical spine, and multisystem trauma. In short, they generally are not survivable events.

The orbital roof consists mainly of the frontal bone, with the anterior cranial fossa superior to it. The lesser wing of the sphenoid has a minor contribution posteriorly. The superior orbital rim is generally rather thick and then rapidly becomes quite thin (<1 mm) posterior from the edge. In elderly patients the orbital roof may be resorbed in select areas, allowing the dura to become confluent with the periorbita. This should be kept in mind during orbital dissection and elevation in this region for both trauma and tumor work in that population. Generally, the anterior portion of the orbital roof is occupied by the supraorbital extension of the frontal sinus. The frontal sinus begins to form around the age of 6 years and is unilateral in 5% of adults and lacking in another 5%. Anterolaterally there is a smooth broad fossa that houses the lacrimal gland. At the most medial extent is the trochlea, approximately 4 mm behind the rim. There the cartilaginous pulley has a dual insertion for the superior oblique muscle tendon. At the junction of the medial one-third and lateral two-thirds of the superior rim is the supraorbital notch. In one-fourth of adults, a supraorbital foramen is found, secondary to the ossification of the ligament crossing the inferior extent [6]. When reflecting coronal flaps, a small triangular wedge ostectomy should be performed in these individuals to relieve the encased supraorbital nerve and vessels and to allow for a relaxed reflection of tissues at the rim.

The orbital floor is bordered laterally by the inferior orbital fissure. However, there is no distinct border medially (◘ Fig. 24.2). The orbital floor is formed primarily by the orbital process of the maxilla-anterolaterally by a portion of the zygomatic bone and posteriorly by a small portion of the palatine bone. The maxillary sinuses are present at birth and reach the orbital floor and infraorbital canal by age 2 years [7]. The inferior orbital fissure gives rise to the infraorbital groove from its midportion, which is about 2.5–3 cm from the infraorbital rim. The infraorbital fissure converts to a canal halfway forward, carrying the infraorbital nerve and vessels and opening approximately 5 mm below the rim of the maxilla as the infraorbital foramen (◘ Table 24.1) [8]. The infraorbital nerve provides sensory innervation to the upper lip, lower eyelid, lateral nose, and anterior maxillary teeth and mucosa. The orbital floor can be as thin as 0.5 mm, with its weakest portion just medial to the infraorbital groove and canal. This explains the phenomenon that most blunt traumas resulting in orbital floor blowouts are manifested primarily with injury and sagging of the medial orbital floor and orbital contents into the underlying maxillary sinus with extension laterally to the infraorbital canal.



◘ **Fig. 24.2** Right bony orbit. The inferior orbital fissure can be seen converting to a canal angling medially at the Y-shaped divide. The lacrimal fossa is characteristically thin. The lamina papyracea occupies the majority of the medial wall, with the frontoethmoidal suture at the superior extent

Table 24.1 Orbital fissures/canals and their contents

Location	*Contents*
Superior orbital fissure—lesser and greater wings of the sphenoid Inferior orbital fissure—greater wing of the sphenoid; palatine, zygomatic, and maxillary bones Optic canal—lesser wing of the sphenoid Anterior ethmoid canal—frontal and ethmoid bones Posterior ethmoid canal—frontal and ethmoid bones Nasolacrimal fossa—lacrimal and maxillary bones	Motor nerves: III (superior and inferior divisions), IV (trochlear), V (abducens) Sensory nerves: V_1 (frontal, lacrimal, nasociliary), sympathetic fibers Vessels: superior ophthalmic vein, anastomosis of recurrent lacrimal and middle meningeal arteries Sensory nerves:V_2 (infraorbital and zygomatic), parasympathetic branches of the pterygopalatine ganglion Vessel: inferior ophthalmic vein and branches to pterygoid plexus Optic nerve, meninges, ophthalmic artery, sympathetic fibers Nerve: anterior ethmoid becomes dorsal nasal Vessel: anterior ethmoid artery Nerve: posterior ethmoid Vessel: posterior ethmoid artery Nasolacrimal sac and duct

The lateral wall of the orbit is formed mainly by the greater wing of the sphenoid and portions of the zygoma. Although this tends to be the strongest wall, it is fairly commonly fractured along the frontozygomatic junction, extending slightly posteriorly and then running vertically along the thinnest portion of the suture line, where the greater wing of the sphenoid and zygoma meet. This wall separates the orbit from the temporalis muscle. Owing to the heavy nature of this muscle and the direction of blunt forces, generally there is some mild degree of inward displacement. The lateral orbital walls, if they were to be extended posteriorly, would form a 90° angle to each other. Each lateral orbital wall forms a 45° angle at the orbital apex, with its medial wall counterpart. This is important to bear in mind when attempting to realign or reconstruct fractured walls. The superior orbital fissure separates the greater and lesser wings of the sphenoid and serves as the delineation between the orbital roof and lateral wall. At the orbital apex, the lesser wing of the sphenoid forms the lateral portion of the ring of the optic canal. One centimeter below the frontozygomatic suture, and just internal (3–4 mm) to the lateral orbital rim, is Whitnall's tubercle (lateral orbital tubercle). This gentle outcropping of bone functions as the insertion point for the lateral retinacular structures. The lateral retinaculum is composed of the lateral horn of the levator aponeurosis; the lateral canthal tendon of the eyelids; and the inferior suspensory (Lockwood's) ligament and multiple fine check ligaments of the lateral rectus muscle. These soft tissue attachments are found anatomically in this order proceeding inferiorly and posteriorly from the rim. These multiple structures become confluent to form the common lateral retinaculum, which is the actual insertion to the tubercle [6]. Clinically the point to remember is that reattachment of the lateral canthal tendon should be to the lateral orbital tubercle.

The medial wall of the orbit is by far the most complex and potentially problematic to manage in severe trauma. The medial orbital wall is composed anterior-to-posterior by a portion of the maxillary, lacrimal, ethmoid, and sphenoid bones. The majority of the medial wall is formed by the extremely thin (0.2–0.4 mm) lamina papyracea of the ethmoid bone. Housed along the frontoethmoidal junction are the anterior and posterior ethmoidal foramina. The anterior ethmoidal foramen is 20–25 mm behind the medial orbital rim, and 12 mm beyond this is the posterior ethmoidal foramen. The foramina can be found approximately two-thirds of the way up the medial orbital wall, within the frontoethmoidal suture line, and serve as important surgical landmarks identifying the level of the corresponding cribriform plate. These arteries serve as the landmarks for the superior extent of orbital wall decompression in surgery of that region. The anterior ethmoidal foramen transmits the anterior ethmoidal artery and anterior ethmoidal branches from the nasociliary nerve from the orbit coursing into the nasal cavity. This is why otolaryngologists sometimes use a medio-orbital approach to ligate or cauterize the anterior ethmoidal artery to control recalcitrant nasal bleeding. Although the anterior ethmoidal vessel can be cauterized with few long-term deliterious effects, the contents of the posterior ethmoidal foramen (posterior ethmoidal artery and, variably, a sphenoethmoidal nerve from the nasociliary nerve) are generally allowed to remain intact since they serve as a useful delineation to the posterior extent of safe medial wall dissection.

Once beyond the orbital rims, subperiosteal dissection generally proceeds fairly easily, except for points of nerves or vessels perforating through foramina, orbital fissures, or muscle origins such as that of the inferior oblique. When encountering resistance, surgeons should attempt to identify the exact anatomic reason for the resistance, such as structures that may need to be preserved or periorbital tissues that have become entrapped in fracture lines. Knowledge of the limits of safe subperiosteal dissection is imperative. Also important is knowing the distance from the intact orbital rim, where vital structures can be identified. Generally, a subperiosteal dissection from the inferior lateral rims can be safely extended for 25 mm. An exploration distance of 30 mm from the superior orbital rim or anterior lacrimal crest (found on the frontal process of the maxilla) can be safe [5]. A high medial wall dissection places the orbital apex and optic canal at risk. One caveat to these "safe surgical exploration distances" is that they are averages of known landmarks to intact adult orbital rims. When traumatic forces displace a portion of a rim, it is generally in a posterior or medial direction, which effectively reduces these distances. Knowledge of the bony orbital anatomy, with its foramina, fissures, and attachment areas, helps the surgeon to avoid injuries to vital structures contained within them [1]. Average distances for locating these critical structures as they relate to identifiable bony landmarks are contained in Table 24.2. Surgeons should avoid disrupting the medial canthal tendon, lacrimal apparatus, a pulley of the superior oblique muscle, supraorbital nerves and vessel, attachments to Whitnall's tubercle, and the origin of the inferior oblique muscle.

Table 24.2 Distance of vital orbital structures from Bony Landmarks

Structure	*Reference landmark*	*Mean distance (mm)*
Midpoint of inferior orbital fissure	Infraorbital foramen	24
Anterior ethmoidal foramen	Anterior lacrimal crest	24
Superior orbital fissure	Zygomaticofrontal suture	35
Superior orbital fissure	Supraorbital notch	40
Optic canal (medial aspect)	Anterior lacrimal crest	42
Optic canal (superior aspect)	Supraorbital notch	45

The anterior boundary of the orbit is defined by the orbital septum. The upper and lower eyelids are anatomically similar in their composition, with corresponding layers anteriorly to posteriorly. When one is looking downward, the lid retractors enable the lower eyelid to roll with the globe, thus avoiding a visual field cut. The lids have a very thin keratinized epithelium that is loosely attached to the underlying orbicularis oculi muscle (▶ Box 24.1). The orbicularis oculi muscle is innervated by cranial nerve VII and acts as a sphincter and closing force for the eyelids. In the relaxed state, the orbicularis oculi is opposed in the upper eyelid by the levator palpebrae superioris, which is innervated by cranial nerve III. The resting tone and level of the upper eyelid are partly determined by the amount of sympathetic input to Müller's muscle. The orbicularis oculi have two distinct layers: the outer superficial fibers (orbital portion) and the deeper fibers (palpebral portion). The palpebral section medially has intricate insertions and envelops the lacrimal sac by dividing into intertwined deep and superficial heads. The superficial portion inserts onto the anterior lacrimal crest. The inner deep head inserts into the fascia of the lacrimal sac and posterior lacrimal crest. The medial canthal tendon is formed by the condensation of the orbicularis muscle fibers. It is the superficial head of the canthal tendon that has a tenacious insertion into the anterior lacrimal crest. This is beneficial during orbital approaches since the anterior insertion offers considerable resistance to dissection, which helps one avoid inadvertent injury to the lacrimal sac. At the lateral edge of the orbicularis oculi, the superficial fibers form an indistinct raphe, and it is the deeper fibers that comprise the lateral canthal tendon, inserting onto Whitnall's tubercle [9]. The upper and lower lids should form a 30–40° angle at the lateral canthus, which is situated 1 cm below the frontozygomatic suture. Typically, the lateral canthus is situated 2–4 mm above the medial canthus.

Box 24.1 Eyelid layers: cutaneous (anterior) to conjunctival (posterior)

Skin
Subcutaneous areolar tissue
Striated muscle (orbicularis oculi)
Submuscular areolar tissue (contains main sensory nerves to lids)
Fibrous layer with tarsal plates
Nonstriated smooth muscle
Mucous membrane or conjunctiva

Just posterior to the orbicularis oculi is the orbital septum. The orbital septum is continuous with the orbital periosteum and the periosteum of the facial bones overlying the rims. One to two millimeters below the inferior rim, where these layers converge on the facial aspect, is a periosteal thickening called the arcus marginalis [5]. This is a useful landmark when performing an infraciliary or preseptal transconjunctival approach to the inferior rim. If one stays in front of the orbital septum and incises below the arcus marginalis, then orbital contents and fat do not herniate into the field. The distal edges of the orbital septum are inserted into the superior edge of the tarsal plates. The orbital septum and these insertions prevent the pre-aponeurotic orbital fat from herniating out into the eyelids. Superiorly there is a central and medial fat pad, and inferiorly there are three distinct fat pads (medial, central, and lateral). With aging, the orbital septum can become lax and, particularly in the lower lids, result in "baggy lids." Severe sagging of the lower lids is referred to as festooning.

The primary elevator of the upper eyelids is the levator palpebrae superioris muscle. Inferiorly it forms an aponeurosis below Whitnall's ligament that attaches broadly over the anterior tarsal plate. Approximately 15–20 mm above the tarsal plate, the aponeurosis consists of a thickened fascial band, which is termed Whitnall's ligament. This is a suspensory ligament of the lid. The Müller's muscle arises beneath the levator muscle and inserts into the superior border of the tarsal plate. Müller's is a smooth muscle that receives sympathetic input for its tone and helps regulate the resting position of the upper eyelids while the eyes are open. Increased stimulation or sympathetic input causes a "wide-eyed" look and a more alert appearance [10]. The capsulopalpebral fascia and the inferior tarsal muscle in the lower eyelids are also termed the lower lid retractors. The lid retractors are formed from the fibrous attachments of the inferior rectus and inferior oblique muscles, and fuse with Lockwood's inferior suspensory ligament.

The tarsal plate is formed by dense fibrous connective tissue and is primarily responsible for the convex form of each of the lids. The tarsal border parallels the free margin of the eyelid. The horizontal length of each tarsus is approximately 30 mm. The height is greatest in the mid-portion of the lid. The height of the upper tarsus is 10 mm, whereas in the lower lid it is 4 mm. Embedded within the tarsal plates are a fine network of meibomian (sebaceous) glands. When obstructed and chronically inflamed, these glands can form a cyst-like mass called a chalazion.

The lacrimal system is responsible for the lubrication and wetting of the globe. Accessory lacrimal glands perform normal wetting of the eye, and the lacrimal gland produces reflex tearing. The lacrimal gland, which is situated in the anterior aspect of the superior lateral orbit, is divided into two lobes by the levator aponeurosis. The larger orbital lobe lies above the levator aponeurosis, and its tear ducts traverse the palpebral lobe, which has 6–12 tear ductules that empty into the superior lateral fornix. When drilling in this region, such as during a repair of a frontozygomatic fracture, one must take care not to injure the palpebral lobe or to inadvertently remove it, thinking that it is herniated fat; this error often results in a problematic dry eye. Lacrimal secretions, or tears, traverse medially and inferiorly across the globe, wetting the cornea, and accumulate at the medial inferior aspect of the eye. The fluid is then either drawn or pumped into the lacrimal puncta of the upper and lower eyelids. These puncta are only 0.2–0.3 mm in diameter. The upper punctum is usually just slightly medial in relation to the lower punctum. When the lids close, the puncta come into contact. The upper and lower canaliculi travel within the lids, first vertically (2 mm), then horizontally for 8–10 mm, paralleling the lid margin. They join to form a common canaliculus just before entering the lateral aspect of the lacrimal sac, which is one-third of the way down from the upper portion of the sac. Typically, the lacrimal sac is 1 cm in length and 5 mm in diameter. The palpebral portion of the orbicularis oculi has densely intertwined insertions that envelope the lacrimal sac. Inferiorly, the sac drains into the nasolacrimal duct, which has a 12 mm intrabony canal coursing inferiorly and posteriorly that opens into the inferior meatus of the nasal cavity below the inferior concha. This opening is 30–35 mm from the edge of the external nares. Reflux of tears and nasal mucus back up into the nasolacrimal duct is prevented by a mucosal fold called Hasner's valve. With persistent epiphora following trauma or surgical intervention, it is important to establish the precise point of mechanical obstruction that exists within the lacrimal drainage system. Irrigation of the inferior canaliculus may relieve temporary obstruction owing to dry or thickened secretions. A dye disappearance test, Jones I or II, nasolacrimal irrigation, or dacryocystography can help one determine the precise point of obstruction and guide surgical planning. Following trauma or operative intervention, epiphora may be due to hypersecretion from a corneal abrasion, lash ptosis, foreign bodies, or entropion, all of which serve as persistent stimuli leading to reflex lacrimal gland secretion.

24.3 Fracture Configurations

Isolated orbital wall fractures account for 4–16% of all facial fractures. If fractures that extend outside the orbit are included, such as those of the zygomatic complex (ZMC) and naso-orbitoethmoid (NOE), then this accounts for 30–55% of all facial fractures [11, 12].

ZMC fractures are the most commonly occurring facial fracture, second only to nasal fractures. By definition, ZMC fractures are the most common fracture with orbital involvement [13]. The ZMCoften hinges about the frontozygomatic suture with a medial, inferior, and posterior vector of rotational displacement. This is due to the direction and force of blunt trauma and the variable thicknesses of the components of the ZMC. The frontozygomatic area offers the thickest pillar. When fractured there is usually a slight vertical displacement with a reasonable anteroposterior alignment. The much thinner anterior maxillary and lateral orbital floor offers little resistance to fracture and displacement.

Fractures of the NOE are most often due to severe blunt midface trauma. These fractures create cosmetic deformities with a flattening of the nasal dorsum and a widening of the intercanthal distance; they can also be accompanied by a violation of the underlying dura with a cerebrospinal fluid (CSF) leak. Any persistent or copious clear nasal drainage should be tested to determine a β_2-transferrin level to rule out a CSF leak. It is uncommon for the canthal tendons to become disinserted from the bones. This is particularly true of the lateral canthal tendon. Traumatic telecanthus with NOE fractures is a result of a flattening of the nasal bridge and a lateral splaying of the orbital rims and anterior lacrimal crest. Reduction and fixation of these bony segments and, less frequently, direct transnasal wiring are necessary for adequate restoration of medial intercanthal distance and alignment. In adult Caucasians this is typically 29–32 mm; it is slightly more in black and Asian individuals. Lacrimal drainage problems can also arise from severe NOE fractures owing to canalicular or lacrimal sac disruption or scarring.

Internal orbital fractures occur in numerous patterns. These fractures are typically described by their location and the size of the defect. Three basic patterns of internal orbital fractures have been described: linear, blow-out, and complex [14]. Linear internal orbital fractures maintain periosteal attachments and typically do not result in a defect with orbital content herniation; however, they can result in a significant enlargement of the orbital volume with a resulting late enoph-thalmos. Blow-out fractures are the most common. By definition, these are limited to one wall and typically are 2 cm or less in diameter. The most commonly involved wall with a blow-out fracture is the anterior medial orbital floor, followed by the medial wall and, less frequently, the orbital roof, which can present as a *blow-in* fracture. Exploration, repair, or reconstruction of an orbital roof fracture may be indicated if a dural tear is suspected or to prevent a "pulsatile globe." This rhythmic inward and outward movement of the eye is due to the cerebrovascular pulsation and the influence of respiration on the overlying cerebral hemispheres. This phenomenon is typically not present acutely but occurs after the resolution of edema, with the recovered patient complaining of persistent blurred or double vision. Complex internal orbital fractures consist of extensive fractures affecting two or more orbital walls; they often extend to the posterior orbit and may involve the optic canal. These complex fractures are usually associated with more severe trauma and surrounding fractures such as Le Fort II, Le Fort III, and frontal sinus fractures.

24.4 Clinical Examination

Even in the most severely injured patient, the mechanism of injury and surrounding history should be ascertained before performing a clinical examination of the orbit and globe. A systematic approach assessing both the globes and orbits further defines functional and cosmetic defects. The initial ophthalmologic evaluation should include periorbital examination, visual acuity, ocular motility, pupillary responses, visual fields, and a fundoscopic examination. Often, clinical exam is made difficult by edema, blood, debris, and other distractions (Fig. 24.3).

Fig. 24.3 Clinical exam is difficult when the patient is intubated, edematous, and covered in blood and debris

Visual acuity should be independently tested on each eye using a Snellen chart at a standard 6 m (20 ft.) distance or with reading of a standard-type print at 40 cm (16 in.). The patient should wear their corrective lenses during this examination. The eyelids and periorbital region should be inspected for edema, chemosis, ecchymosis, lacerations, ptosis, asymmetric lid drape, canalicular injury, and canthal tendon disruption. With significant acute periorbital ecchymosis, there should be an increased suspicion of a direct blunt globe injury or an internal orbital wall fracture. A lid retractor (Desmarres) is useful for separating swollen tight lids so that the globe and pupil can be adequately examined. Also, this retractor may serve to lift the edge of the lid to examine its inner aspect. With an upper eyelid laceration, any fat that is herniating below the level of the brow through the wound should cause concern that an underlying injury has occurred to the levator muscle. Likewise, if the palpebral conjunctiva has been violated, it is prudent to consult an ophthalmologist to rule out a globe perforation. With a medial vertical laceration of the lids, particularly the lower, gentle lateral retraction may reveal a cut canaliculus or medial canthal tendon disinsertion. Canalicular disruption warrants an urgent ophthalmology consult and usually requires surgical reanastomosis and silicone tube placement into the nasolacrimal system and surrounding supportive repair to prevent outflow obstruction and epiphora.

