Evidence-Based Medicine: Orbital Floor Fractures

Michael S. Gart, M.D.
Arun K. Gosain, M.D.

Chicago, Ill.

Learning Objectives: After studying this article, the participant should be able to: 1. Explain the epidemiology, anatomy, and pathophysiology of orbital floor fractures. 2. Select the optimal timing of—and understand the indications for—operative repair of orbital floor fractures. 3. List advantages and disadvantages of the surgical approaches and materials available for orbital floor reconstruction. 4. Identify special considerations in treating pediatric patients presenting with orbital floor fractures.

Summary: This maintenance of certification module reviews the anatomy, pathophysiology, diagnosis, and management of orbital floor fractures in addition to special considerations for pediatric patients. The Appendix shows the evidence rating scale used for the literature review in creating this maintenance of certification article. (*Plast. Reconstr. Surg.* 134: 1345, 2014.)

ANATOMY AND PATHOPHYSIOLOGY

The orbit is a bony pyramid bounded by the roof, floor, and medial and lateral walls; the orbital aperture forms the base of the pyramid.[1] The orbital floor, which forms the roof of the maxillary sinus, slopes upward toward the apex of the pyramid, which lies roughly 44 to 50 mm posterior to the orbital entrance. The total volume of the bony orbit is approximately 30 ml, of which the globe occupies 7 ml; these volumes vary slightly with sex and race.[1,2]

The bony orbit protects the globe and is involved in the majority of midfacial fractures.[3] Fractures of the orbital rim and floor commonly occur in the presence of zygomaticomaxillary complex fractures. When the orbital floor is involved, this is often referred to as a "blowout" fracture. Most often, the orbital floor is fractured in conjunction with the inferior orbital rim ("impure" blowout fracture), but "pure" orbital floor fractures can be seen in 22 to 47 percent of orbital injuries.[4,5] The following are key presentations of orbital floor fractures.

Blowout Fractures

"Pure" orbital floor fractures were first described by Lang in 1889.[6] In 1901, Rene Le Fort concluded that blowout fractures occurred through force transmission from the more rigid infraorbital rim to the relatively weak orbital floor, known as the "buckling" theory.[7–9] It was not until 1948 that this contention was challenged by Pfeiffer, who observed a case series of globe-directed trauma resulting in blowout fractures, leading him to propose the "hydraulic" theory, which states that hydraulic pressure from the globe is transmitted to the bony orbit, resulting in fracture of the thin orbital floor.[10]

Initial attempts to prove one theory or the other were flawed in their experimental design.[11–20] More recent efforts have shown that both mechanisms produce orbital blowout fractures, but with different characteristics.[21,22] Buckling tends to produce smaller, linear fractures along the anterior orbital floor, with little or no periorbital herniation and a lower likelihood of clinical enophthalmos.[21–23] In contrast, the hydraulic mechanism tends to produce larger, more posterior fractures of both the floor and medial wall, with frequent herniation and a higher likelihood of enophthalmos.[21–23] When these two mechanisms combine, the resulting fracture is significantly larger than with either mechanism acting independently.[23]

Trapdoor Fractures

A "trapdoor" orbital fracture is a pure orbital floor fracture, where a bony fragment, often hinged medially, is transiently displaced inferiorly, allowing herniation of orbital contents into

From the Division of Pediatric Plastic Surgery, Lurie Children's Hospital of Northwestern University Feinberg School of Medicine.
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the maxillary sinus, which are then entrapped as the bony fragment returns toward its initial position[24–26] (Fig. 1). Extraocular movements should be evaluated to assess for extraocular muscle entrapment. If present, there will usually be restriction in upward gaze caused by entrapment or herniation of the periorbital soft tissues through an orbital floor defect. The inferior oblique and rectus are the most commonly entrapped muscles. Entrapment of these muscles causes restriction of upward gaze and diplopia, although downgaze or no restriction may also be seen.[24] In the unconscious or uncooperative patient, extraocular muscle entrapment can be evaluated using a forced duction test, where the examiner uses a forceps to grasp the conjunctiva at or near the attachment of the inferior rectus muscle and attempts to move the globe through a full range of motion.