Extraocular movements are evaluated to rule out mechanical entrapment or paresis. Diplopia, and the field of gaze in which it occurs, should be noted (Fig. 24.4). Of greatest concern is diplopia in the primary (straight-ahead) and downward gazes. These are the two fields that are used most often. Mild or equivocal restriction (<5°) in extreme fields of gaze is common in the setting of severe orbital trauma with hemorrhage or edema. Computed tomography (CT) scan findings should be correlated with any clinically noted entrapment. If mechanical entrapment is suspected, then the eye should be topically anesthetized and a forced duction performed with a fine-toothed forceps. Typically, an Adson forceps is used at the inferior fornix with the

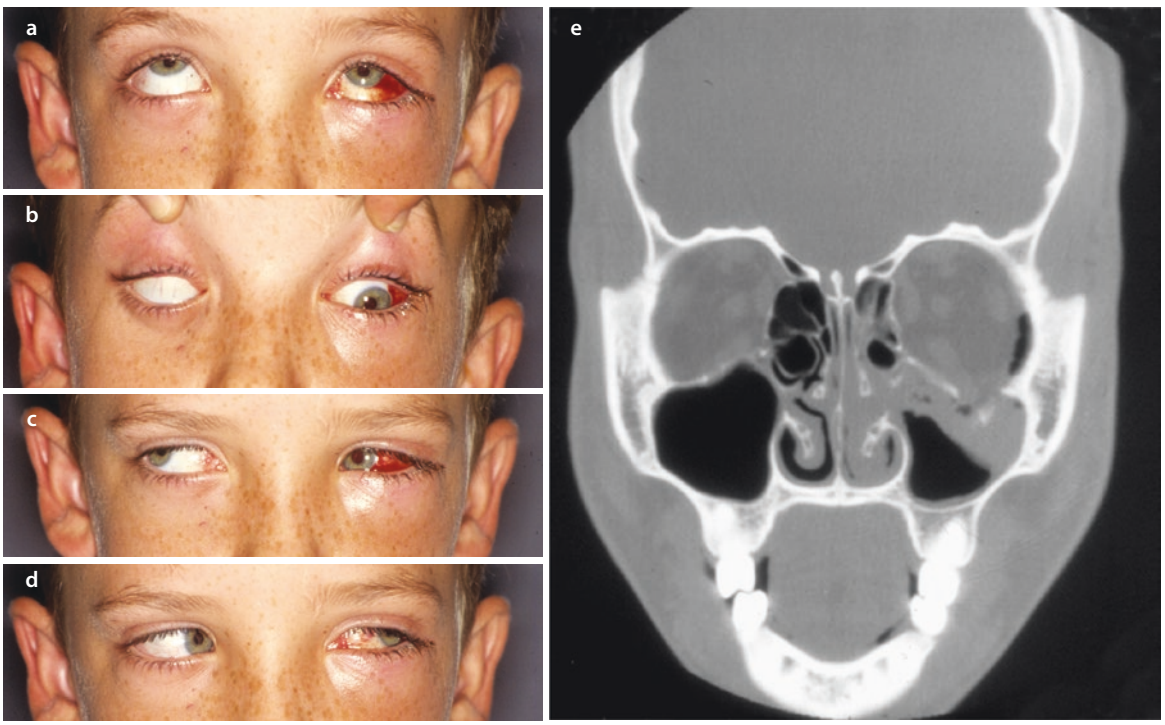


Fig. 24.4 This 9-year-old child presented with a complaint of "double vision and cheek numbness" after being struck in the left orbital region with a hardball. **a** Note the lateral subconjunctival hemorrhage and that there was no difficulty in the upgaze. **b** In downgaze he had severe firm-fixed restriction of the left eye that was positive to a forced duction test. **c** The right lateral gaze had trace restriction. **d** The left lateral gaze was unremarkable. **e** Direct coronal computed tomography (CT) scan of the bony window revealed a trapdoor fracture of the left orbital floor with herniation and a probable impingement of the inferior oblique muscle and fascial framework. **f** Diploic visual fields (Goldman visual field test). With binocular testing, patients are asked to look at the grid and track a pointed light that is shown from behind the chart. When patients experience double vision, they respond to the examiner who charts the abnormality. In this case, the upper grid was recorded at the initial presentation. Diplopia was experienced in all areas below the line (10–12°). This child's severely limited downgaze, correlated with the CT findings, prompted surgical exploration, and orbital floor repair within 12 hours. The lower grid was recorded at 10 days postoperatively and showed marked improvement in the downgaze, with diplopia occurring at 40° inferiorly. **g** An Adson forceps is used at the inferior fornix with the beaks open, pressing inward against the depth of the fornix and toward the globe side, until the globe rolls downward slightly. **h** The beaks are then pressed together, grasping the insertion of the inferior rectus muscle. **i** Upward, downward, and lateral motions can be evaluated

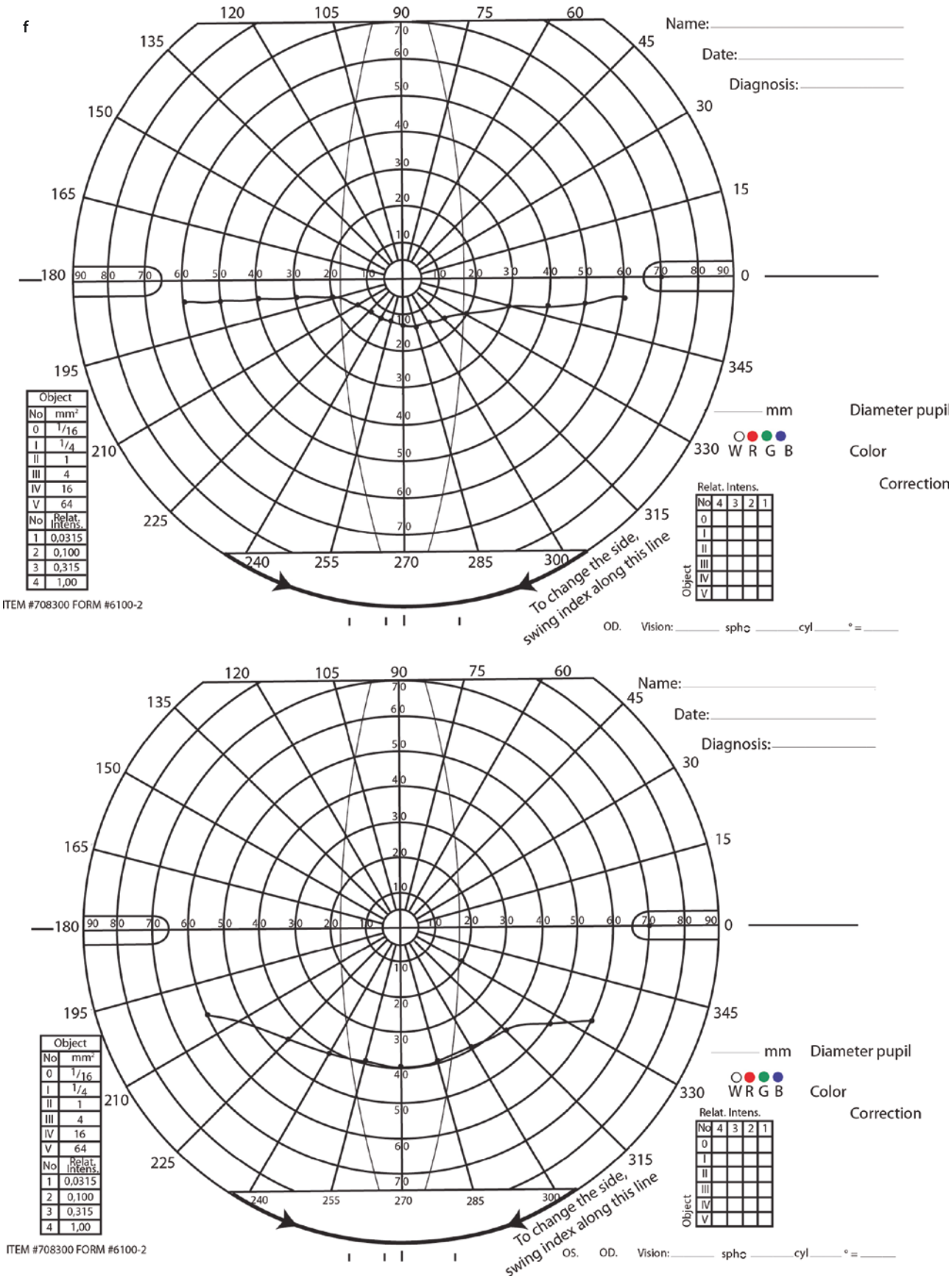


Fig. 24.4 (continued)

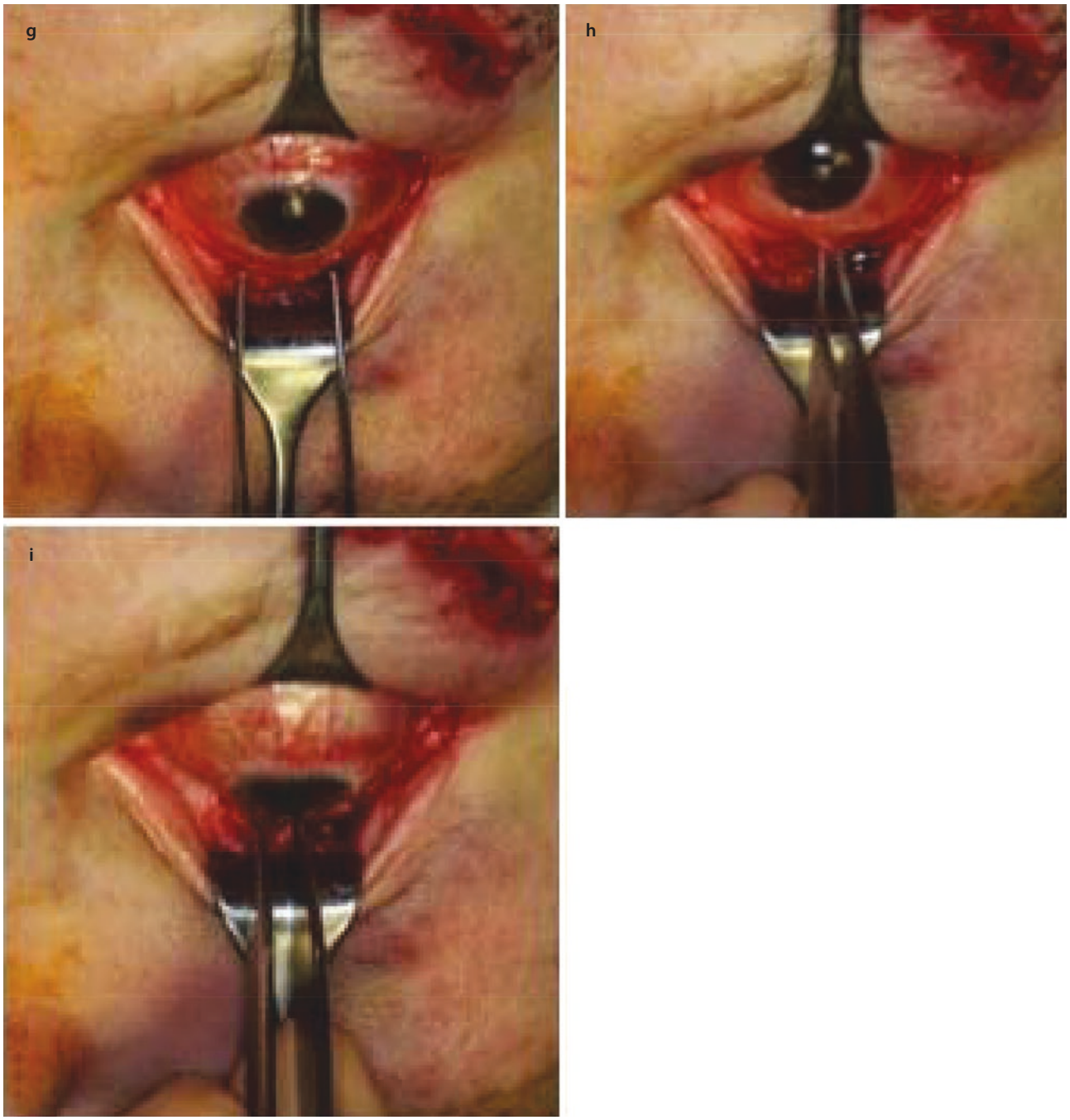


Fig. 24.4 (continued)

beaks open, pressing inward against the depth of the fornix and toward the globe side, until the globe rolls downward slightly. The beaks are then pressed together, grasping the insertion of the inferior rectus. Upward, downward, and lateral motions can be evaluated (Fig. 24.4b). The point of doing a forced duction test is to determine whether the diplopia is due to a restriction of a muscle or paresis of a muscle.

Fig. 24.5 Laceration of the right lower medial eyelid that extends through the margin. At first the examiner thought, there was simply a strand of clotted blood on the medial globe. Recognition of the irregular-pointing pupil led to the suspicion of a globe perforation, which was confirmed with a dilated ophthalmologic examination

Pupillary light reactivity, size, shape, and symmetry should all be assessed and noted. If unequal pupils (anisocoria) or an irregularly pointing pupil is found, then the patient should be queried regarding previous ocular trauma or eye surgery (cataracts). An irregular pupil often points toward the site of a globe penetration or injury. This is often teardrop shaped, with the narrow portion pointing toward the perforated side of the globe, which is usually concealed beneath the lid (Fig. 24.5). An ophthalmologist should be consulted immediately and precautionary measures instituted, including a protective Fox shield over the eye, head-of-bed elevation, bed rest, analgesics, and antiemetics to avoid sudden increases in intraocular pressure owing to Valsalva forces.

Both globes should be evaluated for any acute enophthalmos, exophthalmos, or vertical dystopia. This is often ascertained from above or by standing directly in front of the patient. Visual fields are tested for each eye, one at a time, by confrontation. The examiner and patient faces should be positioned directly toward each other, 0.6 m apart. The patient is asked to stare directly into the examiner's eyes, while the examiner's hand is held in their own extreme field of gaze, midway between the patient and the examiner. The patient is then asked to detect numbers of fingers showing, motion, or the digit displayed. In essence, the examiner's peripheral field of gaze is serving as a control for the patient.

Quadrant defects are indicative of post-chiasm injury. A fundoscopic examination should be performed in a dimly lit room to help maximize pupillary dilatation and ease of the examination. Lens dislocation, vitreous hemorrhage, retinal detachment, and foreign bodies may be noted or may be the cause for not being able to view the fundus. If history and initial clinical findings warrant a dilated fundoscopic examination, then neurologic status should be reevaluated and confirmed, and clearance from the primary treating physician or neurosurgeon first obtained. A dilated fundoscopic examination with indirect ophthalmoscopy is generally performed by an ophthalmologist to rule out more occult injuries or examine a greater portion of the globe toward the equator. The ophthalmologist may elect to perform tonometry or a slit-lamp examination. A loss of red reflex is one of the earliest fundoscopic findings of vitreous hemorrhage and must make the clinician aware of possible ocular trauma. Tonometry indirectly measures intraocular pressure by placing the instrument on the surface of the eye. Normal (10–20 mm Hg) or symmetric bilateral readings are reassuring. However, this does not rule out a penetrating injury. With elevated pressures but an otherwise unremarkable examination, a history of glaucoma should be elicited. An acute abnormally high intraocular pressure with exophthalmos, limited globe movement, and resistance to retropulsion is indicative of a retrobulbar hematoma, which may require acute evacuation via a lateral canthotomy. A "soft eye" with a relatively low pressure or deep anterior chamber is suggestive of a posterior scleral rupture.

A slit-lamp examination is generally performed with the patient in an upright position; if the patient is confined to a bed, a modified examination can be performed with a penlight. A handheld portable slit lamp can be used in the trauma setting. The purpose of this examination is to evaluate the surface contour of the globe and cornea to rule out conjunctival chemosis (swelling), hemorrhage, emphysema, and foreign bodies. The anterior chamber should be evaluated for depth, clarity, and hyphema (blood in the anterior chamber). Hyphema, if found, should be evaluated by an ophthalmologist so that surgical evacuation or medical management may be instituted in an effort to avoid occlusion of the trabecular meshwork, which may lead to glaucoma or a fixed iris.

The iris's shape and reactivity should also be noted. If a corneal abrasion or laceration is suspected, this may be more thoroughly evaluated with fluorescein dye and a Wood's lamp (cobalt blue light). The fluorescein dye pools in the laceration or abrasion and fluoresces with a bright lime-green hue under the lamplight (Fig. 24.6).

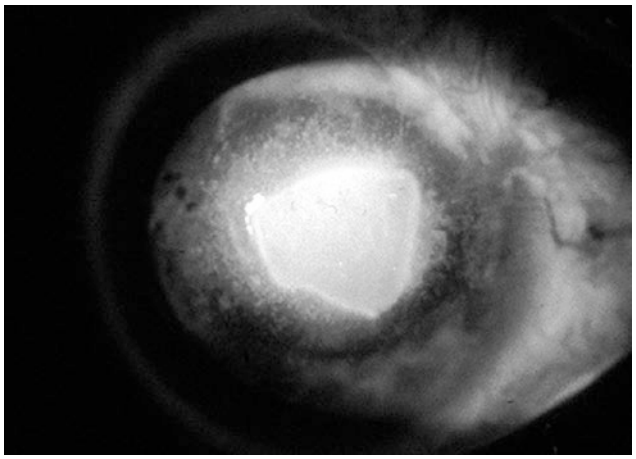


Fig. 24.6 A broad corneal abrasion of the right eye illustrated with the pooled fluorescein dye under a cobalt blue (Wood's) lamp

Finally, the bony orbital rim should be palpated for steps, crepitus, and mobility. The patient should be queried about altered or lack of sensation, and neurosensory testing should be performed to evaluate the supraorbital, supratrochlear, and infraorbital nerves.

24.5 Imaging

Once a complete ophthalmologic and facial examination has been performed, selected studies such as CT or magnetic resonance imaging (MRI) can be ordered with defined parameters to provide meaningful results. Imaging is essential for the proper diagnosis and treatment of orbital trauma. Noncontrasted CT is the primary imaging modality currently used for evaluating injuries from blunt or penetrating trauma, as well as for localizing most orbital foreign bodies [15]. Other imaging modalities, such as plain radiography, reconstructed three-dimensional CT, MRI, ophthalmic ultrasonography, color Doppler imaging, and angiography, may provide necessary additional information in select instances. CT scans have become the standard of care in evaluating acute orbital injuries. Standard radiography is a readily available and inexpensive method for primary evaluations of orbital fractures. Plain radiography, however, is inadequate when used in evaluating internal orbital fractures, and it is difficult to localize foreign bodies with plain films alone.

If plain films reveal an internal orbital fracture that possibly warrants surgical intervention, then CT scans should be obtained. The fracture can then be fully evaluated for surgical treatment planning. CT allows excellent visualization of orbital soft tissues and permits one to simultaneously assess the cranial vault and brain. The standard imaging approach for facial trauma is to obtain direct (non-reformatted) 3–5 mm sections in the axial and coronal planes. Intravenous contrast offers no advantages to the evaluation of acute bony facial injuries. . A dedicated orbital CT provides 1–1.5 mm axial cuts. The axial images with fine detail (1 mm slices) must be obtained to allow for meaningful reformatted image quality. If an optic canal fracture is suspected, then 1–1.5 mm axial cuts should be obtained [16, 17]. This allows a better determination and correlation of any afferent visual defect owing to possible bony impingement. Newer helical CT scanning techniques now allow for reformatting that is very precise. Reformatted views in the sagittal plane allows for better visualization of the anteroposterior extent of the orbital floor defect that may sometimes be missed or not as well appreciated on coronal or axial views (Fig. 24.7).

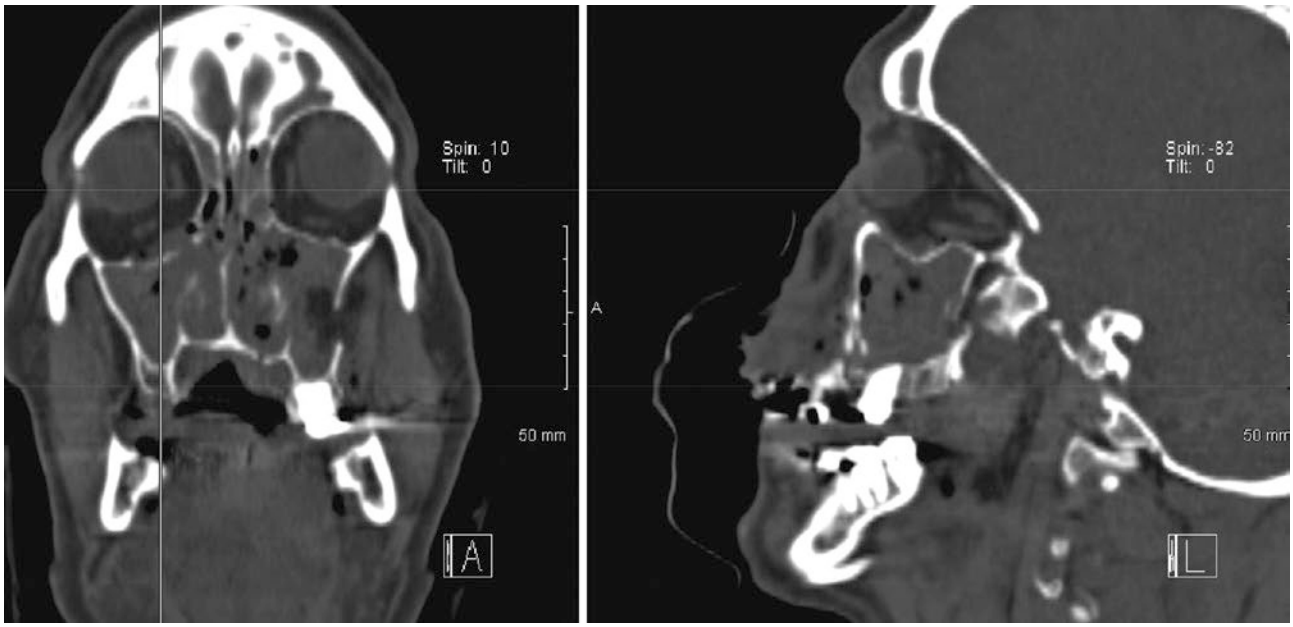


Fig. 24.7 Reformatted views in the sagittal plane allow for better visualization of the anteroposterior extent of the orbital floor defect that may sometimes be missed or not as well appreciated on coronal or axial views

Although MRI is generally accepted as a superior soft tissue imaging modality, CT scans adequately assess lens dislocation, vitreous hemorrhage, ruptured globe, retrobulbar hemorrhage, or avulsion of the optic nerve. CT is the imaging of choice in localizing metallic and most nonmetallic foreign bodies in relation to the globe, muscular cone (the area inside the extraocular muscles), and the optic nerve [15, 18]. The location and extent of any subperiosteal hematoma formation, with possible mass effects, can also be adequately assessed with CT imaging. Computer-generated three-dimensional CT imaging can provide superior views and spatial orientation of fragments for complex orbital and facial fractures. In the majority of acute facial fractures, three-dimensional CT scanning is unnecessary. However, with complex facial trauma with severe displacement, or for secondary reconstruction, three-dimensional CT scanning is invaluable for surgical treatment planning [19]. Generally, 1–1.5 mm fine axial cuts are obtained; the patient must remain motionless for the entire scan, which may include more than 100 slices.

Obtaining CT scans in a trauma victim may have some technical difficulties. Sedation may be warranted in pediatric or uncooperative trauma patients. However, with facial bleeding, possible concomitant mandible fractures, or obtundation from alcohol or street-drug use, a secure airway must be maintained throughout the radiology procedure. This may require endotracheal intubation. CT scans may fail to reveal radiolucent foreign bodies such as wood or vegetative matter [20]. In these instances ultrasonography and MRI are most useful in detecting the radiolucent foreign body and localizing it. These studies should be obtained when the CT scans are equivocal or when physical examination suggests the presence of foreign bodies.

MRI can be useful in the setting of orbital trauma to assess soft tissue injury or entrapment of extraocular muscles in the area of the orbital suspensory framework. Standard radiographs or CT scans should be obtained before MRI is performed on patients with suspected intraocular or intraorbital ferromagnetic bodies because of the potential for displacement of the metallic fragments, resulting in further significant ocular or brain injury [21, 22]. With CT imaging, wood can appear isodense with fat or mimic intraorbital air. If the history or clinical examination indicates that fragments of wood may have penetrated the orbit or globe, then an MRI should be ordered. An MRI should also be performed when an apparent orbital emphysema (focal air collection) fails to resorb rapidly (within several days); this may suggest a space-occupying foreign body [23].

Ophthalmic ultrasonography is seldom used but is a readily available, safe, inexpensive, and noninvasive imaging modality [24]. Foreign bodies located in the orbit can be identified with ultrasonography but are much more difficult to detect when located in the orbital apex owing to signal reflection. Wood and other radiolucent materials can be detected with ultrasonography [25]. Color Doppler imaging is an ultrasound technique that provides simultaneous two-dimensional images and visualization of blood flow [26]. It can be useful in evaluating a post-traumatic high-flow carotid cavernous fis-

tula. However, angiography remains the study of choice for definitively establishing this diagnosis.

With the advent of cone beam CT scanners in offices and outpatient areas, this is becoming a useful entity. The images of a cone beam CT of the orbit however miss much detail especially of the soft tissue but could be a useful adjunct in intraoperative navigation assisted reconstruction of the bony structures of the orbit [27].

24.6 Ocular Injuries and Disturbances

Patients who sustain midfacial trauma, particularly in motor vehicle accidents, often have concomitant neurologic and multisystem injuries. A neurologically impaired or uncooperative patient presents additional challenges in performing an adequate orbital and ophthalmologic examination. It is paramount that the primary tenets of advanced trauma life support be adhered to in securing the airway and protecting the cervical spine. When orbital fractures caused by severe blunt force trauma are detected, additional associated injuries should be sought, such as orbital canal or apex involvement, retrobulbar hematoma, or globe perforation. When there are multiple midface fractures, such as those of the ZMC, NOE, and frontal sinus, and Le Fort II or Le Fort III fractures, then more severe intraorbital injury, bleeding, and globe perforation are likely. Basilar skull fractures, as evidenced by clinical signs such as CSF otorrhea or rhinorrhea, Battle's sign, or CT evidence such as fracture lines or intracranial air, are generally caused by high-velocity impact and are often associated with severe neurologic injury.

Superior orbital fissure syndrome is characterized by impairment of cranial nerves III, IV, V, and VI secondary to compression by a fractured bony segment or hematoma formation in the region. Orbital apex syndrome has all the hallmarks of superior orbital fissure syndrome, with the addition of optic nerve (cranial nerve II) injury. Between 0.6 and 4% of patients suffering orbital fractures have a globe injury or optic nerve impairment, resulting in a significant or total loss of vision in one eye [28, 29]. This fact highlights the need for a thorough initial ophthalmologic and visual acuity assessment, with follow-up serial examinations as indicated.

24.6.1 Visual Impairment

Visual impairment or total vision loss can occur at various levels along the optic pathway. Direct injury or forces transmitted to the globe by displaced fracture segments can result in retrobulbar hematoma, globe rupture, hyphema, lens displacement, vitreous hemorrhage, retinal detachment, and optic nerve injury. Patients with orbital fractures and any degree of visual impairment who complain of severe ocular pain should be evaluated for retrobulbar hematoma. It is often the "less impressive" orbital fracture that leads to retrobulbar hematoma formation (◘ Fig. 24.8). This is due to bleeding within a relatively closed compartment and the lack of a potential drainage pathway through paranasal sinuses, such as the ethmoids or maxillary sinus. In essence, there is a compartment syndrome resulting from an elevation of intraorbital pressure, which leads to central retinal artery compression, or ischemia of the optic nerve. The increased intraorbital pressures can secondarily raise the intraocular pressure, which, in turn, compromises the ocular blood supply [30–32]. In most instances requiring emergent treatment, there is a degree of exophthalmos and excessive tension of the lids. Although CT scanning to confirm the diagnosis is desirable, there should not be unnecessary delay in the surgical management. The immediate or urgent surgical management for retrobulbar hematoma evacuation consists of a lateral canthotomy, with or without inferior cantholysis, and disinsertion of the septum along the lower eyelid in a medial direction. A small Penrose drain is left in place for 24–48 hours to ensure adequate drainage and to prevent reaccumulation. Additional maneuvers to lower the intraocular pressure include administration of intravenous mannitol or acetazolamide or application of various glaucoma medications. Typically, blow-in fractures or inward rotation of the ZMC does not result in increased intraorbital or intraocular pressures with visual impairment. This is most likely due to pressure relief and volume expansion provided by additional orbital wall fractures such as the medial wall into the ethmoid or the floor sagging into the maxillary sinus or a large ZMC component to the fracture complex (◘ Fig. 24.9).

◘ **Fig. 24.8** Axial computed tomography scan of a right retrobulbar hematoma. This diffuse infiltrative pattern is characteristic, whereas the discreet clot mass is less common

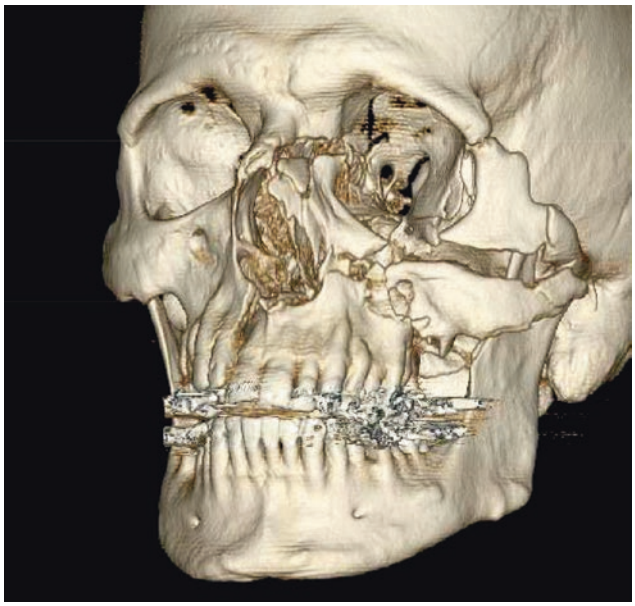


Fig. 24.9 Accompanying medial orbital fractures as well as ZMC fractures can cause significant expansion of the orbital volume

A penetrating globe injury can result from what appears to be an innocuous small laceration or from horrific blunt-force trauma. When an eyelid laceration is accompanied by an asymmetric pupil, without a prior history of surgery, then a globe perforation likely exists (Fig. 24.10). Blunt trauma can lead to globe perforation owing to a scleral rupture from the sudden instantaneous increased intraocular pressure. The most common site for scleral rupture is at the site of previous cataract surgery, at the limbus, or just posterior to the insertion of the rectus muscles onto the globe, which is 5–7 mm from the edge of the limbus. The area under the muscle insertion is anatomically the weakest and thinnest portion of the sclera. With suspected globe perforation, pupillary dilatation and inspection by an ophthalmologist is mandatory. The inspection may be difficult—the injury may not be visible on fundoscopic examination since it is anterior to the equator of the globe and externally may be hidden underneath the rectus muscle insertion. Detection and surgical access for repair may require dissection of the bulbar conjunctiva with retraction of the extraocular muscles and external globe inspection. The penetrating injuries should be treated emergently, or within 12 hours, to decrease the risk of infection or ocular content herniation. The ultimate visual outcome directly correlates with the presenting visual acuity. Few eyes that cannot detect hand motions or have no light perception (NLP) regain useful vision. Globe injuries should be addressed before any facial lacerations are repaired. The exception is significant active blood loss from a severed vessel.

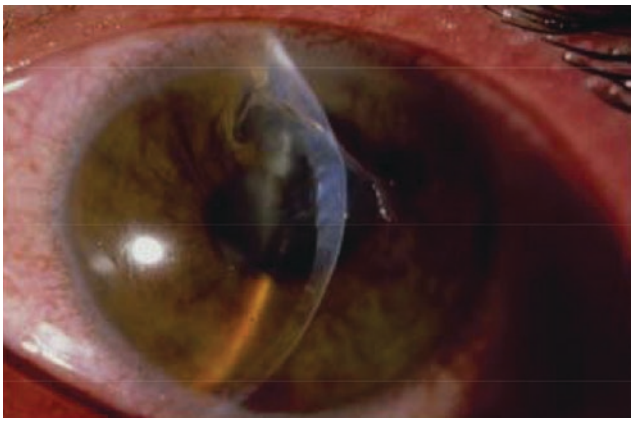


Fig. 24.10 A full-thickness corneal laceration and an irregular pupil of the right eye is seen during a slit-lamp examination

Sympathetic Ophthalmia Sympathetic ophthalmia is a rare, bilateral granulomatous uveitis that occurs after either surgical or accidental trauma to one eye. It occurs in less than 0.2% of penetrating globe injuries. The ocular inflammation in the normal eye becomes apparent usually within 3 months after injury, often much earlier. Clinical presentation is an insidious or acute anterior uveitis with gradual decreasing visual acuity in the contralateral uninjured globe Sympathetic ophthalmia is thought to represent an autoimmune inflammatory response, mediated by T-cells, against choroidal melanocytes, which are structurally shielded from the peripheral circulation until injury "exposes them". Diagnosis is based on clinical findings and a history of previous ocular trauma or surgery. Treatment of sympathetic ophthalmia consists of systemic anti-inflammatory agents or immunosuppression. The role of enucleation after the diagnosis of sympathetic ophthalmia remains controversial. Visual prognosis is reasonably good with prompt wound repair and appropriate immunomodulatory therapy [33].

Hyphema is blood in the anterior chamber of the eye. It can be as severe as a complete obliteration of the anterior chamber, termed "eight-ball hyphema," or more commonly a thin 1–2 mm layering at the inferior margin in the upright position (Fig. 24.11). Some hyphemas are termed microhyphemas, with red blood cells floating in the anterior chamber and not layering out. The level and severity of the hyphema should be noted and recorded. The bleeding is from the rupture of an iris or ciliary body vessel and usually is the result of blunt trauma. Patients often complain of eye pain and, occasionally, visual loss if the amount of bleeding is severe. Medical management of hyphema is aimed at preventing rebleeding and venous congestion and promoting clearance of the existing blood. This may include hospitalization, bed rest, head-of-bed elevation, and longer-

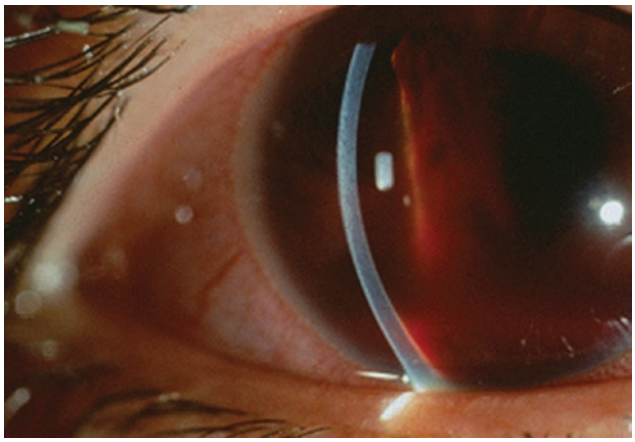


Fig. 24.11 This partial hyphema of the right eye resulted from a punch to the face; a computed tomography scan showed a minimally displaced orbital floor fracture. The slit-lamp examination shows early layering. This patient received nonoperative management

acting cycloplegics (topical agents such as scopolamine or atropine). Cycloplegics maintain a dilated pupil and thus immobilization of the iris, which discourages further rebleeding. Topical steroids may be administered to decrease further rebleeding and reduce intraocular inflammation. Oral aminocaproic acid is an antifibrinolytic recommended to reduce the incidence of rebleeding into the anterior chamber. In moderate to severe cases there should be daily monitoring of intraocular pressures and control of any high pressure increases with intravenous carbonic anhydrase inhibitors (acetazolamide, which limits aqueous humor production) or hyperosmotics (mannitol). With severe hyphema, intraocular surgery to irrigate, aspirate, and evacuate the clot may be necessary to prevent optic atrophy owing to elevated pressures, or to avoid permanent corneal blood staining [34]. The anterior chamber washout is the most commonly performed procedure for this purpose.

Vitreous hemorrhage can result from blunt trauma with the rupture of ciliary, retinal, or choroidal vessels. If, during fundoscopic examination, the retina cannot be visualized despite a normal-appearing anterior chamber and lens, vitreous hemorrhage is most likely present. An early loss of red reflex is also an indication of vitreous hemorrhage. As with hyphema, initial management typically involves hospitalization, bed rest with head-of-bed elevation, and serial clinical examinations. Vitreous hemorrhage is slow to resolve, and it may take months for this to clear, with symptomatic visual improvement [35]. A vitrectomy may be required after 6 months if satisfactory resorption has not occurred.

Lens dislocation may be detected by fundoscopic or slit-lamp examination. The lens, in its normal anatomic position, physically separates the anterior and posterior chambers, but it can be dislocated either partially or totally into either one. Symptoms include monocular diplopia and blurred vision; thus, it is important to check each eye's visual acuity independently. Posterior dislocation may be well tolerated; however, complete anterior dislocation can result in glaucoma and usually requires emergency extraction of the lens.

Rhegmatogenous retinal detachment (RRD) and peripheral tears result from blunt force trauma. Characteristic symptoms include flashing lights and a field loss best described as a curtain or window shade coming over the eye. On fundoscopic examination, the retina may not be clearly visualized, or undulations may be present. Retinal detachments require surgery [35]. An emergency consultation with an ophthalmologist and initial maneuvers should be instituted. Maneuvers involve bed rest in a head-up position and assurance that there is no Valsalva-type exertion; these prevent further extension of the detachment. Operative management may include any or all of the following: a scleral buckle, cryotherapy a vitrectomy, or endolaser. In-office pneumatic retinopexy works well with superior detachments: an inert expandable gas is injected into the vitreous and indirect laser treatment is applied. Retinal ischemia also presents itself similar to a retinal detachment. Should a fundoscopic exam rule out detachment, a high index of suspicion should be maintained for venous or arterial occlusion from micro-emboli causing retinal ischemia and the patient should be worked up to find causes of the same. Orbital ischemic syndrome may be a delayed manifestation of carotid dissection and precede cerebral hypotension in trauma patients [36].

Optic nerve injury or compromise can result from orbital fractures in the posterior region or optic canal. It is characterized by decreased visual acuity, diminished color vision, and a relative afferent pupillary defect. It is possible to retain very good vision and yet still have an optic nerve injury manifested by color deficits, afferent papillary defect, and visual field loss. Detection of early subtle changes requires that a cooperative patient undergoes visual acuity testing, consisting of testing with a Snellen chart, finger counting, detection of motion, or light perception. Patients may present with NLP, which mandates an emergency consultation with an ophthalmologist and a fine axial CT imaging of the orbital apex. If NLP persists >48 hours, then rarely does any meaningful vision return to the affected eye. Patients with NLP or severely decreased visual acuity may be suffering from traumatic optic neuropathy and should be given high-dose systemic methylprednisolone therapy for at least 48–72 hours (initial loading dose of 30 mg/kg IV methylprednisolone sodium succinate, followed by 15 mg/kg IV 2 h later and q6h thereafter) [37–39]. If the patient is uncooperative, heavily sedated, or unconscious, pupillary reaction can be monitored and fol-

lowed as a sensitive test of optic nerve (cranial nerve II) function. This is best achieved in a dimly lit room; a penlight is moved alternating from one eye to the other every 2–3 seconds, and the pupillary response is observed. In the swinging flashlight test, with the light shining into the normal eye, both pupils should exhibit a brisk constriction, this indicates intact direct and consensual reflex. If the light is then directed from the uninjured to the injured eye the pupil on the injured eye will dilate. This is indicative of an optic nerve injury (relative afferent pupillary defect or Marcus Gunn pupil). A unilateral, fixed, dilated pupil is usually due to an efferent pathway injury (cranial nerve III), or some form of intracranial injury or bleed, which is usually accompanied by other neurologic lateralizing signs. Early loss of monocular dyschromatopsia (impaired color vision) especially red desaturation that can be demonstrated using a "red card test" and may be a very early sign of optic neuritis after an ocular injury.

24.6.2 Diplopia

When a patient complains of seeing a double image of the same object, the examiner should first test each eye independently by covering the opposite eye to determine whether the diplopia is monocular or binocular. Monocular diplopia is usually due to lens dislocation or opacification, or another disturbance in the clear media along the visual axis. Acute binocular diplopia, secondary to trauma, derives from one of three basic mechanisms: edema or hematoma, restricted motility, or neurogenic injury. The most common cause of binocular diplopia following trauma is orbital edema and hematoma. This is usually found in peripheral fields of gaze, and, if other findings are absent, diplopia in the primary and downward gazes usually resolves along with the edema in 7–10 days. Slight diplopia in extreme peripheral fields of gaze may persist for months but is rarely problematic since individuals seldom require these extreme views for everyday function. Also the patient may complain that the phenomenon is transitory and that sudden looking "upward and outward" (superiorly and laterally, such as when looking in a rearview mirror) may cause instantaneous but brief diplopia. Binocular vision without diplopia is most important in the primary (straight-ahead) and downward fields of gaze. The majority of our daily activities, such as conversing, reading, and walking, use these visual fields. If diplopia persists, an ophthalmologic consultation should be sought. Systemic corticosteroids hasten the resolution of orbital edema and the resulting diplopia, which is fairly common following blunt trauma to the orbit.

Persistent post-traumatic diplopia is best evaluated by an ophthalmologist. It is important to establish an accurate diagnosis and precise etiology. The basic evaluation should include assessing the symmetry of the corneal light reflexes and testing of ductions (following a finger in all eight fields of gaze) including a selective forced duction. The forced duction helps distinguish between restricted motion from entrapment, scarring, or fibrotic contractures versus a neurogenic motility disorder (cranial nerves III, IV, or VI). Ophthalmologists use diplopic visual fields (see ◘ Fig. 24.4f) to quantify and categorize the diplopia; serial examinations allow accurate tracking of spontaneous recovery or postsurgical progress. In the acute setting, restrictive disorders are managed with early bony orbital surgery and reconstruction, whereas neurogenic disorders are managed with the injection of botulinum toxin into select extraocular muscles whose forces are unopposed by the injured or restricted muscles. Following bony orbital reconstruction or selective botulinum toxin injections, there should be a 6- to 12-month waiting period for the diplopia to stabilize. Then, any residual and stable diplopia can be addressed with strabismus (extraocular muscle) surgery. Strabismus surgery has two basic maneuvers: a repositioning of muscle insertions onto the sclera or a weakening of the opposing muscles. After a period of healing, selective botulinum toxin injections or more minor revision strabismus surgery may be required to fine-tune the result. The important point to stress is that a healed abnormal bony wall position or orbital volume changes, resulting in enophthalmos or vertical dystopia, typically do not cause stable significant diplopia. In fact, vertical dystopia of up to 1 cm can be accommodated by the brain and should not result in diplopia in the primary fields of gaze. Therefore, any bony wall revision or reconstruction should be performed to correct a cosmetic or other functional defect without promise of correction or improvement in any coexisting diplopia. These reconstruction procedures should be performed and allowed to heal, and the diplopia allowed to stabilize for 6 months prior to the strabismus surgery, which would address the diplopia.

In the trauma setting, diplopia may be due to restricted ocular motility from a prolapse of the periorbital contents into the medially fractured ethmoid air cells or underlying maxillary sinus. Such diplopia may also be due to entrapment or direct impingement on the fine suspensory ligamentous system of the orbit or, less frequently, of the extraocular muscles. Restricted motility or entrapment is commonly found with orbital floor and medial wall fractures, less frequently with roof fractures, and rarely with lateral wall fractures. Significant medial wall fractures are manifested primarily by enophthalmos owing to volume expansion.

When testing range of motion, if there is repeatedly a firm-fixed limited stop of unilateral eye motion, the eye should be anesthetized topically and a forced duction test performed. Occasionally the entrapment or incarceration of the supporting structures or muscles is mild, and during the forced duction, initial resistance may be encountered and then relieved. In such an instance, the positive forced duction test was both diagnostic and therapeutic. However, if the forced duction test is positive and mimics the voluntary active point of restricted motion, this should be correlated with CT scan findings (see ◘ Fig. 24.4) [40]. A repeatable fixed point of limitation is usually due to direct entrapment of the extraocular muscles or the capsulopalpebral fascia (fascia of Tenon). This is more common in linear floor trap door fractures than in comminuted multiple wall fractures. Patients with muscle or Tenon capsule incarceration confirmed by CT are candidates for urgent exploration and repair (within 12 h). Prolonged muscle entrapment with ischemia can lead to fibrosis (Volkmann's contracture) with permanent diplopia, despite surgical release of the entrapped tissues. When exploring these fractures, the entrapped fascia or muscle can be difficult to release. This classically occurs in the pediatric patient with an anteroposterior linear fracture of the orbital floor with no accompanying rim fracture. When an area of resistance is encountered initially and correlates to this same anatomic location on CT, then consideration should be given to inserting an instrument within the anterior fracture line and gently twisting or prying to open up the fracture, or taking a fine osteotome or instrument to fracture away a small adjacent strip of the orbital floor so that a thin blunt malleable retractor on either side of the entrapped area can gently lift and reduce the entrapped soft tissues back into the orbit. Direct grasping of the tissues and tugging to reduce them back into the orbit may result in further contusion and injury.

Diplopia can be due to a central ophthalmoplegia owing to impairment of cranial nerves III, IV, or VI. The fourth nerve is the most commonly injured at the point where it passes over the petrous ridge of the temporal bone. This results in vertical diplopia and a compensatory head tilt to the opposite shoulder. These nerves have fairly long intracranial tracts and can be injured by direct skull fractures or be compressed by intracranial bleeds or diffuse cerebral edema after blunt head trauma. Cranial nerve palsies often spontaneously recover within 6–9 months. Recovery is quite variable and is dependent on severity and the type of injury.

24.6.3 Posttraumatic Enophthalmos

Posterior and inferior displacement of the globe and dystopia can result from fractures that increase the orbital volume. Generally, a 1 cm^3 increase in orbital volume results in 0.8 mm of enophthalmos. Patients with clinically noticeable enophthalmos can present with deepening of the supratarsal fold, decreased anterior globe projection compared to the uninjured one, pseudoptosis of the upper lid, shortening of the horizontal dimension of the palpebral fissure and a decrease in the canthal angles. Prolapse and herniation of the soft tissues through fractured orbital walls, fat atrophy and scarring all contribute to this clinical picture. Usually enophthalmos is a late sequalae that may be initially masked by the intraorbital tissue swelling and hematoma. Reconstructing the fractured orbital walls to restore the orbital volume and repositioning the prolapsed orbital tissues is one of the key elements in the decision making of pursuing surgery to restore the anterior globe projection and avoid the posttraumatic unaesthetic outcome. Chronic loss of periorbital fat and scarring is harder to manage. While one may be able to reconstruct the lost bony orbital volume successfully, it is harder to recreate the lost orbital soft-tissue loss. Intraorbital hyaluronic acid and autogenous fat injections may also be used to restore the orbital volume [41].

24.6.4 Oculocardiac Reflex

Oculocardiac reflex is bradycardia in response to manual compression of the eye. The afferent limb of the reflex is carried through the ophthalmic division of the trigeminal nerve to the main sensory nucleus. The efferent pathway is carried through the vagus nerve to depressor nerve fibers in the myocardium. An abrupt heart rate greater than 10% decrease is considered diagnostic. This phenomenon is often described in pediatric trapdoor orbital trauma. Surgeons and anesthesiologists should be aware to limit overzealous pressure on the globe during management [42].