Superior Orbital Fissure and Orbital Apex Syndromes

The superior orbital fissure is a bony hiatus near the orbital apex that transmits structures from the middle cranial fossa to the orbit, including the oculomotor, trochlear, and abducens nerves; the ophthalmic division of the trigeminal nerve (V_1); and the superior and inferior ophthalmic veins.[27–29] Fractures involving the superior orbital fissure, although rare, can cause paralysis of these nerves, resulting in upper eyelid ptosis, from loss of tone in the Müller muscle and/or levator palpebrae superioris[29,30]; proptosis of the globe caused by loss of normal retractile pull of the extraocular muscles and/or obstruction of the ophthalmic veins; ophthalmoplegia caused by loss of extraocular muscle function; fixed dilation and loss of accommodation of the pupil from interrupted parasympathetic innervation of the pupillary ciliary muscle; and sensory loss of the forehead and upper eyelid from the involvement of the ophthalmic division of the trigeminal nerve.[27–32] Similarly, tumors, expanding hematomas, aneurysms, or other space-occupying lesions can cause compressive neuropathies with similar findings.

According to Kurzer and Patel, this constellation of symptoms, known as superior orbital fissure syndrome, was first described in 1858 by Hirschfield.[32] When superior orbital fissure syndrome occurs in combination with ipsilateral blindness, involvement of the optic canal, which transmits the optic nerve and ophthalmic artery through the greater wing of the sphenoid, has occurred. Blindness in conjunction with superior orbital fissure syndrome is known as orbital apex syndrome, first described by Kjoer in 1945.[32]

As these entities are rare, there are no consensus recommendations for treatment, although recent trends have favored the use of high-dose corticosteroids with or without operative intervention.[33–37] Treatment is directed at the underlying cause and individualized for each patient based on likely causes.[38] Interested readers are directed to the references list for more in-depth review.

HISTORY AND PHYSICAL EXAMINATION

Common signs and symptoms of orbital floor fractures include localized pain, diplopia and ecchymosis of the periorbita, eyelid edema, subconjunctival hemorrhage, and sensory deficits in the inferior orbital nerve distribution. The physical examination should begin with inspection of the orbit and periorbital tissues. Any lacerations or bony stepoffs are noted, and the patient should be assessed for enophthalmos and/or hypoglobus. Enophthalmos, the posterior displacement of

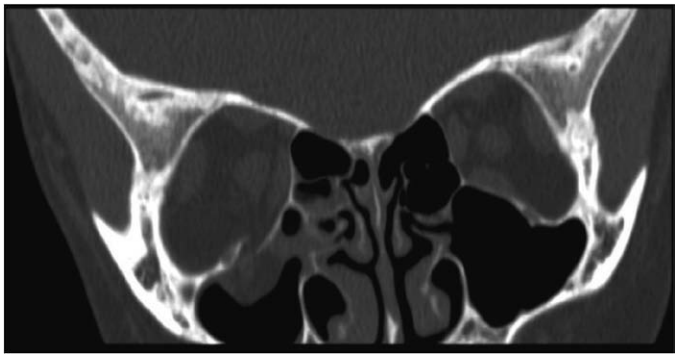

Fig. 1. Coronal computed tomographic section showing right orbital trapdoor fracture with displacement of inferior rectus muscle through fracture. Note the rounded shape (increased height-to-width ratio) of the muscle belly compared with the unaffected eye.

the globe along the anteroposterior axis, is clinically detectable at 2 mm. Hypoglobus occurs when the entire globe is displaced inferiorly, often as a result of trauma to the orbital floor, and is a cause of pseudostrabismus, where the visual axes of both eyes remain aligned despite vertical asymmetry of the globes. This is in contrast to hypertropia and hypotropia, where the visual axes of the focusing eye are higher or lower than the contralateral eye, respectively, leading to a true strabismus.

An increase in orbital volume due to blowout of the orbital floor causes relaxation of the soft tissues into the enlarged bony space. A 5 percent increase in total volume is enough to result in clinically significant enophthalmos.[39–42] If the lateral orbital rims are intact, accurate measures of globe projection can be obtained using a Hertel exophthalmometer, which measures the sagittal position of the globe in relation to the unaffected eye. Hypoglobus can be detected through careful evaluation of a light reflex centered on each pupil revealing vertical asymmetry between the eyes.

As mentioned above, extraocular movements can be evaluated in the unconscious or uncooperative patient with forced duction testing. Because of potential for significant discomfort, this test should be performed under sedation, local anesthesia, or general anesthesia.

Visual acuity assessment is critical in evaluating the orbital trauma patient. Gross vision, visual acuity, and baseline acuity should be evaluated and documented. Color perception is useful in evaluating the status of the optic nerve, as loss of color saturation, most noticeable in reds, is one of the earliest signs of traumatic optic neuropathy.