24.6.5 Eyelid Lacerations

Eyelid lacerations, particularly those extending to the lid margin and gray line, should be thoroughly evaluated for lacrimal drainage system injury, canthal tendon disruption, or injury to the tarsal plate and levator apo-

neurosis. After antibiotics and tetanus prophylaxis have been administered as necessary, the wound should be cleansed and débrided, taking care to protect the globe, possibly with a contact lens. The eyelid laceration should be repaired in a layered fashion, starting with the tarsal plate repair (with 6–0 polyglycolic acid), lid margin (two to three interrupted sutures with 6–0 silk, which is nonirritating to the cornea), orbicularis muscle reapposition (multiple 6–0 plain gut sutures), and finally skin (with 6–0 nylon or 6–0 fast-absorbing gut). Topical ophthalmic ointment should be prescribed since these agents come in contact with the globe frequently, and sutures should be removed in 5 or 6 days. Patients should be followed up and monitored for potential complications such as scar contracture or lid notching. Several weeks post-repair, if significant lid contracture or focal thickening is noted, then selective judicious steroid injections (triamcinolone acetonide, 40 mg/mL) can be administered with accompanying daily massage by the patient.

Avulsion or loss of eyelid soft tissue is rare. When this occurs, it is usually from an abrasive crushing macerated-type laceration sustained in such accidents as a rollover in an all-terrain vehicle or ejection from a motor vehicle. In evaluating these injuries, the examiner should moisten the rolled edges of the laceration and attempt to gently realign them. One should not abnormally align the tissues, borrowing them from the periphery and shortening them in the vertical dimension. This can result in lid retraction or lagophthalmos, with risks of corneal exposure and ulceration. It is best either to leave a small amount of denuded underlying tissues, which will reepithelialize secondarily, and possibly perform a temporary tarsorrhaphy, or, for larger defects, to harvest a thin defatted skin graft for primary reconstruction (◘ Fig. 24.12).

◘ **Fig. 24.12** **a** This young male sustained a macerated forehead, and eyelid and nasal lacerations after being ejected from a motor vehicle in an accident. **b** After moistening, redraping, and suturing the soft tissues, it was apparent that there was a significant defect (8 × 10 mm) of skin on the right upper lid. **c** A full-thickness skin graft was harvested from the right posterior auricular area, which was closed primarily with the aid of releasing Z-plasty incisions. **d** The undersurface (dermal side) of the graft was thinned and defatted. **e** The graft was first perforated and inset over the skin defect. A temporary tarsorrhaphy was maintained for 1 week to minimize motion and shearing forces. **f** Facial appearance 3 months after repair. **g** Passive lid patency was achieved. There was no further revision surgery

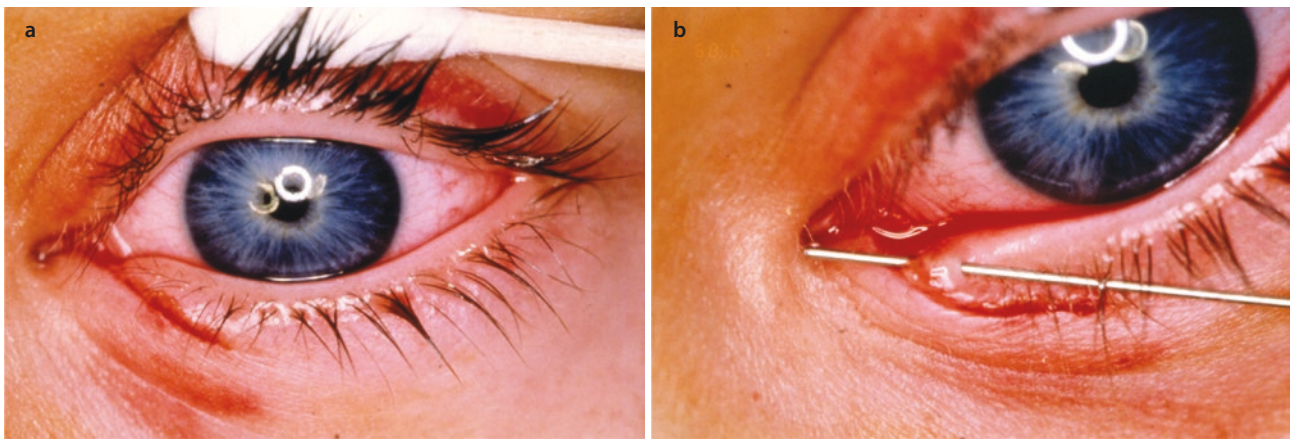
24.6.6 Lacrimal Injuries

Injuries to the lacrimal drainage system most often result from direct eyelid lacerations at the medial edge of the lid, which traverse the lid margin and disrupt the inferior canaliculus. Canalicular lacerations also occur indirectly when strong forces are applied to the lateral aspect of the lids. This tension directed laterally causes the eyelid to split at the weakest point, which is just medial to the punctum (◘ Fig. 24.13). Damage to the lacrimal drainage system can also be seen with severe medial rim and orbital wall fractures. A disruption in the lacrimal system can be detected by passing a lacrimal probe through the punctum and visualizing the blunt-tipped probe within the laceration or wound. It is especially important to detect this with the inferior canaliculus since this system is dominant in the vast majority of patients.

Repair involves reanastomosis of the canaliculus and either mono- or bicanalicular intubation. With bicanalicular intubation, the repair is performed by passing a silicone intubation tube through the puncta into the laceration and then locating the distal cut end of the drainage system for passing the tube into the nose, which is retrieved with a hook beneath the inferior turbinate. Typically both the superior and inferior canaliculi are intubated (usually one is uninjured); both silicone tubes are passed into the nose and are tied to each other. This allows for retention of the looped tube for 6–12 weeks. Intraoperatively, the silicone tubes are stretched toward the external nares, tied together, and typically oversewn or tied with a fine silk suture to allow for long-term retention. If no tension is applied to the cut ends of the silicone tubing while tying, then, postoperatively, the loop formed at the canaliculi puncta will migrate laterally toward the cornea, causing irritation or an annoying visual field disturbance.

24.6.7 Telecanthus

Traumatic telecanthus typically results from severe midfacial trauma (NOE) with displacement and splaying of the bones that serve as attachments for the medial canthal tendons. It is less frequently due to laceration and actual physical disruption and disinsertion of the canthal tendons from the underlying bone. Therefore, traumatic telecanthus from these injuries is best treated early (within 7–10 d) following injury to prevent scarring and secondary maladaptive changes that compromise the reestablishment of the more normal narrow intercanthal distance. Preoperatively, one should determine whether the increased intercanthal distance is due to either a unilateral or a bilateral injury. Treatment typically includes an approach via a coronal incision, a Lynch (lateral nasal) approach, or a combination, with



◘ **Fig. 24.13** **a** Innocuous-appearing small left lower medial lid laceration sustained from an incidental grab along the cheek during a touch football game. **b** Slight lateral traction on the lower lid and probing of the inferior punctum revealed a full-thickness lid laceration medial to the punctum with severance of the inferior canaliculus. An oculoplastic surgeon repaired and managed this injury within 8 hours

reduction and fixation of the displaced bones or direct transnasal wiring. External splinting rarely yields satisfactory results.

An effective technique to reattach direct canthal tendon with transnasal wire fixation is by passing a doubled-end loop of 30-gauge stainless steel wire transnasally from the contralateral medial orbital wall and then suturing the medial canthus to the wire loop. The wire is then drawn to the opposite side by gradually twisting the two ends around a short section of titanium microplate situated in the opposite medial orbital wall (Fig. 24.14).

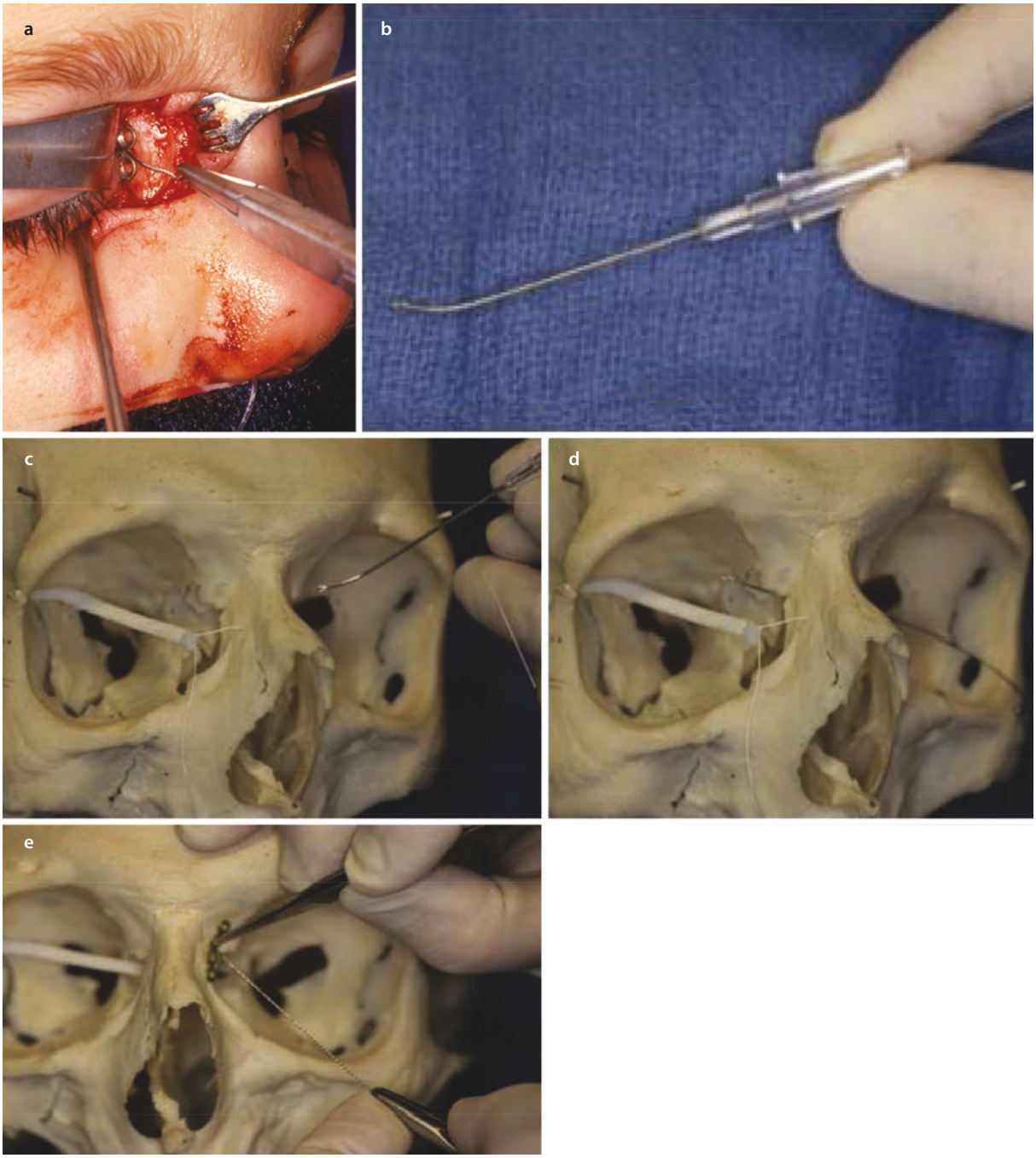


Fig. 24.14 **a** Reattachment and repositioning of the left medial canthus is fine-tuned by twist ing a 30-gauge wire over a section of microplate situated along the right medial orbital wall just behind and above the posterior lacrimal crest. **b** Use a 30 guage needdle to help pass this wire around plate. **c** The looped 30 gauge wire is placed transnasally, drilling previously through the septum and uninjured side. **d** The half-round needle that has sutured the medial canthal tissues is then passed through the wire loop and tied. **e** The free wire limbs are then passed on either side of a microplate and then twisted while placing tension so as to draw the medial canthus inward and "fine tune" it's position

24.7 Nonoperative Management of Orbital Fractures

Indications for nonoperative or, as it has previously been termed, *conservative* management of orbital fractures has been controversial for many years. Some historic perspective and review are warranted since it provides insight into the evolution and current thinking regarding nonoperative orbital fracture treatment. In 1957 Smith and Regan coined the term *blow-out fracture* and advocated early surgical intervention for orbital floor fracture repair [43]. Following this, Converse and Smith endorsed surgical exploration and repair of all orbital fractures within the first 3 weeks of injury [44]. Even with surgical exploration and repair, they found that enophthalmos or functional difficulties would develop, and they attributed this to the blunt trauma forces and tissue damage rather than the surgical intervention. Crikelair and colleagues in 1972 promoted the concept that orbital floor fractures were overdiagnosed on plain films and, thus, were over-operated [45]. They introduced the concept of repairing only select orbital floor fractures, which were confirmed by tomography and only if diplopia or enophthalmos persisted after an observational period of 2 weeks. This marked an important change in thinking toward a more selective approach for surgical intervention of orbital floor fractures. This change was, in part, prompted by reports and articles documenting unacceptable complications such as a total loss of vision following surgical exploration of asymptomatic floor fractures [46]. In 1974 Putterman and colleagues reported on a series of 57 patients whom they had observed and on whom they had performed no surgical intervention whatsoever [47]. Only a few of these individuals had any persistent diplopia, and there were no visual acuity disturbances 4 months following the trauma. This landmark article created a drastic shift in thinking—nonsurgical treatment of all orbital fractures was advocated. Putterman and colleagues proposed that patients with persistent diplopia should be managed by contralateral eye muscle surgery, or contralateral fat resection, to mask the enophthalmos or altered visual access of the injured side. Although this retrospective study and series of patients received much criticism from both the ophthalmology and facial trauma specialties, it did reveal that many orbital floor fractures healed uneventfully without surgical intervention and with the performance of eye-movement exercises.

Following Putterman and colleagues' report were a series of articles by various practitioners who attempted to refine and delineate the indications for surgical exploration and repair of orbital floor fractures. Dulley and Fells reported that only 50% of all patients with orbital floor fractures required surgical intervention [48]. All patients underwent a 2-week observational period; an individual would then undergo surgical intervention if one of the following criteria was present: enophthalmos >3 mm, large herniation of tissue into the antrum, entrapment with limited upward gaze, or significant diplopia. Nevertheless, these criteria were somewhat subjective and were limited by the current imaging techniques. Crumley and colleagues used similar indications for surgery to those of the Putterman group, but based on these criteria, almost 90% of all their patients with orbital floor fractures underwent surgical repair [49]. Converse and Smith developed and further refined these same indications for orbital floor surgery and reinforced the need and importance of serial clinical examinations in patients who had shown no initial indications for surgery [50]. This group promoted the concept that serial examinations revealing the development of enophthalmos should be the criterion for surgical intervention and not simply that a large or comminuted floor fracture existed. They proposed that the development of significant postinjury enophthalmos is variable and could be due to either resolving hemorrhage and edema or orbital fat atrophy. In 1982 a survey by the American Society of Ophthalmic, Plastic and Reconstructive Surgery revealed that two-thirds of oculo-plastic surgeons were operating within 2 weeks of injury with few serious complications or sequelae [51]. Although this was reassuring that the current surgical approaches and techniques were safe, there was no inquiry into what the criteria or determinates were for undertaking surgical repair.

What was helpful was that several ensuing studies began to delineate which patients exhibiting functional deficits might benefit from surgical exploration as opposed to observation. Koorneef, in an anatomic study, showed that fine connective tissue septa surrounded the extraocular muscles [52]. He advocated eye movement exercises in patients with mild or moderate restrictive motility as long as there was demonstrated serial improvement in motility. He purported that edema, hemorrhage, and connective tissue entrapment were responsible for the majority of limited motility in patients with orbital floor injuries.

In 1984 Smith and colleagues introduced the concept that Volkmann's contracture might occur as a result of elevated intraorbital compartment pressures [53]. Although this phenomenon was well-known, documented, and proven in the orthopedic literature to occur with extremities, it was unproven to occur in the orbit. Volkmann's contracture is a paresis from muscle shortening and fibrosis that results in limited mobility. Applying this concept to the orbit, Smith and colleagues recommended surgical intervention in the elderly, in individuals who are hypotensive, and for small or linear

orbital floor fractures with coexisting diplopia. They felt that these situations left patients at an increased risk for orbital compartment syndrome, thus developing permanent limited mobility owing to Volkmann-like contractures. Concurrent with these theories and recommendations was the report by Hawes and Dortzbach that emphasized the need for surgical repair within 2 weeks following injury in patients with persistent diplopia within a 30° range of the primary visual (straight-ahead) gaze [54]. They based this on their findings that there were poor results when late repairs were performed in this patient group.

Clearly the advent and ready availability of CT for use in diagnosing "trapdoor" fractures with mechanical impingement of the orbital structures helped to refine diagnostic capabilities and to aid treatment planning. Several groups of authors emphasized the need for correlating a positive forced duction test with CT evidence of incarceration or impingement [55, 56]. Without specific evidence of a trapdoor phenomenon or direct impingement, orbital floor fractures with limited motility were observed for 2 weeks. Persistent symptoms or findings then prompted surgical intervention. Trapdoor fractures or fine linear breaks without rim fractures are much more common in pediatric patients. When severe limitation of movement is encountered (typically upward or downward gaze, or both) and is correlated with CT findings, this is a true emergency that should be treated surgically to relieve the entrapment as soon as possible.

Since his initial controversial 1974 article, Putterman has revised his indications for surgical intervention [57]. Putterman and his colleagues' indications are now comparable to those of other surgeons. They advocate 7 days of systemic corticosteroids to speed the resolution of diplopia within the first 3 weeks. This may aid in resolving edema and helping determine who might benefit from surgery. Although persistent functional limitations are usually clear indications for surgery, controversy remains in treating those patients who demonstrate a steady but slow resolution of their diplopia that persists beyond 3 weeks.

When the surgeon is confronted with any orbital fracture, it is helpful to categorize the clinical deficits and goals of surgical treatment as being either functional or cosmetic. Simply operating on a radiographic finding because it exists is not satisfactory. The surgeon, with the assistance of his ophthalmology colleagues, should determine what, if any, functional deficits and cosmetic deformities exist. A specific anatomic reason for these should be sought. Then, if the magnitude of the functional deficit or cosmetic deformity warrants surgery, the type of surgical approach, repair, and materials should specifically address the structural causes. In a patient with the clinical findings of only "soft" indications for surgery, a 2-week observational period seems prudent. Several studies have addressed cosmetic deformities as they relate to orbital floor fractures, offering indications for surgery versus observation. Hawes and Dortzbach used tomography and felt that orbital floor fractures involving >50% of the surface area should be reconstructed within the first 2 weeks to avoid the predictable development of enophthalmos [54]. They also stated that patients with smaller orbital floor fractures but with >2 mm of enophthalmos present at 2 weeks postinjury should undergo orbital floor reconstruction. This recommendation is based on the fact that later repair is technically more difficult with less optimal outcomes owing to scar contracture and muscle shortening. Parsons and Mathog were able to demonstrate, using a laboratory model, that orbital floor fracture and displacement of equal magnitude with the medial wall fracture and displacement had a much greater effect on globe position [58]. This study supports the practice of most surgeons, which is nonsurgical and observational management of isolated displaced medial wall fractures.

When orbital fractures are associated with other facial fractures such as Le Fort or ZMC fractures, several authors have advocated orbital floor exploration and repair with any evidence of prolapse of the orbital contents into the sinus [59, 60]. In 1991 Putterman and colleagues advocated following patients closely for the development of enophthalmos, using objective measurement with a Hertel exophthalmometer, or serial measurements for vertical dystopia by aligning the top of a clear ruler to both undisturbed medial canthi and noting where the ruler bisects each eye [57]. Despite numerous reports, clinical series, and author suggestions, controversy still remains regarding the management of those patients who develop only mild enophthalmos or hypo-opthalmos (1–2 mm) without any functional deficits during the acute observational period.

24.8 Operative Management of Orbital Fractures

24.8.1 Indications

It is crucial that the surgeon has a complete understanding of the mechanism of injury and potential complications to make a full diagnosis and an appropriate treatment plan in each type of orbital fracture. Patients with a suspected or known orbital fracture should undergo a thorough clinical examination, including fundoscopic examination; visual acuity; pupillary reactivity; detection of diplopia, extraocular movement with any

limitations noted, enophthalmos, and vertical dystopia; forced duction testing; and recording of paresthesias. Radiographic studies should determine the full extent of the orbital fracture and any surrounding and associated facial fractures. CT scans, especially in the direct coronal plane, are the gold standard for use in orbital surgery treatment planning. Contraindications for surgery are hyphema, retinal tears, globe perforation, the patient sees only with the eye on the injured side, and life-threatening instability.

Indications for surgery can be divided into functional and cosmetic categories. A logical systematic approach is prudent in selecting patients who are suitable for acute or early surgical repair versus those who deserve an observational period with intervention when signs or symptoms warrant it (Fig. 24.15). With regard to function, diplopia and decreased visual acuity are the two main areas of concern. The majority of surgeons and articles in published literature support early surgical intervention in a patient with an orbital floor fracture that has mechanical restriction of gaze and a positive forced duction test with a CT scan that has a trapdoor appearance or suggestions of inferior rectus muscle incarceration [61, 62]. This phenomenon occurs more in children with linear fractures owing to the elasticity of their bones [63]. Pediatric or adult patients with these findings warrant early intervention to free up the tissues and hopefully prevent any permanent restriction owing to ischemic necrosis or scar contracture. In patients with less impressive restrictive motility (10–15°), a positive forced duction test, and no CT evidence of muscle entrapment, an observational period of several weeks is reasonable. These patients may only have entrapment of some of the fine connective tissue septa supporting the globe, and with routine daily function and/or eye exercises, this restriction typically steadily improves. Clinical follow-up with a series of examinations (two or three) within the first 14 days, steroid therapy, and eye movement exercises should optimize the outcome. In any patient with an orbital fracture that has persistent mechanical restriction or diplopia within 30° of their primary gaze, especially the downgaze (used during reading), surgical exploration is warranted. Prior to undertaking surgery, however, any neurogenic or central component should be ruled out. Although infrequently employed, electromyography can be used to

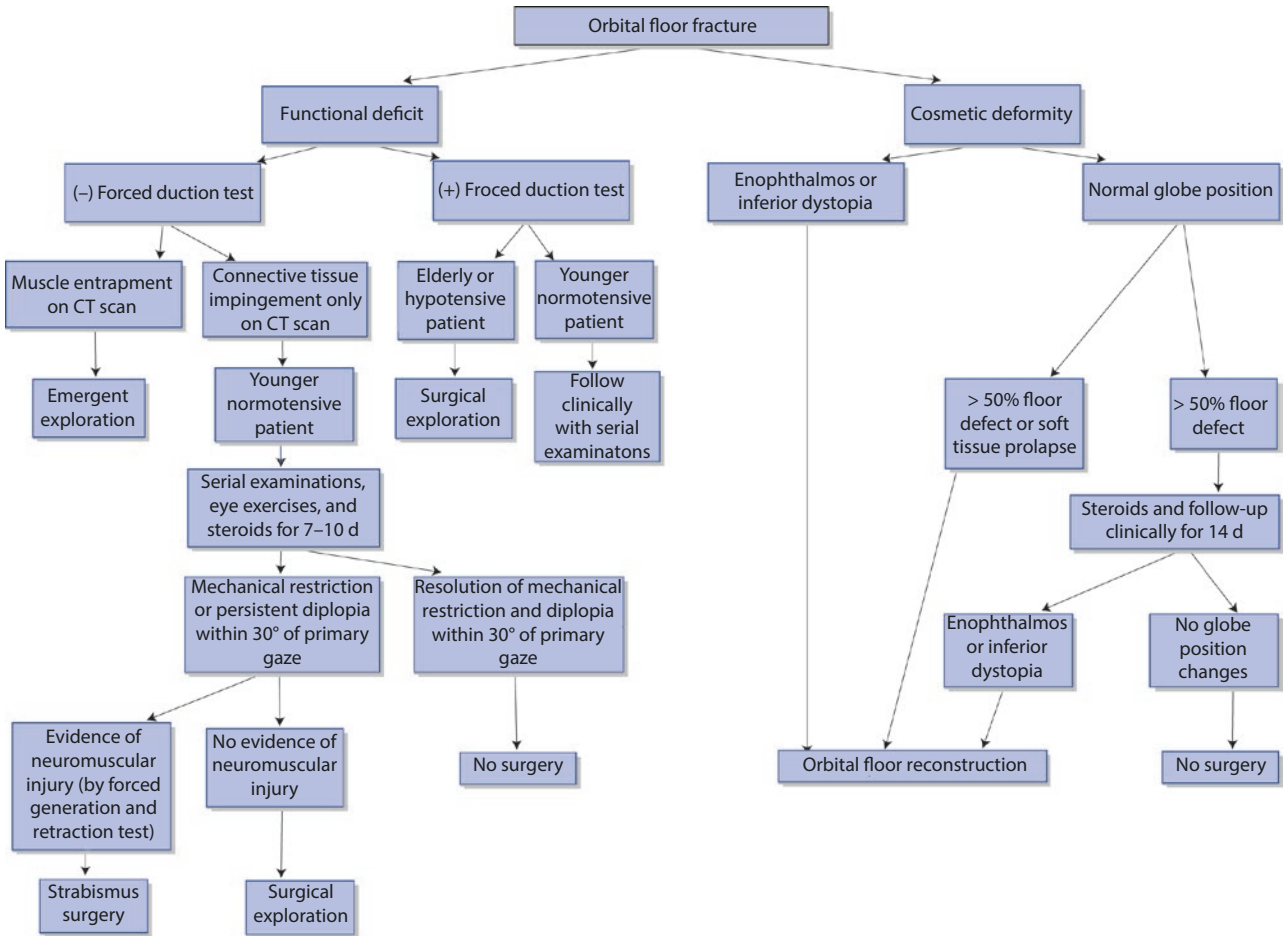


Fig. 24.15 Orbital floor fracture evaluation and treatment decision diagram. CT computed tomography, (–) negative, (+) positive

distinguish neurogenic diplopia from mechanical restriction in problematic or brain-injured patients. Neurogenic or neuromuscular injuries are more suitably treated by strabismus surgery. With regard to decreased visual acuity, an ophthalmologist should assess the patient serially for resolution or improvement. In more severe cases—patients who can only see shadows or figures or who have NLP—the fine-cut axial CT scans of the orbital apex and canal should be reviewed with the radiologist to determine whether there is bony mechanical impingement, hematoma, and/or edema compressing the optic nerve or vascular supply. With the increasing popularity of endoscopic approaches to the cranial base (typically for tumor removal), most major medical centers have neurosurgeons and/or otolaryngology head and neck specialists that are competent in performing transnasal endoscopic optic canal decompression. If at all possible, this should performed within 12 to 24 hours of the confirmed diagnosis of external optic nerve compression within the canal proper.

Cosmetic deformities such as enophthalmos or hypo-ophthalmos result from a bony orbital volume increase, extrusion of intraconal fat into extraconal spaces, or prolapse of orbital contents into the maxillary sinus or ethmoid air cells. Contrary to long-standing dogma, post-traumatic fat atrophy does not play a significant role in the development of these deformities [64]. Most surgeons currently undertake surgical intervention in orbital floor reconstruction if there is 2–3 mm or greater of enophthalmos or hypo-ophthalmos in the presence of orbital edema or hematoma. The rationale is that early repair offers the most favorable outcome and that the cosmetic deformity only worsens as the edema and hematoma resolve. Orbital floor defects of greater than half of the surface area with concomitant CT evidence of the disruption or prolapse into the underlining antrum generally should be repaired. Again, the rationale for this is that as the edema resolves, eventually there is some degree of enophthalmos or vertical dystopia that creates a cosmetically unacceptable or, less frequently, functional problem requiring surgery. With minimal floor disruption (<50%) and no entrapment or significant herniation, observation for 2 weeks is prudent. If the patient develops any functional problems or enophthalmos >2 mm, then surgery can be undertaken to treat the functional or cosmetic defect. Unnecessary delays approaching 6 weeks and beyond make the surgical repair more difficult and the ultimate outcome less desirable owing to scarring and muscle shortening.

24.8.2 Surgical Approaches

Once it has been determined a patient requires surgical intervention, a well-thought-out plan, and sequential approach should be developed. Of paramount importance is the determination of which of the anatomic areas need to be accessed with direct visualization and which intact bony edges or landmarks need to be found or fixated to accomplish the repair. This helps the surgeon determine which soft tissue incision should be employed. In general, most surgeons prefer to first grossly reduce and usually fixate all periorbital and facial fractures prior to accomplishing internal orbital repairs. The most commonly used surgical approaches and methods of reconstruction are presented here so that the surgeon can make an individualized and informed decision.

Inferior and Lateral Orbital Approaches There are three basic incisions used for accessing the orbital floor: the infraorbital, subciliary, and transconjunctival (Fig. 24.16). Although there are three basic approaches, there are numerous technical variations based on surgical training and individual preference. Clearly the subciliary and transconjunctival incisions are the most popular owing to their superior esthetics and generous access, and the fact that surgeons are familiar with their use. The infraorbital or rim incision results in compromised esthetics and offers no advantages over the two former approaches.

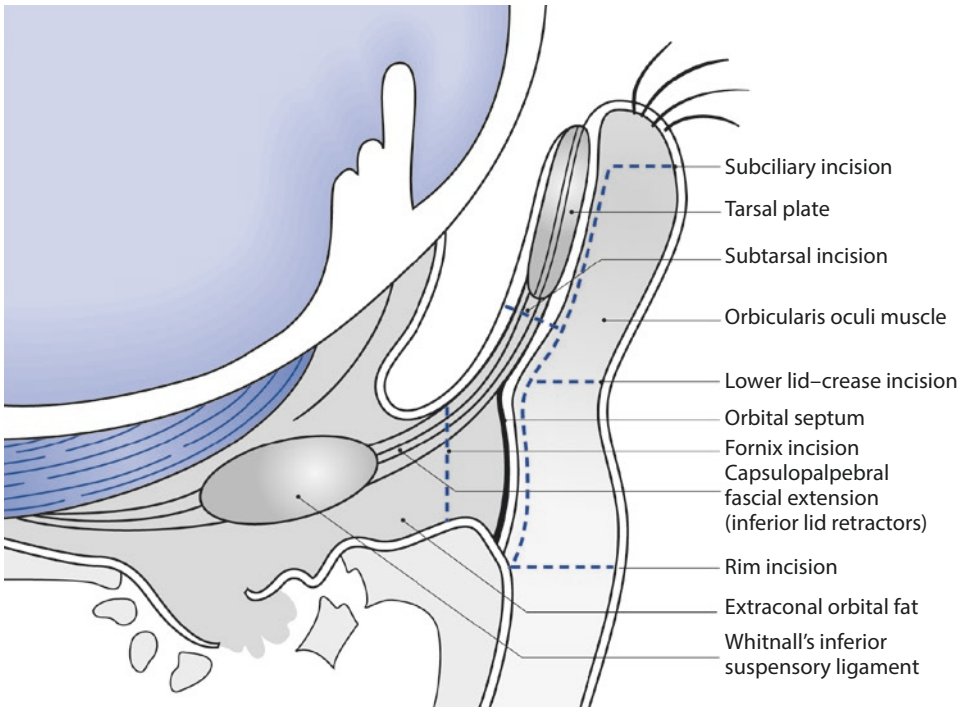


Fig. 24.16 Cross-sectional view of the inferior lid and various floor approach incisions. (Adapted from Ochs and Johns [97])

The subciliary incision was popularized by Converse in 1944 [65]. Typically a gently curved linear skin incision is made several millimeters below the lid edge or eyelash margin, preferably in a skin crease. The skin flap is then undermined in an inferior direction for several millimeters before traversing deeper inward directly through the orbicularis oculi muscle fibers and stopping when the orbital septum is encountered. The rationale for the division of the skin and muscle at different levels (stepping the incision lines) is that it helps to prevent direct or full-thickness scarring and tethering of the eyelid. Once the orbital septum has been encountered, the preseptal approach is then carried out inferiorly to the orbital rim, and the periosteum is incised just below the arcus marginalis. The periosteum of the orbital rim is then reflected upward and inward, and dissection is carried out over the orbital rim. One must bear in mind that the orbital floor drops off several millimeters toward the inferior direction prior to heading straight posteriorly. The orbital floor dissection can then be extended posteriorly for a safe distance of 30 mm. With an intact adult rim, the optic canal is only 40 mm from the anterior lacrimal crest, and with any rim displacement inward, this margin of safety is further decreased. A modification of the subciliary approach is the "skin-only" incision. This technique is comparable to the technique just described, except that after dividing the skin, the inferior dissection is carried out superficially to the orbicularis oculi muscle fibers until the inferior orbital rim is reached, and then the muscle is divided at the same level as the periosteal incision. This approach is used less often owing to the amount of stretching on the unsupported large skin flap and the resultant high rate of ectropion (permanent in 8%) and potential skin necrosis, particularly in the elderly patient who has a history of heavy smoking [66]. These complications prompted the development of an alternative technique called the "skin-muscle flap." With this procedure a similar incision is accomplished 1–2 mm below the lid margin but is carried through both the skin and muscle at the same level down to the tarsal plate. Again, the plane of dissection is carried out anterior or superficial to the orbital septum (preseptal) until the orbital rim is encountered. This approach results in excellent esthetics, a simplified dissection, and a decreased incidence of hematoma formation or skin necrosis. This skin-muscle flap still carries a 6% rate of early ectropion [67]; however, it is generally temporary and resolves within several weeks with gentle massage. This was confirmed by several investigators who correlated preoperative periorbital edema and increased age positively with the development of this temporary ectropion with the subciliary approach [68]. A revision of this approach or technique is to use a relaxed skin tension line incision.

The transconjunctival approach for orbital floor fractures was first popularized by Tessier and Converse and colleagues in 1973 for orbital floor fractures [69, 70]. The two basic variations of this approach to the orbital rim are retroseptal or preseptal approaches. Although the retroseptal approach is a more direct approach to

the rim, it exposes the orbital fat, which herniates into the surgical field and may interfere with the surgery and result in more fat atrophy, especially with cautery, and hence enophthalmos. For this reason, the preseptal approach is generally favored [71]. The preseptal approach (see Fig. 24.16) as described by Tessier involves an incision through the palpebral conjunctiva just 2 to 3 mm below the inferior edge of the tarsus that is extended through the inferior lid retractors and orbital septum [69]. Next, a preseptal vertical dissection is carried out down several millimeters below the orbital rim, and the periosteum is incised. The dissection of the facial aspect of the rim and the floor is then carried out. This obviates orbital fat herniation in a fairly bloodless field. The necessity for a periosteal closure is controversial owing to the possibility of entropion or ectropion with inadvertent suturing of the periosteum to the orbital septum or other layers [69, 72]. Some surgeons advocate a Frost suture for a period of 24–48 hours to allow for proper lower lid redraping during early healing. Most surgeons find this unnecessary. If there is any difficulty in identifying opposing edges of the cut periosteum, then no suturing should be performed rather than an inappropriate tethering of more superficial or superior eyelid layers and structures to the underlying rim. Many instances of "early ectropion" or a "shortened lid" are the result of improper suturing. The transconjunctival preseptal approach enjoys a low incidence of unfavorable scarring with ectropion or entropion (1.2%) [60]. However, one drawback to this approach remains a somewhat-limited view during the preseptal dissection and limited exposure once the orbital floor has been accessed. For this reason, the lateral canthotomy and complete severance of the lower limb of the lateral canthal tendon (inferior cantholysis) were introduced by McCord and Moses in 1979 [73]. This procedure allows for a generous tension-free exposure to the orbital floor, lateral orbital wall, and medial area. The surgical exposure obtained with the transconjunctival approach with the inferior cantholysis is superior to that of a subciliary incision. Also, the much smaller cutaneous incision is placed in a more favorable area of the crow's-feet.

Lateral canthotomy has been associated with some complications. These complications include lateral canthal malposition with ectropion or entropion, webbing, blunting of the lateral canthal angle, and visible scars. A study done by RIU GD et al. reported 12.5% incidence of lateral canthal malposition and 12.5% incidence of visible scars when used lateral canthotomy with transconjunctival approach to the orbital floor [74]. Appling et al. also reported 3 cases that showed lateral canthal malposition, out of 36 patients, when he used lateral canthotomy in conjunction with preseptal transconjunctival approach for orbital fracture repair [75]. The cause of these complications is mainly attributed to the violation of the lateral canthal tendon which is difficult to precisely re-approximate to its exact anatomic presurgical position. This challenge of approximation is sometimes accentuated with the presence of edema during surgery.

24.8.3 Lateral Tarsal Approaches

An alternative approach called the "lateral tarsotomy" or the "lateral tarsal approach" is used by one of the authors (HE) in an attempt to avoid such complications [76].

The approach starts with an external lower lid skin incision. A full-thickness lateral tarsotomy incision is made 3–4 mm anterior to the lateral canthal angle approximately 7–8 mm in length (Fig. 24.17).

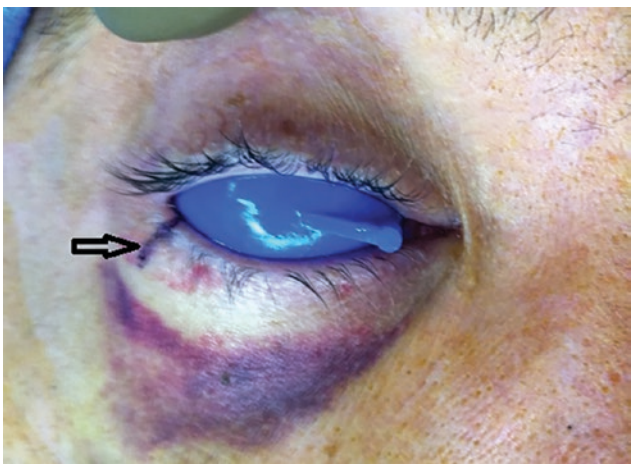


Fig. 24.17 Incision for lateral tarsoraphy approach 3–4 mm anterior to the lateral cathal angle

Fig. 24.18 Leave some eyelashes on either side of incision to allow re-approximation

The incision is through and through, carried inferior-laterally ensuring complete transection of the inferior lid tarsus. It is advantageous to leave some eyelashes on the lateral portion to facilitate an accurate alignment of the grey line upon closure (Fig. 24.18).

Beginning with the tarsotomy releases the lower lid and allows for better access to the fornix and completion of the transconjunctival portion of the approach. A standard preseptal transconjunctival approach is initiated by dissecting a small pocket using tenotomy scissors along the length of the fornix. The pocket is 2–3 mm inferior to the inferior aspect of the tarsus and includes both the conjunctiva and septum on either side. The dissection stops just short off the punctum. Dissection is then carried inferiorly and bluntly using "kittners dissection sponges" in a preseptal plane to the periosteum of the infraorbital rim (Fig. 24.19).

This technique of atraumatic blunt guidance helps contain and maintain the orbital fat in its native respective position without prolapse, in addition, it decrease the risk of injury to the pretarsal muscle by avoiding sharp dissection. The periosteum is then to be excised on the outer aspect of the rim below the arcus marginal. A subperiosteal dissection is then accomplished and the globe is retracted superiorly to gain access to the floor and inferior aspects of the orbital walls.

Fig. 24.19 Pre-septal plane of dissection

In contrast to Lateral canthotomy, the tarsotomy straight line incision and easy identification of the lid margins facilitate a more accurate re-approximation during closure. Many cases have been done by the author (HE) with excellent postoperative esthetic results. Each surgeon's own training, familiarity, and personal preference should guide which rim approach is used.

The majority of surgeons currently use the transconjunctival incision with or without canthotomy or the subciliary incision (preseptal approach) for orbital rim and floor access [69]. Both of these basic incisions provide good exposure with excellent esthetics and an extremely low rate of complications. Each surgeon's own training, familiarity, and personal preference should guide which rim approach is used.

Superior and Medial Orbital Approaches Access to the superior orbital rim and zygomaticofrontal (ZF) suture can be accomplished via a lateral eyebrow incision, upper blepharoplasty incision, coronal incision, or lateral canthotomy incision that is an extension of a subciliary or transconjunctival incision with a superior cantholysis. The eyebrow incision, if performed properly, results in excellent esthetics and is quickly and easily performed; therefore, it is one of the more common approaches used for the lateral orbital rim or ZF suture area. The other incisions described are used more often when extensive facial fractures are present that require extensive skeletal exposure of the superior rim, cranial vault, or zygomatic arch.

The lateral brow incision is placed on the extreme outer aspect of the eyebrow, usually just superior to the ZF suture. The ZF suture line is usually approximately 1 cm above the lateral canthus. Generally, the skin of the lateral brow is tented over the superior lateral orbital rim, and a 1.5 cm curvilinear incision is made in a beveled fashion paralleling the hair follicles. Double-pronged skin hooks are then placed on the skin margins, and traction is maintained with digital palpation of the internal edge of the orbital rim. The skin incision opening is then gently retracted inferolaterally more directly over the ZF suture, and a needle-tipped Bovie cautery is used to divide the orbicularis oculi muscle fibers overlying the rim and ZF suture. Additional undermining and dissection are carried out in an inferolateral direction to provide full and adequate access to the fracture and enough adjacent bone to allow for rigid fixation. The advantages of not extending the skin incision beyond the brow obviously involve esthetics (placing it in the well-camouflaged and hidden area of the hair follicles) but also include that the skin is stepped and muscle incisions are made in distinct layers, which provide for more favorable healing. This incision also allows access for placing a blunt curved instrument deep to the zygomatic arch for the reduction of the ZMC or arch fractures. Closure should be accomplished in three distinct layers of periosteum, subcutaneous tissue, and skin. The periosteal, muscle, and deep subcutaneous closures are particularly important in that they provide the bulk of soft tissue over any plates and screws in the region.

The upper blepharoplasty incision can also be used for access to the ZF suture. The incision is placed in one of the upper eyelid skin creases, preferably the deepest crease (which can be marked preoperative ly, with the patient awake). The skin incision is then carried down through subcutaneous tissue, retracted somewhat laterally, and extended through the orbicularis oculi and periosteum by sharp dissection. Generally a 1 cm length of the lateral blepharoplasty incision is all that is required for complete access to the lateral orbital rim. This is due to the suppleness and mobility of the thin eyelid skin. Care should be taken to not over-retract the tissue, and the skin incision should be extended slightly laterally if excessive retraction forces are apparent. Separate suturing of the periosteum and skin are all that is required.

The coronal incision allows for excellent access to the entire supraorbital rim, roof, frontal sinus, superior aspects of the nasal bone, lateral orbital rim and wall, medial orbital rim and wall, and zygomatic arch [77]. This approach is generally necessary for extensive facial fractures involving the zygoma, frontal sinus, and NOE complex and for Le Fort III fractures. Numerous variations of the incision design exist, but generally a curvilinear incision is placed 4–5 cm posterior to the hairline (in the midline) and then extended posteriorly, paralleling the hairline, and finally inferiorly into the preauricular region. It is generally helpful to carry the vertical component of the coronal incision overlying the temporalis muscle just posterior to the junction of the superior helix and the scalp. It is then sharply angled forward, hugging the anterior helix and preauricular skin crease down to the pretragal area. By doing so, the superficial temporal vessels are generally not encountered or violated and retracted forward with the flap, allowing for a much drier field. It is not necessary to shave the scalp, but a 1 cm area of hair can be trimmed at the incision to allow for ease of closure, postoperative hygiene, and suture removal. Local anesthesia with vasoconstrictors is helpful for hemostasis and often obviates the need for compression (Raney) clips. The incision is carried out through the skin, subcutaneous connective tissue, and galea aponeurotica into the loose areolar tissue in the midline. The subgaleal plane of dissection is contiguous with a plane deep to the temporoparietal fascia in the area of the temporalis muscle. The incision is then extended laterally in the supraperiosteal plane; it is helpful to insert a Metzenbaum or curved Mayo scissors in this plane prior to extending the incision laterally. This prevents inadvertent incising or nicking of the temporalis in an otherwise dry field. The dissection is carried out laterally to the superior temporal line bilaterally. Dissection is then carried anteriorly to the frontal bone, and a horizontal incision is made through the periosteum approximately 2 cm above the superior orbital rim. The incision is carried laterally to the superior temporal line and joined with the preauricular area inferiorly through the superficial layer of the deep temporal fascia to protect the temporal and frontal branches of the facial nerve [78]. The facial nerve courses in a plane superficial to the superficial layer of the deep temporal fascia approximately 1 to 3 cm from the tragus along the zygomatic arch [79]. This approach provides complete access to the medial, lateral, and superior orbital rims. When a more extensive view of the medial orbital wall is required, subperiosteal dissection and release of the superior trochlea can be performed—the flap is retracted more inferiorly over the nasal dorsum, with a direct view of the medial wall. No attempts should be made to reattach the trochlea since, when the soft tissues are redraped, the trochlea re-adheres on its own. Suturing may actually pierce or violate the trochlear tendon and result in ocular motility disturbances. Closure of the coronal flap should include suspending the deep temporal fascia over the temporalis muscle, deep closure of the galea aponeurotica, subcutaneous buried suturing, and closure of the skin. It is important to remember that when a hemicoronal incision is employed, the medial

extent of the incision should be carried beyond the midsagittal plane and extended completely to the hairline. This allows for adequate reflection and retraction over the entire zygoma and orbital rim structures.

When a transconjunctival incision is used with a lateral canthotomy, an extension of the dissection superiorly can be used for access to the ZF suture by severing the superior limb of the canthal tendon [72]. This approach provides good access to the lateral and infraorbital skeleton; however, it is less frequently used because it requires a more complex closure and reanchoring of the lateral canthal tendon complex. Any misalignment results in canthal dystopia, usually in an inferior direction, and a rounded-out "almond-shaped" eye appearance. If the superior canthal tendon and its origin to the internal rim are allowed to remain intact, it provides a highly reliable landmark to which the inferior canthal limb can be sewn, resulting in excellent sharp-angled (30–40°) esthetics.