Pupillary examination should evaluate pupil size, symmetry, and response to light shined directly in each eye, and the response of each pupil to light stimulation of the contralateral eye. The absence of consensual pupillary constriction with light in the contralateral eye can signify injury to the autonomic fibers carried by the oculomotor nerve. One should also check for the presence of an afferent pupillary defect, signaling an injury to the optic nerve, with reduced or absent constriction bilaterally resulting from diminished light perception in the affected eye. Examination will demonstrate paradoxical dilation of the unaffected eye when swinging a light from the unaffected to the affected eye (Fig. 2).

Radiographic Evaluation

Advances in computed tomography technology have made coronal and sagittal reconstructions from axial scans readily available. Coronal images with 1- to 2-mm sections remain the most useful method for assessing orbital floor fractures; however, sagittal reconstructions can be particularly helpful in determining the premorbid shape of the orbit before attempted reconstruction.[43] Computed tomographic scanning provides reliable information on the size of the defect and status of the globe and extraocular muscles, and evidence of entrapment.

OPERATIVE MANAGEMENT

The most important factors to consider when deciding the ideal management of a patient presenting with an orbital blowout fracture are (1) enophthalmos (2) ocular motility, and (3) radiographic findings.

IMMEDIATE REPAIR

Although indications for repair remain controversial,[43–45] there are several clinical findings that warrant urgent or immediate surgical exploration. In most studies, immediate repair is defined as repair occurring within 24 or 48 hours, and should be performed for patients with early enophthalmos greater than 2 mm; defects of the orbital floor or combined floor/medial wall defects larger than 2 cm^2, which are likely to result in delayed enophthalmos; pediatric trapdoor ("white-eyed") fractures; and when computed tomographic evidence of entrapment is associated with symptomatic diplopia, gaze restriction, or nonresolving oculocardiac reflex. These factors have all been associated with improved outcomes, including late enophthalmos and persistent diplopia, when surgery was performed in the first 1 to 2 days.[44]

Oculocardiac Reflex

The oculocardiac reflex is caused by pressure on the globe or entrapment of periorbital soft tissues, and triggers bradycardia, including possible junctional rhythm or asystole, and nausea and vomiting. This reflex is thought to be caused by an increase in vagal tone, with afferent signal being carried by the ophthalmic division of the trigeminal nerve by means of the ciliary ganglion, and the vagus nerve carrying the efferent signals to the heart and stomach.[46] Nonresolution of these symptoms can be fatal; if severe, they warrant immediate surgical exploration of orbital floor fractures to reduce incarcerated periorbital tissues.

Muscular Entrapment

The longer a muscle remains entrapped, the higher the incidence of persistent postoperative diplopia,[24,47,48] and many studies have shown that