The entire lateral wall and rim are easily accessed through a standard blepharoplasty incision that extends only to the lateral orbital rim. This approach is commonly used for lateral orbital decompressions in cases of severe thyroid orbitopathy and it affords excellent exposure also to portions of the orbital roof and to the apex of the orbit laterally.

Medial Orbital Approaches Access to the medial orbital rim and superior aspect of the medial orbital wall can be accomplished through a coronal incision, as previously described. However, a separate lateral nasal incision can be used for isolated medial wall exploration or to access the inferior aspect of the medial orbital floor. This can be a transconjunctival or subciliary approach to the inferior rim and floor. The entire medial wall can be visualized by extending the transconjunctival incision through the caruncle. The medial orbital wall and rim, by definition, are involved in fractures of the NOE complex, Le Fort II and III fractures, extensive frontal sinus fractures, and, occasionally, large blow-out fractures. The lateral nasal incision is most often used for access to the medial orbital rim to reconstruct a detached medial canthal tendon with direct transnasal wiring. This type of injury often occurs with NOE fractures and Le Fort III fractures. As stated earlier, medial orbital wall fractures generally do not result in any entrapment or ocular mobility problems. Generally the upper one-third of the medial orbital wall is uninvolved or nondisplaced, simply because it is the very thick extension of the cranial base. The lower two-thirds of the medial orbital wall overlie the ethmoid air cells and can be displaced inward, resulting in volume expansion. Unless there is extensive involvement, generally the resulting increase in orbital volume does not result in the development of enophthalmos. If the inferior two-thirds of the medial wall or orbital floor are involved and require surgical repair, then the previously described approaches to the orbital floor should suffice. However, fractures that extend farther superiorly (above the frontoethmoidal suture/anterior ethmoidal foramen) may require a lateral nasal approach or coronal incision. The lateral nasal approach involves a vertical gentle curvilinear 1 cm incision approximately 5–10 mm medial to the insertion of the medial canthus. Care should be taken not to place this incision too close to the medial canthus as this can result in a scar contracture with "webbing" and an abnormal epicanthal fold postoperatively. The incision should be placed over the lateral nasal structures properly, and after the skin incision is made, the dissection should be carried straight medially through the skin, subcutaneous tissue, and a rudimentary portion of the orbicularis oculi muscle and periosteum. There is no need to step these layers. The periosteum can then be reflected posteriorly and superiorly to the medial orbital rim and wall. The medial canthal tendon and lacrimal sac lie posterior and just inferior to the incision. The anterior ethmoidal vessels lie posteriorly and superiorly approximately 24 mm from the anterior lacrimal crest. These vessels can be gently divided with bipolar cautery, providing excellent hemostasis and improved access for identifying an intact bony ledge. However, one should bear in mind that any bony violation or entry superior to this line carries the potential risk for entry into the anterior cranial fossa. When an orbital implant is required along the medial wall, anterior fixation of the implant is recommended.

The transcaruncular approach is not a very popular choice for most surgeons to approach the medial orbit but has its indications. The plica semilunaris is the crescent-shaped fold of the conjunctiva and is considered a vestigial remnant of the nictitating membrane. The caruncle is the fleshy keratinized structure attached to the medial side of the plica semilunaris and the common canaliculis is just medial to this. Traction sutures placed on the medial portion of both the upper and lower lids and holding the plica semilunaris with forceps taking care not to damage the puncta and canaliculi, an incision is made through the caruncle with Westcott scissors and extended inferiorly and superiorly by about 10–12 mm. Blunt dissection is then carried medially by opening the Stevens scissors, and the exposed periosteum is incised in a superior-inferior fashion. Retractors are placed to provide an unobstructed view of the medial wall and the floor of the orbit (Fig. 24.20a–c). The incision is closed using a 4.0 fast resorbing suture. The transcaruncular approach to the medial orbital wall allows excellent access and is considered by some to be cosmetically superior to the Lynch approach [80, 81].

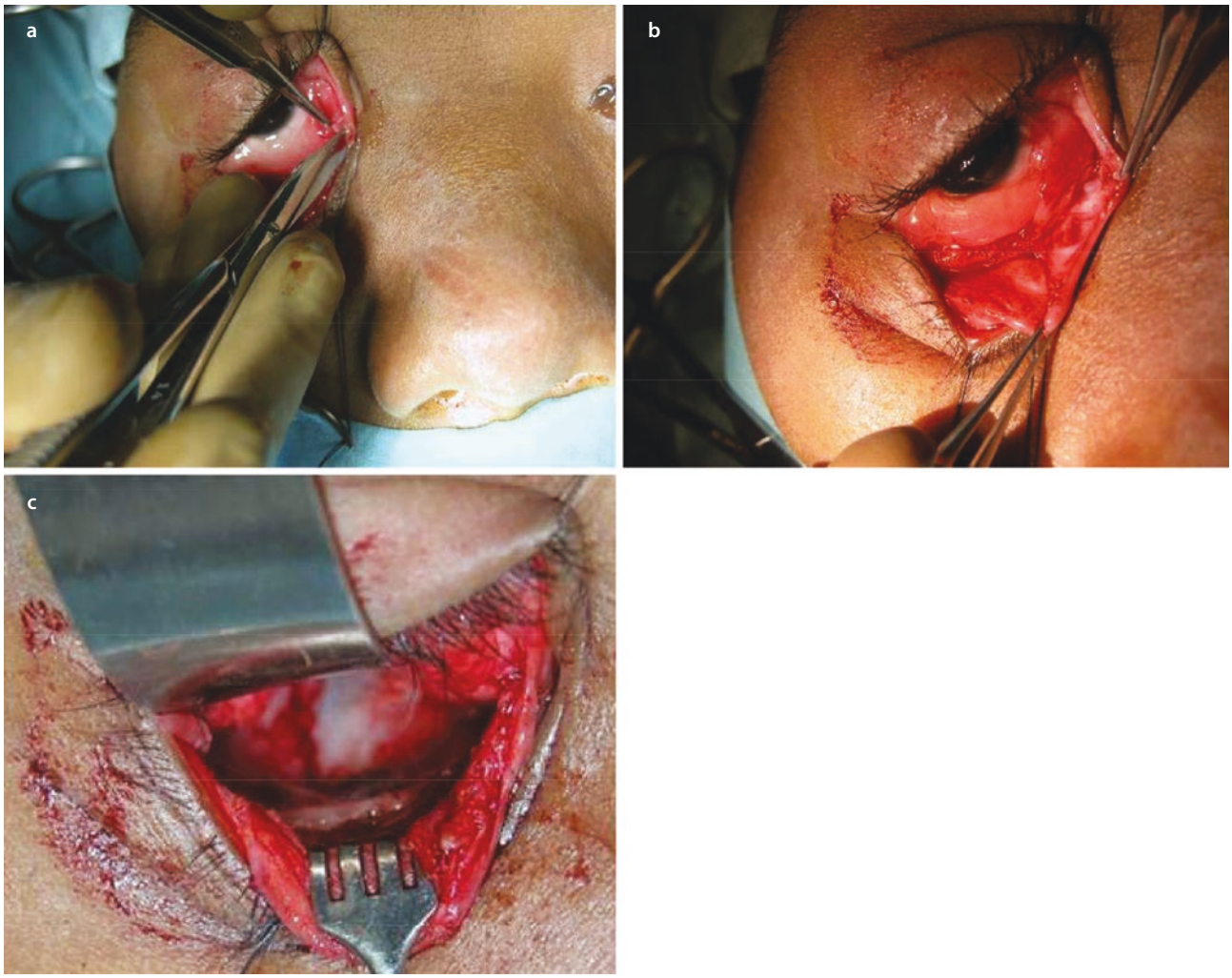


Fig. 24.20 Transcaruncular approach to the medial orbital wall and floor **a**. Incision through the caruncle with Westcott scissors **b**. The incision is extended inferiorly and superiorly 10–12 mm **c**. Blunt dissection medially by opening the Stevens, the periosteum is incised in a superior-inferior fashion, malleable and rake are used for retraction, unobstructed view of the medial wall and the floor of the orbit

Endoscopic Approach The endoscopic approach to the orbit has gained popularity over the years [82]. The approach utilizes a 30 degree 3 mm transantral endoscope to assist with visualization of the orbital floor through a Caldwell-Luc antrostomy window. The sinus mucosa is then dissected around the fractured orbital floor using sinus instruments, and the fracture is delineated. A 0.4 mm Medpor (Porex Surgical, Inc., College Park, Ga) is then folded and inserted into the orbital defect taking support of the intact ledges when unfolded. This approach provides the greatest advantage of avoiding a skin or conjunctival incisions. Disadvantages include limited exposure, a steep learning curve with potentially longer operating times (Fig. 24.21).

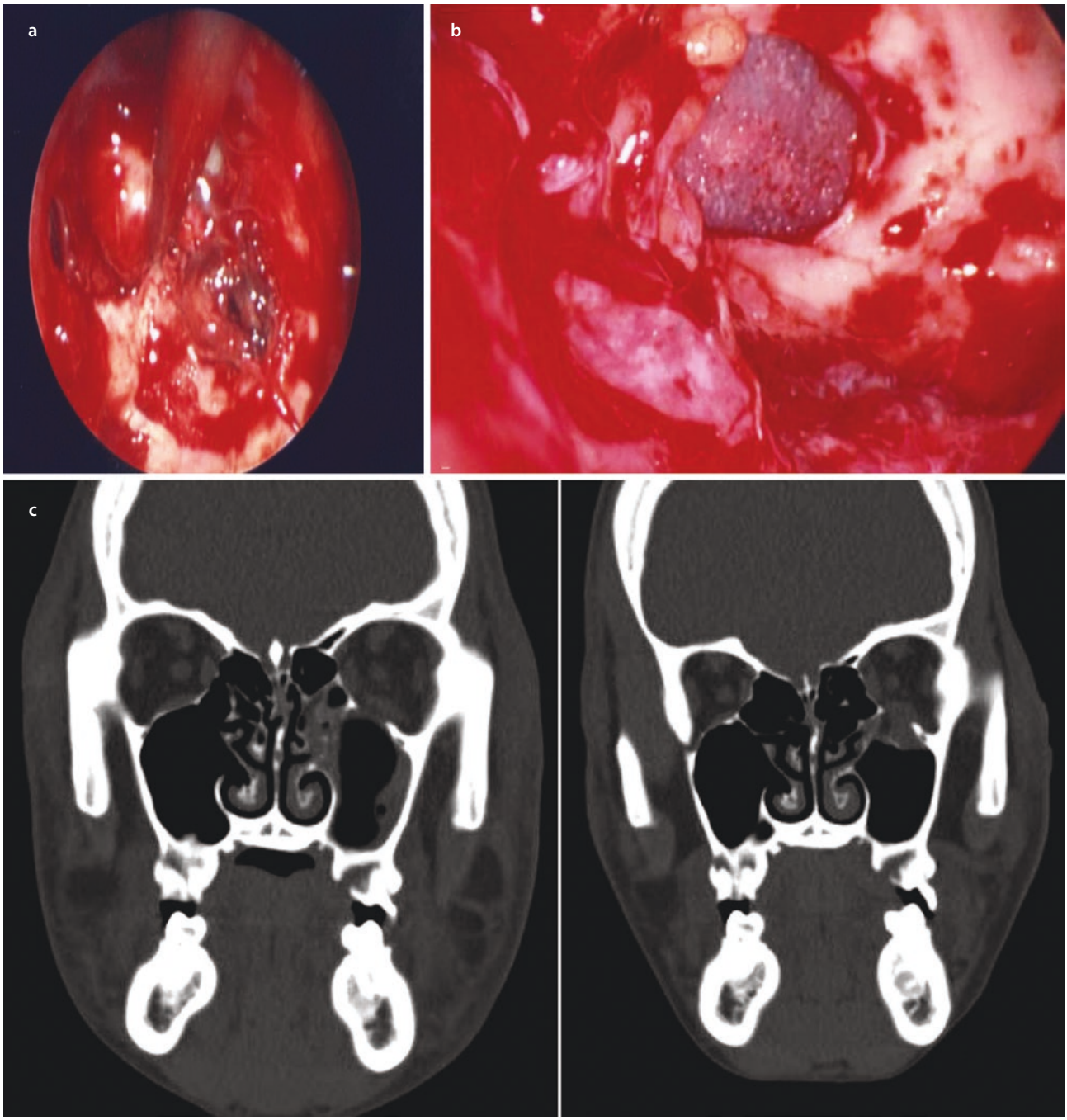


Fig. 24.21 Endoscopic approach to the orbital floor. **a** Endoscopic approach to orbit Endoscopic view through maxillary sinus looking up at the fractured orbital floor and orbital contents herniating. Photos courtesy Dr Rui Fernandes, Jacksonville, FL. **b** Good reduction of orbital contents, removal of all bone, and good placement of 0.4 mm porous polyethylene orbital floor implant spanning the entire defect. Photos courtesy Dr Rui Fernandes, Jacksonville, FL. **c** Pre- and post-operative views of orbital floor fracture repaired endoscopically. Photos courtesy Dr Rui Fernandes, Jacksonville, FL

24.8.4 Acute Repair

Internal orbital fractures have varied patterns and degrees of severity. It is helpful to attempt to classify them either as linear, blow-out, or complex fractures. Linear fractures are those in which the bone fragments and walls remain intact. However, owing to angulation or overlap, they may result in either a bony orbital volume increase or decrease. Overlap fractures generally result in a bony defect of one orbital wall (typically the medial orbital floor) and are the most common orbital fracture. Blow-in fractures can occur in any orbital wall but most commonly occur in the roof and are associated with frontal sinus fractures. Blow-in and blow-out frac-

tures of the orbital roof occur with equal frequency. Complex fractures are those that involve two or more walls, are >2 cm in diameter, or are comminuted with displaced and unretrievable segments. Often these complex fractures are associated with fractures that extend beyond the orbital frame such as Le Fort II or III and frontal sinus fractures. These are termed combined fractures. The goals of acute or primary reconstruction of primary orbital fractures are to alleviate any functional deficit and to restore the facial esthetics.

Linear fractures are generally caused by blunt forces directly to the globe or partially to the rim and most often result in an esthetic deformity such as enophthalmos or hypo-ophthalmos. Functional deformities with entrapment are less common with linear orbital fractures. However, isolated linear fractures can have an instantaneous trapdoor effect owing to momentary expansion and entrap the edge of soft tissues including the inferior rectus. Once tightly pinched between these bony segments, this manifests itself as severe ocular motility restriction that is reproducible on serial examinations at the same point of limitation. There is also a positive result to the forced duction test. This kind of entrapment of the muscle can also lead to the vagally mediated oculo-cardiac reflex [83]. This type of fracture necessitates immediate surgical intervention to prevent the ischemic necrosis of the extraocular muscles. The majority of linear fractures in the orbit do not result in esthetic deformities such as enophthalmos or hypo-ophthalmos unless there is an associated facial fracture such as a fractured ZMC with a medial and downward rotation. It is the volume changes that account for the abnormal globe position. The goal of reconstruction is to restore the anatomic position of the bony rim and associated facial bones and to reapproximate, to the best of one's ability, the normal bony orbital volume with a reconstructive material. Numerous materials have been described in the literature for these purposes, such as porous polyethylene, bioresorbable polydioxanone, nylon, gelatin film, titanium mesh, and autogenous bone grafts (split-thickness calvarium and, less frequently, iliac crest) [84–90]. Each material has advantages and disadvantages related to the strength, application, reactivity, infection rate, biointegration, and complication rate associated with its use.

For linear and blow-out fractures, thin (0.85 mm) porous polyethylene sheeting could be used. This alloplastic material is extremely biocompatible and nonresorptive. It has more than adequate tensile strength and does not cause any capsule formation such as that seen with polymeric silicone sheeting. It has considerable flexibility (which can be improved with placement in an autoclaved saline) and little memory properties. The pore size allows tissue ingrowth, which reduces the risk of migration [91]. Despite that, it is often recommended

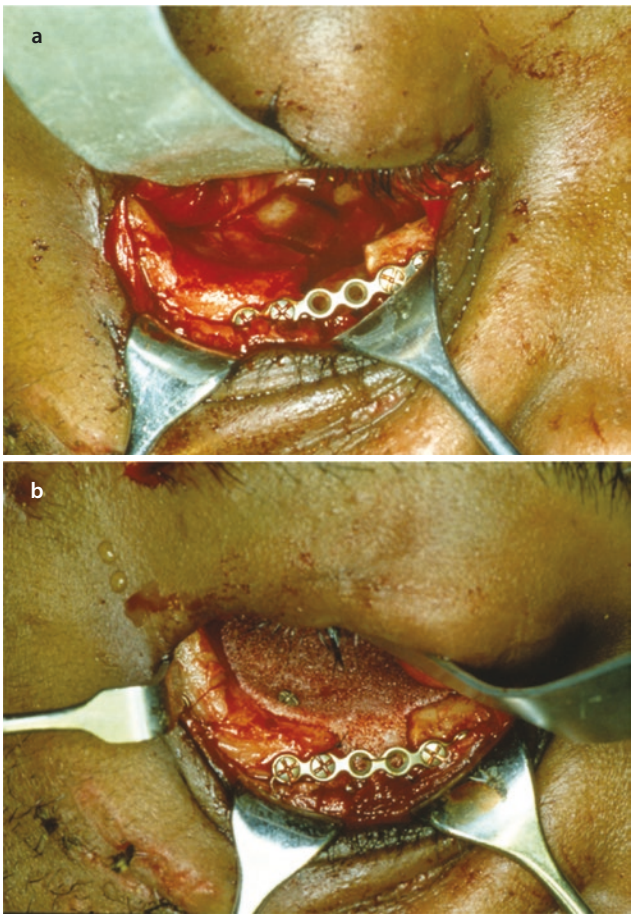


Fig. 24.22 **a** Right inferior orbital rim and floor fracture reduced and fixated with a 1.7 mm microplate. A portion of the mid-rim was suctioned away from the antrum and was missing. **b** The floor defect was reconstructed with 0.85-mm-thick porous polyethylene sheeting secured with a single 4-mm-long 1.7 mm screw at the anterior lateral intact floor. A tab extension of the sheeting was fashioned at the rim defect, curved, and secured with a 5-0 nylon mattress suture

to anchor the porous polyethylene sheeting to the anterior lateral orbital floor with a single titanium screw (Fig. 24.22). The greatest advantages of this material are its ease of contouring, in situ carving, burring, and that it can be layered posteriorly behind the orbital equator to achieve proper orbital volume and contour [57].

Titanium mesh, with fixation to surrounding intact orbital rims, is quite useful when there are severe or comminuted injuries and a cantilevering is required because intact internal medial or posterior bony margins have not been identified or accessed. However, the possibility of unacceptable postoperative scarring to the mesh may occur, resulting in limited ocular motility. Therefore, when titanium mesh is employed, it is recommended to overlay it with either a split-thickness calvarial graft or a sheet of porous polyethylene sheeting. These materials are secured to the underlying mesh with either 30-gauge stainless steel wire or suturing (Fig. 24.23).

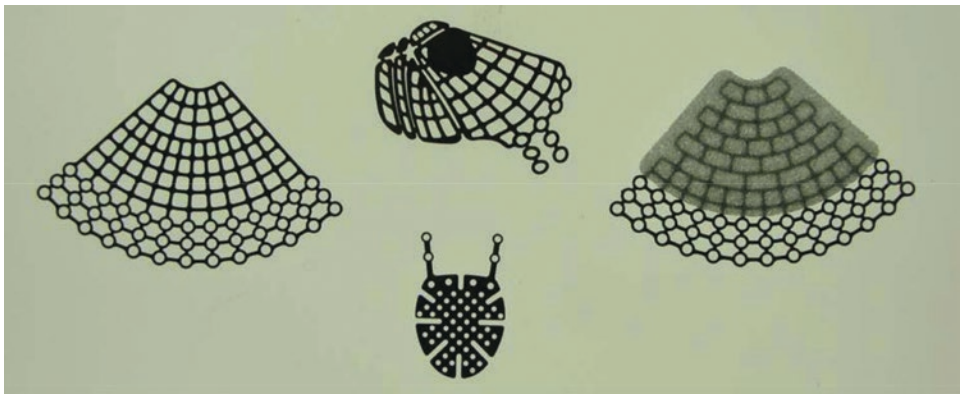


Fig. 24.23 Different types and thicknesses of titanium orbital floor implants

Blow-out fractures typically involve one orbital wall (usually the anterior or medial portion of the orbital floor) and are <2 cm in diameter. Enophthalmos associated with orbital blow-out fractures is due to an enlargement of the orbital bony volume that allows the orbital fat to be distributed within a larger compartment [44]. Fat atrophy contributes little, if anything, to the development of early or late enophthalmos [92]. The reverse mechanism, often referred to as blow-in fracture, may result in a decreased orbital volume. Exophthalmos and ocular motility disturbances are uncommon unless there are surrounding severe associated fractures such as ZMC or frontal sinus fractures.

In 1960 Converse and Smith introduced the concept of "pure" (isolated floor) and "impure" (floor and rim) blowout fractures [44]. Pure fractures are thought to be caused by a sudden instantaneous increase in intraorbital pressures from direct blunt-force trauma to the globe itself. Impure fractures are purported to be caused by direct trauma and compression of the bony rim and collapse of the surrounding facial bones, and result in the disruption of the internal orbital walls. What is most disconcerting is the finding of associated globe trauma such as hyphema, iridoplegia (ciliary body paralysis), and retinal hemorrhage in 90% of patients with pure blow-out fractures. This supports the notion that pure blow-out fractures are created by substantial instantaneous direct globe trauma. This fact should heighten one's awareness of the potential for serious-globe injury when dealing with isolated or pure blow-out fractures.

The goal of primary reconstruction of blow-out fractures is to restore the configuration of the orbital walls, return prolapsed orbital contents to the orbit proper, and eliminate any impingement or entrapment of orbital soft tissues. In contrast to the orbital floor blow-out fractures, isolated blow-out fractures to the roof or medial walls usually do not contribute significantly to the development of cosmetic deformities or result in entrapment or limited ocular motility. As a result, medial and roof defects are managed by observation, serial examinations, and intervention when symptoms warrant. The most difficult area of the orbital floor blow-out fracture to repair is the posterior medial extent, which is beyond the globe axis. Often, an intact bony ledge cannot be identified or the graft material is not extended posteriorly enough to support the orbital contents in this region. This area is often responsible for a failed enophthalmos repair in orbital blow-out fractures. It is the reconstruction of this posterior medial floor to its normal contour that is the key to restoring normal globe position both anteroposteriorly and vertically. It is this scenario that is problematic in delayed reconstructions since attempts to create a normal anteroposterior position of the globe may result in inappropriate overpositioning of the globe in a superior direction. The authors prefer to use gelatin film as a temporary barrier for small or linear defects, simply to prevent entrapment during normal active ocular motion. This film is resorbed rather rapidly and does not provide much structural support; therefore, it is not used for larger defects in which herniation of contents into the underlying sinus is a possibility. Generally, the orbital blow-out fracture is explored in all of the intact bony walls identified. Once the malleable ribbon or globe retractors have supported the globe and orbital contents superiorly, then the reconstructive material can be slid underneath them and overlap the intact bony margins slightly at the majority of areas to provide adequate support. The newly developed orbital retractor (Fernandes orbital retractor; Biomet, Jacksonville FL.) offers the advantage of adjustable working ends and a graduated surface that allow the operator to gauge the depth of retraction (Fig. 24.24). Using a transparent Jaeger retractor – the lucite lid plate has the particular advantage of constantly being able to inspect the pupil intraoperatively while retracting the contents of the orbit while working on the orbital floor. Additionally, it is nonconductive and inadvertent touching of monopolar

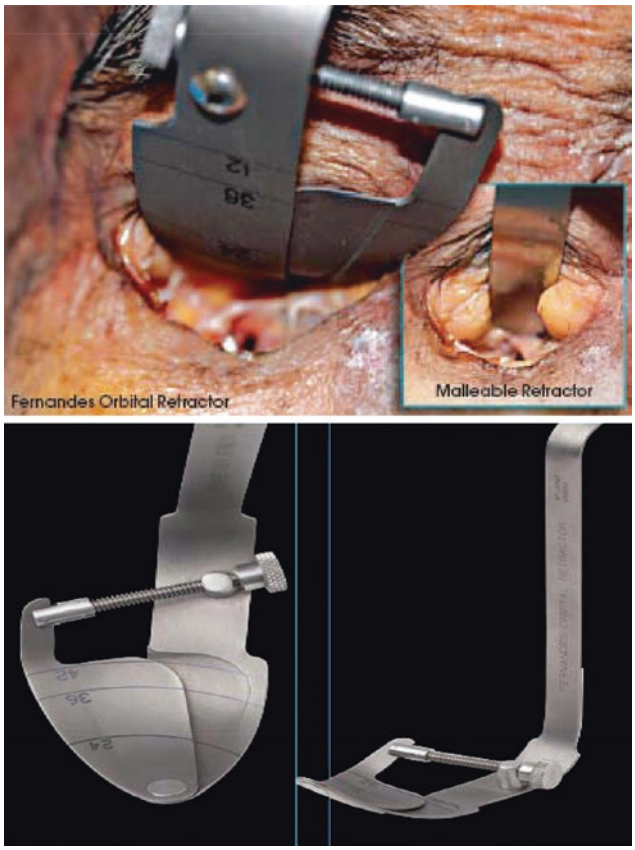


Fig. 24.24 Fernandes orbital retractor; Biomet, Jacksonville FL

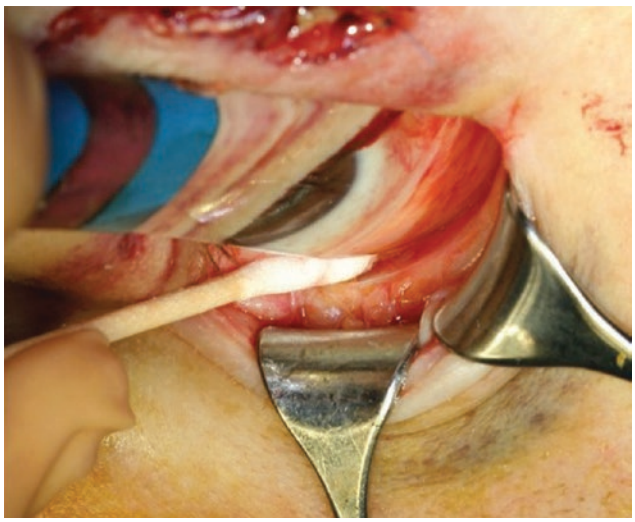


Fig. 24.25 Using the Jaeger lid retractor made of Lucite

or bipolar cautery against the retractor surface will not cause harm to the cornea or globe (Fig. 24.25).

Several surgeons prefer to use porous polyethylene for moderate to large blowout fractures. The porous polyethylene sheeting can be secured with a single positional screw (usually 1.7 mm external thread diameter) or an extended tab of this material can be sutured to the orbital rim orbital plate (see Fig. 24.22). Care should be taken to not extend the grafts up to the orbital rim or over the edge since these will be palpable and would improperly reconstruct the normal anatomic contour to the floor, which should dip down behind the rim for several millimeters before proceeding posteriorly. Also, the extension of semirigid grafts onto the orbital rim has an undesirable ramping effect, which tends to position the globe in an abnormal posterior direction, resulting in enophthalmos. After the floor graft is placed and secured, trimming or smoothing should be accomplished and a forced duction test performed prior to any wound closure to ensure that no impingement of the soft tissues has occurred.

Complex orbital fractures are generally associated with additional surrounding midfacial and frontal sinus fractures. Primary reconstruction of these defects is challenging owing to the extent of these injuries, the lack of any normal identifiable anatomy, and poor surrounding bony support for rigid fixation and anchoring of reconstructive materials. However, it is in this group of individuals that primary repair with normal anatomic realignment is critical for acceptable esthetic and functional outcomes. Delaying the primary repair beyond 7–10 days usually results in some secondary soft tissue changes, the inability to completely retrieve small bony segments, and a less-than-desirable outcome. The initial step in the reconstruction of complex facial fractures is adequate exposure of all midfacial structures with adequate alignment and reduction prior to rigid fixation of any components with plates and screws (Figs. 24.26 and 24.27). This helps one avoid misalignment, overreduction, or improper angulation of these segments. Achieving adequate exposure requires more extensive subperiosteal dissection than is done for most other orbital fractures. It may be desirable to also completely dissect and expose all internal orbital fractures prior to fixation of the surrounding periorbital or midfacial fractures. Generally the orbital rim is plated with 1.7 mm or finer plating systems. Care should be taken at the inferior orbital rim and especially the lateral orbital rim to keep the plates several millimeters from the edge of the rim; otherwise, they will be annoyingly palpable once the soft tissue edema has subsided. Once the orbital rims and midfacial bones have been fixated, the moderate to large orbital floor defects are generally repaired with porous polyethylene and anchored to the anterior-inferior floor with a single screw. Sometimes layering of this material with an additional sheet posteriorly is required to achieve correct anteroposterior globe positioning. More extensive defects may require titanium mesh or orbital floor plates with screw fixation

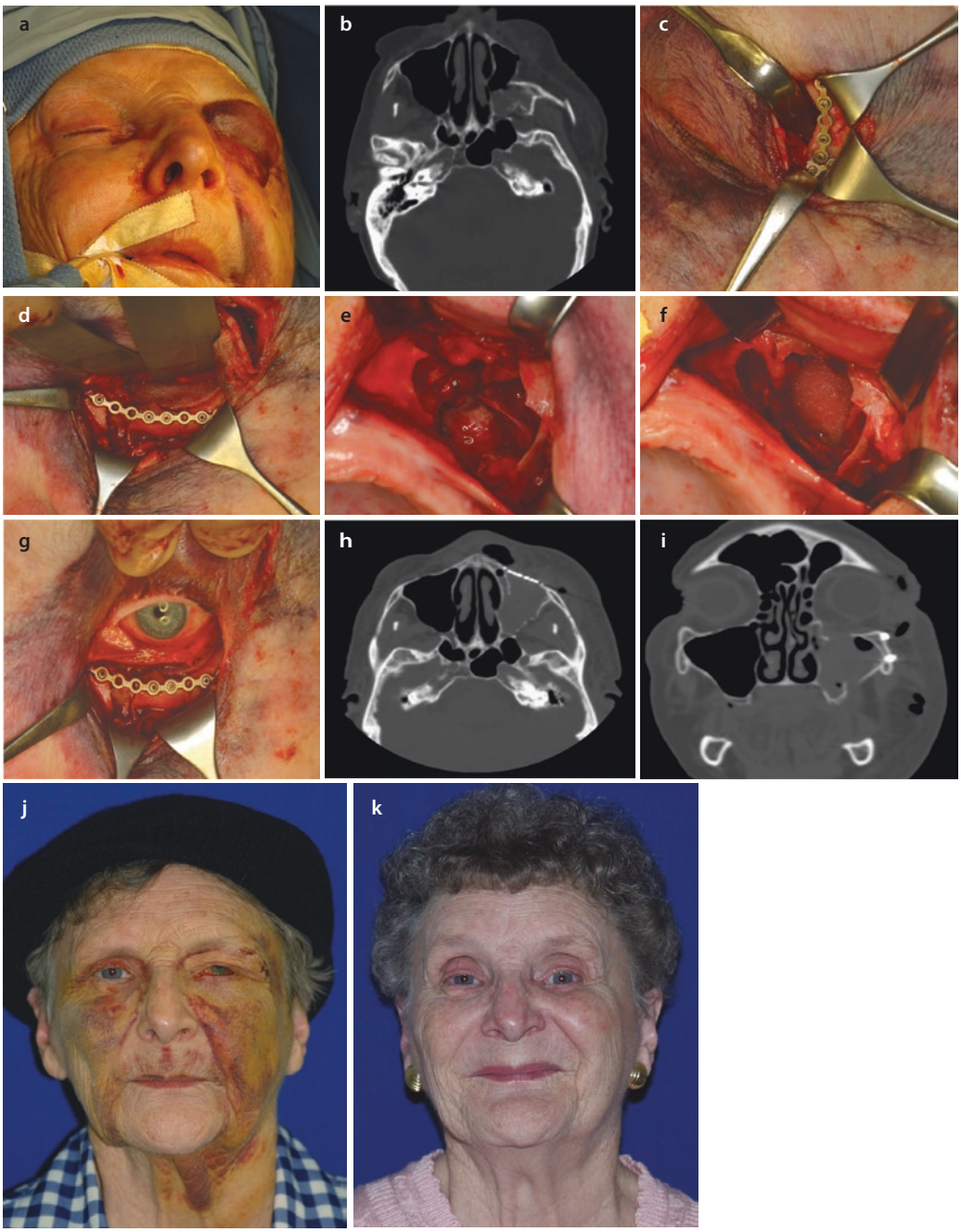


Fig. 24.26 **a** An elderly female sustained a severely displaced left zygomatic complex (ZMC) fracture with >75% orbital floor disruption. She was on warfarin sodium and had moderately decreased left visual acuity with increased ocular pressures. **b** Axial CT scan revealed a ZMC fracture with a severe posterior, medial, and moderate inferior displacement. **c** The patient was taken urgently (within 12 h) for surgical treatment to reduce the fracture and re-expand the orbital volume. Serial examination and ocular pressure checks were performed every 2 hours pre- and postoperatively. Owing to cardiac risk factors, the anticoagulation was not reversed nor was the patient treated with fresh frozen plasma. The zygomaticofrontal (ZF) suture area was first approached through a lateral brow incision. After the intraoral vestibular and then transconjunctival approaches were accomplished, the ZF fracture was plated. **d** The infraorbital rim was fixated with a 1.2 mm titanium plate, and the floor was reconstructed with 0.85 mm porous polyethylene sheeting. **e** The left maxillary sinus anterior wall defect visualized through the vestibular incision along the edentulous ridge. Note the herniated orbital soft tissues. **f** After retrieval of the orbital soft tissues from above and insertion of the porous polyethylene floor graft, the repair was inspected from below ensuring that there was no tissue prolapse or entrapment. The fracture was then spanned from the buttress to the intact medial maxilla with a 1.7 mm plate. The anterior maxillary wall defect was not grafted. **g** The eye position was assessed with the contralateral side, and a forced duction test revealed a free and full range of motion. **h** The patient had a routine hour follow-up computed tomography scan of the head, as per the request of the neurosurgeon. The images of the patient's face demonstrated excellent realignment. Postoperatively she had greatly improved vision, and no neurologic impairment. She was discharged home on postoperative day 2 on warfarin sodium. **i** The reformatted coronal images show good orbital floor support of the globe. **j** Facial appearance at 1 week postoperatively. **k** Six weeks postoperatively this patient had no complaints, and her baseline visual acuity had returned

to the rims and autogenous bone grafts. Several bone grafts can be secured to the metallic mesh framework to independently reconstruct the floor, medial wall, and, less frequently, the lateral orbital walls. The advantage of having bone overlie the metallic mesh is that remodeling can occur—secondary revision surgery is enhanced when dissecting along a healed bony surface versus bare mesh. In severe or large defects with comminution, overcorrection of the enophthalmos component (but not a hyper-ophthalmic deformity) by several millimeters is often necessary to take into account the orbital edema that exists. In addition, with bone grafts, some mild resorption can take place with subtle settling. However, it is the resolution of the edema that accounts for the majority of postoperative globe position changes.

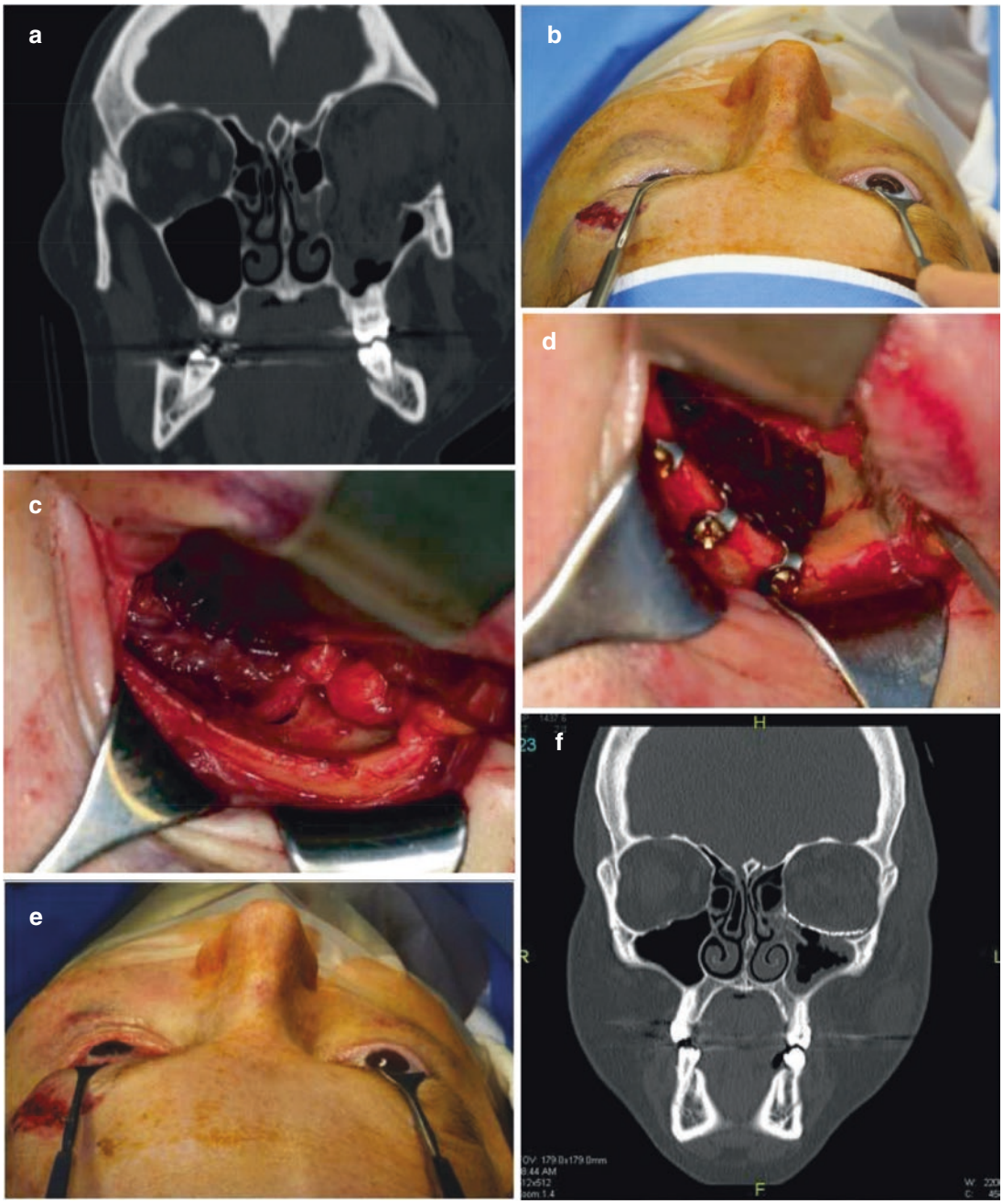
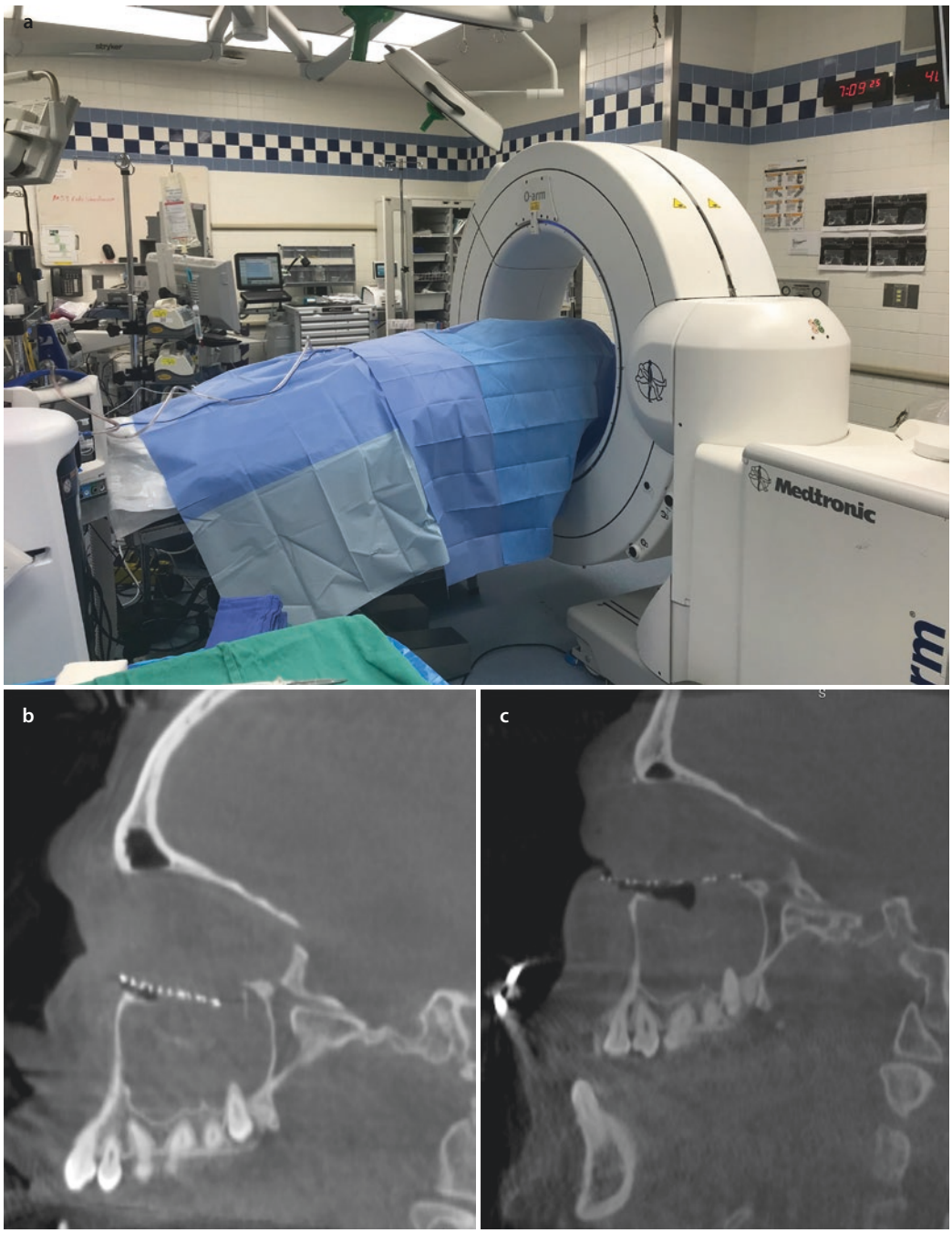


Fig. 24.27 **a** Coronal CT showing large orbital floor defect. **b** Preoperative bird's eye view of severe enophthalmos resulting from the large orbital floor defect. **c** Herniated contents released and fracture reduced. **d** Orbital floor reconstructed using titanium orbital floor mesh fixated at inferior orbital rim. **e** Postoperative bird's eye view of repaired orbital floor defect correcting the severe enophthalmos. **f** Coronal CT of repaired orbital floor

24.8.5 Virtual Surgical Planning and Mirror Imaging Overlay(MIO)

Recent advances in imaging and computer software with the ability to provide custom-made implants have significantly decreased then incidence of postoperative complications and the need for revision surgeries. Steriolithoscopic models proved to be useful in preoperative plate contouring, symmetry and precise positioning. Computer-assisted surgical simulation without physical models has also been used. The ability to mirror image the contralateral unaffected side (Mirror Imaging Overlay-MIO) and superimposing the image on the fracture side allowed the production of an accurate template for fabrication of a precise custom-made implants and accurate symmetrical reconstruction (◘ Figs. 24.28 and 24.29).