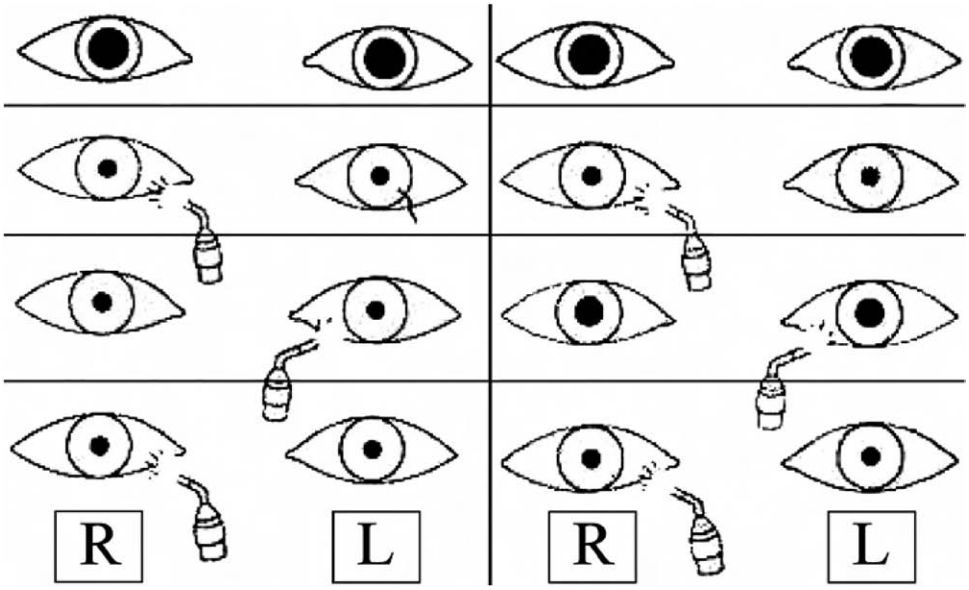

Fig. 2. Drawings depicting normal (*left*) and abnormal (*right*) pupillary reactions to light. A bright light shined into the right eye causes equal pupillary constriction. A bright light shined into the left eye causes equal pupillary constriction and the same degree of constriction as obtained by shining the bright light into the right eye. Swinging the bright light back and forth between the eyes after a 2- to 3-second pause confirms that both pupils are equally constricted without changing size, regardless of which eye the light is shined in. (*Right*) A bright light shined into the right eye causes good pupillary constriction in both eyes. The same light shined into the left eye also causes some pupillary constriction, but less than that obtained from shining the light in the right eye. When swinging the light back and forth between the two eyes, it is often easiest to simply watch one eye. An abnormality in ocular function in one eye will be discernible by observing either eye. In the drawings on the right, it is the left eye or optic nerve that is transmitting less light. (Reprinted from Soparkar CN and Patrinely JR. The eye examination in facial trauma for the plastic surgeon. *Plast Reconstr Surg*. 2007;120(Suppl 2):49S–56S.)

fracture repair within 48 hours of injury significantly reduces this risk.[44,49–52] Persistent diplopia following release of entrapped extraocular muscles is thought to be myogenic (caused by muscle ischemia and fibrosis) or neurogenic (caused by traumatic neuropathy of nerves innervating the extraocular muscles); both factors are likely to contribute to diplopia. When severe, motility surgery must be performed for correction of persistent diplopia.

Enophthalmos

Several studies have shown that an increase in orbital soft-tissue volume of roughly 5 percent will result in clinically detectable enophthalmos if the orbital floor is not anatomically reconstructed.[39–42] Many surgeons continue to rely on size of the defect, with most surgeons operating for fractures larger than 1 to 2 cm^2 or a defect greater than 50 percent of the orbital floor[53,54] (Fig. 3). In one study, fracture size was a strong or very strong influence to operate for 87 percent of surgeons.[53]

Similarly, several authors have described rounding of the inferior rectus muscle associated with orbital blowout fractures. The inferior rectus muscle normally assumes a flattened

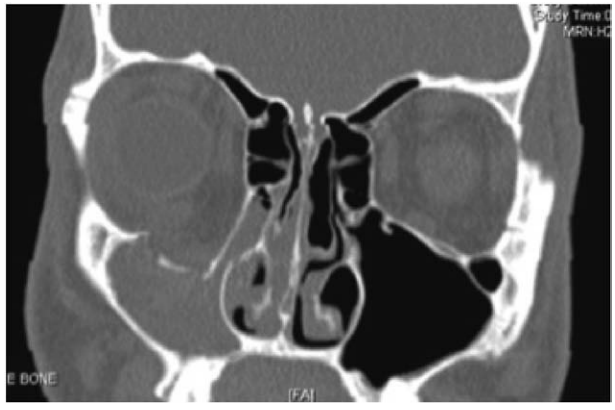

Fig. 3. Large right orbital floor/medial wall defect without evidence of periorbital entrapment.

oval appearance in cross-section, with the long axis oriented transversely (i.e., a height-to-width ratio of <1). Distortion in this shape can occur with orbital floor fractures and has been attributed to intramuscular edema, hemorrhage, or loss of soft-tissue support for the periorbita. This finding has been shown to be predictive of postoperative enophthalmos.[55,56] Moreover, Matic et al. have shown that a height-to-width ratio greater than 1.00 in the inferior rectus muscle is predictive of late enophthalmos but not persistent diplopia.[57]

DELAYED REPAIR (WITHIN 2 WEEKS)

Patients who present without findings that necessitate immediate repair should be seen at 2-week follow-up to evaluate for progressive or nonresolving symptoms, including progressive infraorbital nerve hypesthesia, diplopia, and delayed enophthalmos.[44]

Infraorbital Hypesthesia

Progressive V_2 hypesthesia may be indicative of nerve compression, and one report suggests that operative intervention may improve symptoms,[58] although the evidence is limited to case reports. The risks of surgery should be discussed with patients and weighed carefully against the potential for sensory recovery.