◘ **Fig. 24.28** **a** O-Arm™ set up for an intraoperative orbital floor imaging. **b** Inadequate extension of the orbital floor plate. **c** Repositioned orbital floor plate as seen on O-Arm™ image

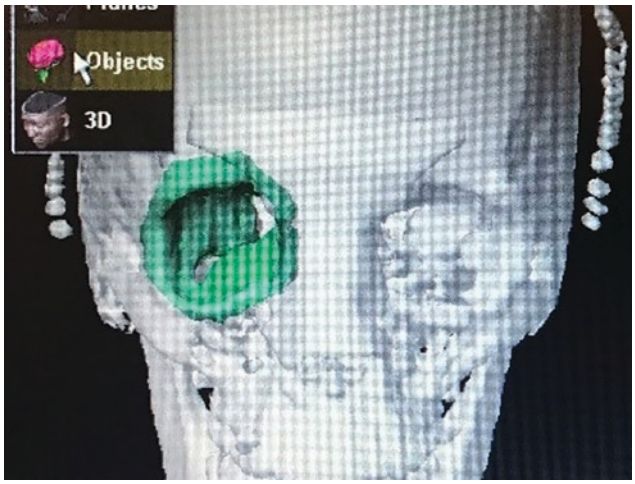
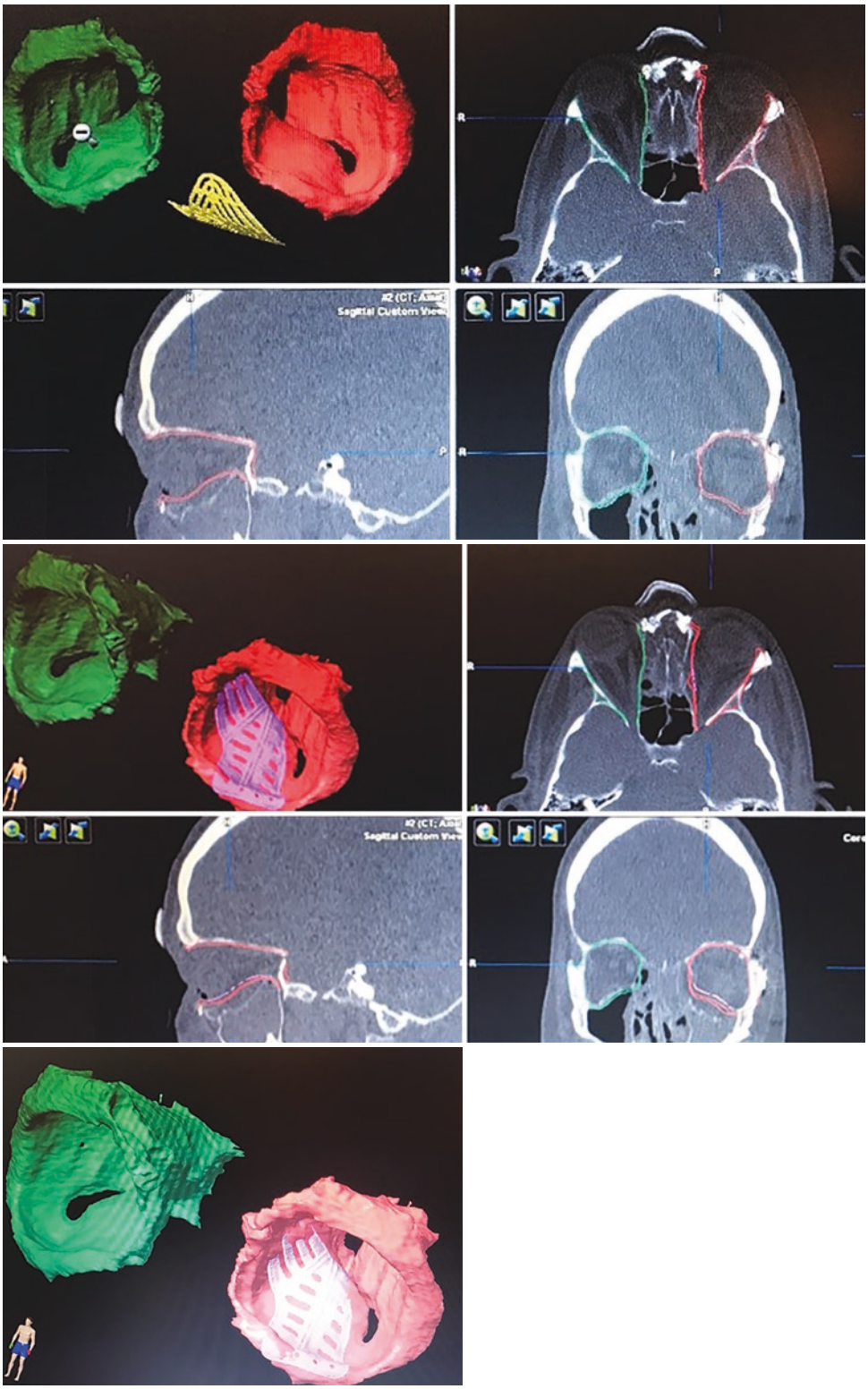


Fig. 24.29 A steps in 3D planning include digitally mirroring the uninjured side based on CT scan imaging

24.8.6 Navigation-Guided Implant Placement

With complex orbital fractures involving multiple regions, it can be difficult to determine whether the orbital contents have been fully repositioned, the posterior edge of the fracture has been bridged, or the implant is within a safe distance from the optic canal. Once the implant is in place, intraoperative navigation can be used to navigate along its entire surface to confirm its position.

The use of virtual surgical planning and navigation-guided surgery techniques have positively influenced the outcome of complex cases requiring secondary revision surgeries. The associated muscle damage/atrophy, scar tissues, bone defects make it very difficult to achieve an acceptable symmetry in these cases. With the help of the aforementioned technology, the production of a customized implant to achieve symmetry provided very satisfactory results [93] (Figs. 24.30, 24.31, and 24.32).



Figs. 24.30, 24.31, and 24.32 Custom implant fabrication using navigation technology

24.8.7 Intraoperative Imaging

Intraoperative CT scanning either with a CBCT or a helical CT has been proven to be a practically effective aid in the management of orbital floor reconstructions [94]. This imaging permits real-time assessment of placement of the orbital floor hardware and significantly reduced the need to take patients back to surgery for re-exploration and replacement of the hardware based on a postoperative imaging. Different options for intraoperative imaging are available. An O-Arm™ is a mobile unit that has been most reported in the literature for this purpose [95].

Some discussion is warranted here, as ZMC fractures relate to orbital involvement and appropriate intraoperative sequencing. Only after reduction and stabilization of the entire external orbital framework and surrounding facial bones should the internal orbital defects be repaired (see ◘ Fig. 24.26c–g), The internal orbital injuries associated with fragmented ZMC fractures usually involve multiple orbital walls and larger defects. Therefore, more extensive exposure is generally necessary, and more rigid materials are usually required for reconstruction.

Conclusion

Orbital fractures are often associated with ocular injuries and midfacial fractures. A thorough ophthalmologic evaluation is mandatory to detect ocular injuries and to preserve vision. Surgical intervention should be based on either a functional deficit or a cosmetic deformity. The surgical sequencing and timing of the repair should be well thought out. When visual compromise exists, an ophthalmologist should be involved in the treatment planning. Advances in maxillofacial imaging, virtual surgical planning, and navigation-guided surgery have become very useful tools in improving the postsurgical functional and esthetic outcomes in orbital fractures repair.

Acknowledgments The authors wish to acknowledge the contributions that have been made to this chapter by the senior author of its previous editions – Mark W Ochs DMD MD who is a Professor of Oral Maxillofacial Surgery and a Special Assistant to the Dean for Community Engagement and Global Affairs at University of Pittsburgh's School of Dental Medicine. The current version of this chapter is an updated form of Dr. Ochs's work.

References

1. Rontal E, Rontal M, Guilford FT. Surgical anatomy of the orbit. Ann Otol Rhinol Laryngol. 1979;88(3 Pt 1):382–6.
2. Waitzman AA, Posnick JC, Armstrong DC, Pron GE. Craniofacial skeletal measurements based on computed tomography. Part II. Normal values and growth trends. Cleft Palate Craniofac J. 1992;2:118–28.
3. Ochs MW, Buckley MJ. Anatomy of the orbit. Oral Maxillofac Surg Clin North Am. 1993;5:419–29.
4. Frenkel REP, Spoor TC. Neuro-ophthalmologic manifestations in trauma. In: Spoor TC, Nesi FA, editors. Management of ocular, orbital, and adnexal trauma. New York: Raven Press; 1988. p. 195–245.
5. Rootman J. Basic anatomic considerations. In: Rootman J, editor. Diseases of the orbit. Philadelphia: JB Lippincott; 1988. p. 3–18.
6. Hollinshead WH. The head and neck. 3rd ed. Philadelphia: Harper and Rowe; 1982. p. 93–155.
7. Som PM, Shugar JM, Brandwein MS. Anatomy and physiology of the sinonasal cavities. In: Som PM, Curtin HD, editors. Head and neck imaging. St. Louis: Mosby; 2003. p. 87–147.
8. Zide BM, Jelks GW. Surgical anatomy of the orbit. New York: Raven Press; 1985.
9. Bergin DJ. Anatomy of the eyelids, lacrimal system, and orbit. In: McCord Jr CD, Tanenbaum M, editors. Oculoplastic surgery. 2nd ed. New York: Raven Press; 1987. p. 41–71.
10. Hart WM Jr. The eyelids. In: Hart Jr WM, editor. Adler's physiology of the eye. 9th ed. St. Louis: Mosby; 1992. p. 1–16.
11. Nakamura T, Gross C. Facial fractures: analysis of five years of experience. Arch Otolaryngol. 1973;97:288–90.
12. Gwyn PP, Carraway JH, Horton CE, et al. Facial fractures—associated injuries and complications. Plast Reconstr Surg. 1971;47:225–30.
13. Ellis E III. Fractures of the zygomatic complex and arch. In: Fonseca RJ, Walker RF, editors. Oral and maxillofacial trauma, vol. 1. Philadelphia: WB Saunders; 1991. p. 435–514.
14. Hammer B. Orbital fractures, diagnosis, operative treatment and secondary corrections. Gottingehn (Germany): Hogrefe and Huber; 1995. p. 10–1.
15. Kelly JK, Lazo A, Metes JJ. Radiology of orbital trauma. In: Spoor TC, Nesi FA, editors. Management of ocular, orbital and adnexal trauma. New York: Raven Press; 1988. p. 247–68.
16. Unger JM. Orbital apex fractures: the contribution of computed tomography. Radiology. 1984;150:713–7.
17. Guyon JJ, Brant-Zawadzki M, Seiff SR. CT demonstration of optic canal fractures. AJR Am J Roentgenol. 1984;143:1031–4.
18. Lindahl S. Computed tomography of intraorbital foreign bodies. Acta Radiol. 1987;28:235–40.
19. Gillespie JE, Isherwood L, Barker GR. Three dimensional reformations of computed tomography in the assessment of facial trauma. Clin Radiol. 1987;38:523–6.
20. Roberts CF, Leehey PJ III. Intra-orbital wood foreign bodies mimicking air at CT. Radiology. 1992;185:507–8.
21. Kelly WM, Paglen PG, Pearson JA, et al. Ferromagnetism of intraocular foreign body causes unilateral blindness after MR study. AJNR Am J Neuroradiol. 1986;7:243–5.
22. Otto PM, Otto RA, Virapongse C, et al. Screening test for detection of metallic foreign objects in the orbit before magnetic resonance imaging. Invest Radiol. 1992;27:308–11.

23. Sprecht CS, Varga JH, Jalai MM, Edelstein JP. Orbitocranial wooden foreign body diagnosed by magnetic resonance imaging: dry wood can be isodense with air and orbital fat by computed tomography. Surv Ophthalmol. 1992;36:341–4.
24. Byrne SF, Green RL. Trauma and periodontal disease. In: Byrne SF, Green RL, editors. Ultrasound of the eye and orbit. St. Louis: Mosby Year Book; 1992. p. 431–61.
25. Reshef DS, Osoinig KC, Nerad JA. Diagnosis and intraoperative localization of a deep orbital organic foreign body. Orbit. 1987;6:3–15.
26. Berges O. Color Doppler flow imaging of the orbital veins. Acta Ophthalmol. 1992;204:55–8.
27. Zizelmann C, Gellrich NC, Metzger MC, Schoen R, Schmelzeisen R, Schramm A. Computer-assisted reconstruction of orbital floor based on cone beam tomography. Br J Oral Maxillofac Surg. 2007;45:79–80.
28. McCoy FJ. Applications to new advances to treatment of facial trauma. Ann Plast Surg. 1986;17:354–5.
29. Tschanz A, Hammer B, Prein J. Visusverlust bei verletzungen der orbita [unpublished medical thesis]. Basel (Switzerland): University Hospital; 1994.
30. Katz B, Herschler J, Brich DC. Orbital hemorrhage and prolonged blindness: a treatable posterior optic neuropathy. Br J Ophthalmol. 1983;67:549–53.
31. Kersten RC, Rice CD. Subperiosteal orbital hematoma: visual recovery following delayed drainage. Ophthalmic Surg. 1987;18:423–7.
32. Ord RA, El Attar H. Acute retrobulbar hemorrhage complicating a malar fracture. J Oral Maxillofac Surg. 1982;40:234–6.
33. Damico FM, Kiss S, Young LH. Sympathetic ophthalmia. Semin Ophthalmol. 2005;20(3):191–7.
34. Ahn BH, Baek NH, Shin DH. Management of traumatic hyphema. In: Spoor TC, Nesi FA, editors. Management of ocular, orbital, and adnexal trauma. New York: Raven Press; 1988. p. 69–80.
35. Hammer ME, Grizzard WS. Management of retinal and vitreous injuries. In: Spoor TC, Nesi FA, editors. Management of ocular, orbital, and adnexal trauma. New York: Raven Press; 1988. p. 81–128.
36. Galetta SL, Leahey A, Nichols CW, Raps EC. Orbital ischemia, ophthalmoparesis, and carotid dissection. J Clin Neuroophthalmol. 1991;11(4):284–7.
37. Seiff SR. High-dose corticosteroids for treatment of vision loss due to indirect injury to the optic nerve. Ophthalmic Surg. 1990;21:389–95.
38. Spoor TC, Hartel WC, Lensink DB, Wilkinson MJ. Treatment of traumatic optic neuropathy with corticosteroids. Am J Ophthalmol. 1990;110:665–9.
39. Mauriello JA, DeLuca J, Krieger A, et al. Management of traumatic optic neuropathy—a study of 23 patients. Br J Ophthalmol. 1992;76:349–52.
40. Fujino T, Makino K. Entrapment mechanisms and ocular injury in orbital blow-out fractures. Plast Reconstr Surg. 1980;65:571–6.
41. Ebrahimi A, Motamedi MHK, Rasouli HR, Nagdi N. Enopthalmos and orbital volume changes in zygomaticomaxillary complex fractures: is there a correlation between them? J Oral Maxillofac Surg. 2018;77:134e1–9.
42. Woernley T, Wright T, Lam DN, Jundt J. Oculocardiac reflex in an orbital fracture without entrapment. J Oral Maxillofac Surg. 2017;75:1716–21.
43. Smith B, Regan W. Blowout fractures of the orbit. Am J Ophthalmol. 1957;44:733–9.
44. Converse JM, Smith B. Blowout fractures of the orbit. Trans Am Acad Ophthalmol Otolaryngol. 1960;64:676–88.
45. Crikelair G, Rein J, Potter G. A critical look at the blowout fracture. Plast Reconstr Surg. 1972;49:374–9.
46. Nicholoson D, Guzak S. Visual loss complicating repair of orbital floor fractures. Arch Ophthalmol. 1971;86:369–76.
47. Putterman AM, Stevens T, Urist MJ. Nonsurgical management of blowout fractures of the orbital floor. Am J Ophthalmol. 1974;77:232–8.
48. Dulley B, Fells P. Orbital blowout fractures. Br Orthoped J. 1974;31:47–54.
49. Crumley R, Leibsahn J, Krause C, Burton T. Fractures of the orbital floor. Laryngoscope. 1976;87:934–47.
50. Converse JM, Smith B. Editorial on the treatment of blowout fractures of the orbit. Plast Reconstr Surg. 1978;62:100–4.
51. Wilkins RB, Havins WE. Current treatment of blowout fractures. Ophthalmology. 1982;89:464–6.
52. Koorneef L. Current concepts on the management of orbital blowout fractures. Ann Plast Surg. 1982;9:185–200.
53. Smith B, Lisman RD, Simonton J, DellaRocca R. Volkmann's contracture of the extraocular muscles following blowout fractures. Plast Reconstr Surg. 1984;74:200–16.
54. Hawes M, Dortzbach RL. Surgery on orbital floor fractures: influence of time on repair and fracture size. Ophthalmology. 1983;90:1066–70.
55. deMann K. Fractures of the orbital floor: indications for exploration and for the use of a floor implant. J Oral Maxillofac Surg. 1984;12:73–7.
56. Dortzbach R, Elner V. Which orbital floor blowout fractures need surgery [editorial]? Adv Ophthalmic Plast Reconstr Surg. 1987;6:287–9.
57. Dutton JJ, Manson P, Putterman A. Management of blowout fractures of the orbital floor [editorial]. Surv Ophthalmol. 1991;35:279–80.
58. Parsons GS, Mathog RH. Orbital wall and volume relationships. Arch Otolaryngol Head Neck Surg. 1988;114:743–7.
59. Roncevic R, Malinger B. Experience with various procedures in the treatment of orbital floor fractures. J Oral Maxillofac Surg. 1981;9:81–4.
60. Zingg M, Chowdhury K, Ladrach K. Treatment of 813 zygoma-lateral orbital complex fractures. Arch Otolaryngol Head Neck Surg. 1991;11:611–20.
61. Thaller S, Yvorchuk W. Exploration of the orbital floor: an indicated procedure? J Craniomaxillofac Surg. 1990;1:187–90.
62. Sacks A, Friedland J. Orbital floor fractures: should they be explored early? Plast Reconstr Surg. 1979;64:190–3.
63. deMann K, Hes WJ, deJong PT, Wijingaarde R. Influence of age on the management of blowout fractures of the orbital floor. Int J Oral Maxillofac Surg. 1991;20:330–6.
64. Manson PN, Clifford CM, Su CT, Iliff NT. Mechanisms of global support and posttraumatic enophthalmos. I. The anatomy of the ligament sling and its relation to intramuscular cone orbital fat. Plast Reconstr Surg. 1986;77:193–202.
65. Converse JM. Two plastic operations for repair of the orbit following severe trauma and extensive comminuted fracture. Arch Ophthalmol. 1944;31:323–5.
66. Wray RC, Holtman BN, Rebaudo JM, et al. A comparison of conjunctival and subciliary incisions for orbital fractures. Br J Plast Surg. 1983;10:309–13.
67. Heckler F, Songcharoen S. Subciliary incision and skin-muscle eyelid flap for orbital fractures. Ann Plast Surg. 1983;10:309–13.
68. Pospisil OA, Fernando TD. Review of the blepharoplasty incisions as a surgical approach to zygomatic orbital fractures. Br J Oral Maxillofac Surg. 1984;22:261–8.
69. Tessier P. The conjunctival approach to the orbital floor and maxilla in congenital malformation and trauma. J Oral Maxillofac Surg. 1973;1:3–8.

70. Converse JM, Firmin F, Wood-Smith D, Friedland J. The conjunctival approach in orbital floor fractures. Plast Reconstr Surg. 1973;52:656–7.
71. Ochs MW. Use of preseptal transconjunctival approach in orbital reconstruction surgery [discussion]. J Oral Maxillofac Surg. 2001;59:291–2.
72. Manson PN, Ruas E. Single eyelid incision for exposure of the zygomatic bone and orbital reconstruction. Plast Reconstr Surg. 1987;79:120–6.
73. McCord C, Moses J. Exposure of the inferior orbit with fornix incision and lateral can-thotomy. Ophthalmic Surg. 1979;10: 53–63.
74. Riu GD, Mloni SM, Gobbi D, Soma A, Baj A, Tullio A. Subciliary versus swinging eyelid approach to the orbital floor. J Craniomaxillfac Surg. 2008;36:439–42.
75. Appling WD, Patrinely JR, Salzer TA. Transconjunctival approach vs subciliary skin-muscle flap approach for orbital repair. Arch Otolaryngol Hed Neck Surg. 1993;119:1000–7.
76. Emam HA, Stevens MR, Larsen PE, Jatana CA. Lateral tarsotomy: a practical alternative to lateral canthotomy to increase orbital access. Oral Surg Oral Med Oral Pathol Oral Radiol. 2016;122:e1–4.
77. Ellis E III, Zide MF, editors. Coronal approach. In: Surgical approaches to the facial skeleton. Philadelphia: Williams and Wilkins; 1995. p. 63–94.
78. Stuzin JM, Wagstron L, Kawamoto H, et al. Anatomy of the frontal branch of the facial nerve: the significance of the temporal fat pad. Plast Reconstr Surg. 1989;83:265–71.
79. Al-Kayat A, Bramley P. A modified preauricular approach to the temporomandibular joint and malar arch. Br J Oral Surg. 1979;17:91–103.
80. Shorr N, Baylis HI, Goldberg RA, Perry JD. Transcaruncular approach to the medial orbit and orbital apex. Ophthalmology. 2000;107:1459–63.
81. Balch KC, Goldberg RA, Green JP, Shorr N. The transcaruncular approach to the medial orbit and ethmoid sinus: a cosmetically superior option to the cutaneous (Lynch) incision. Facial Plast Surg Clin North Am. 1998;6(1):171–8.
82. Fernandes R, Fattahi T, Steinberg B, Schare H. Endoscopic repair of isolated orbital floor fracture with implant placement. J Oral Maxillofac Surg. 2007;65(8):1449–53.
83. Jackson BF. Orbital trauma, bradycardia, and vomiting: trapdoor fracture and the oculocardiac reflex: a case report. Pediatr Emerg Care. 2010;26(2):143–5.
84. Rubin L. Biomaterials in reconstructive surgery. St. Louis: CV Mosby; 1983.
85. Berghaus A. Porous polyethylene in reconstructive head and neck surgery. Arch Otolaryngol Head Neck Surg. 1985;111: 154–60.
86. Ilizuka T, Mikkonen P, Paukku P, Lindqvist C. Reconstruction of orbital floor with poly-dioxanone plate. Int J Oral Maxillofac Surg. 1991;20:83–7.
87. Loftfield K, Jordan DR, Fowler J, Anderson RL. Orbital cyst formation associated with Gelfilm use. Ophthal Plast Reconstr Surg. 1987;3:187–91.
88. Rubin PA, Shore JW, Yaremchuk MJ. Complex orbital fracture repair using rigid fixation of the internal orbital skeleton. Ophthalmology. 1999;99:553–9.
89. Ilankovan V, Jackson T. Experience in the use of calvarial bone grafts in orbital reconstruction. Br J Oral Maxillofac Surg. 1992;30:92–6.
90. Gruss JS, MacKinnon SE. The role of primary bone grafting in complex craniomaxillofacial trauma. Plast Reconstr Surg. 1985;75:17–24.
91. Cestero HJ, Salyes KE, Toranto IR. Bone growth into porous carbon, polyethylene, and polypropylene prostheses. J Biomed Mater Res. 1975;9:1–7.
92. Whitehouse RW, Batterbury M, Jackson A, Noble JL. Prediction of enophthalmos by computed tomography after "blowout" orbital fracture. Br J Ophthalmol. 1994;78:618–20.
93. Bly RA, Chang SH, Cudejkova M, Liu J, Moe KS. Computer-guided orbital reconstruction to improve outcomes. JAMA Facial Plast Surg. 2013;15(2):113–20.
94. Nazimi AJ, Khoo SC, Nabil S, Nordin R, Lan TH, Rajandram RK, Rajaran JR. Intraoperative computed tomography scan for orbital fracture reconstruction. J Craniofac Surg; 2019. Epub.
95. Borad V, Lacey MS, Hamlar DD, Dresner HS, Yadava GK, Schubert W. Intraoperative imaging changes management in orbital fracture repair. J Oral Maxillofac Surg. 2017;75(9): 1932–40.
96. Ochs MW, Johns FR. Orbital trauma. In: Fonseca RJ, Marciani RD, Hendler BH, editors. Oral and maxillofacial surgery: trauma, vol. 3. Philadelphia (PA): W.B. Saunders; 2000. p. 207.
97. Ochs MW, Johns FR. Orbital trauma. In: Fonseca RJ, Marciani RD, Hendler BH, editors. Oral and maxillofacial surgery: trauma, vol. 3. Philadelphia (PA): W.B. Saunders; 2000. p. 208.