Diplopia

Diplopia is common following orbital floor fractures, and is usually caused by simple muscle contusion and/or edema. Diplopia related to periorbital edema should show signs of resolution by 2 weeks after injury. However, prolonged diplopia may be caused by muscle hemorrhage, edema, or motor nerve palsy that may improve with time.[44] Here, correlation of radiographic and examination findings is key. Persistent diplopia in the central 30 degrees of gaze that is symptomatic and/or associated with evidence of potential soft-tissue entrapment by computed tomographic scan or a positive forced duction test should prompt surgical exploration and repair.[44]

Delayed Enophthalmos

One argument for delayed operative treatment of orbital fractures is that it allows for resolution of traumatic edema before reconstruction. Resolution of edema may reveal enophthalmos or hypoglobus not appreciated on initial examination, and development of these signs within the first 6 weeks after injury should be considered a strong relative indication for repair.[43,59] As in the acute setting, enophthalmos greater than 2 mm in the anteroposterior direction is considered clinically significant and can be considered an indication for late repair.

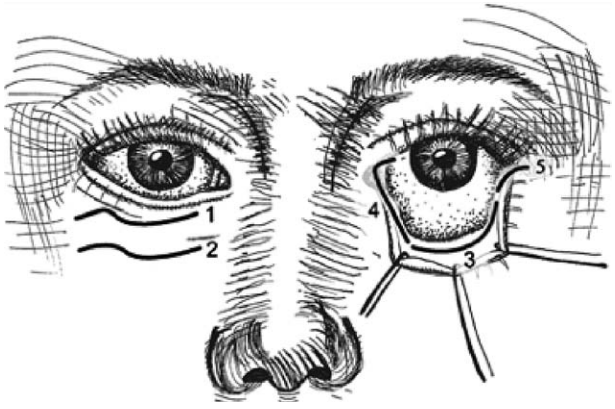

Fig. 4. Frontal view of incisions for orbital floor exploration. *1*, subtarsal; *2*, infraorbital; *3*, transconjunctival; *4*, transcaruncular; *5*, transconjunctival with lateral skin extension. (Reprinted from Kothari, NA, Avashia YJ , Lemelman BT, Mir HS, and Thaller SR. Incisions for orbital floor exploration. *J Craniofac Surg*. 2012;23(Suppl 1):1985–1989.)

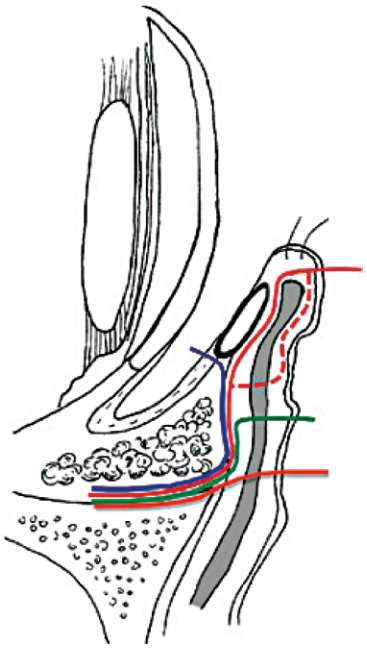

Fig. 5. Cross-sectional view of incisions for orbital floor exploration. *Solid red*, nonstepped subciliary; *dotted red*, stepped subciliary; *blue*, transconjunctival; *green*, subtarsal; *orange*, infraorbital. (Reprinted from Kothari, NA, Avashia YJ , Lemelman BT, Mir HS, and Thaller SR. Incisions for orbital floor exploration. *J Craniofac Surg*. 2012;23(Suppl 1):1985–1989.)

METHODS OF REPAIR

Incision Patterns

There are several approaches described to access the orbital floor (Figs. 4 and 5). Many have abandoned the subciliary incision, which has demonstrated an unacceptably high risk of cicatricial ectropion.[60] The transconjunctival approach has been most extensively studied,[60] and shows low rates of complications and leaves no visible scar; however, this approach often requires lateral canthotomy for complete exposure and there is a small risk of cicatricial entropion with this incision pattern. Furthermore, a higher incidence of ectropion was found in patients with previous external eyelid incisions.[61] The subtarsal incision offers direct orbital floor access and is less technically demanding, but may leave visible scarring.[60]

A recent comprehensive review of incision techniques found insufficient high-level evidence to suggest one pattern over another, but did show a low incidence of complications with transconjunctival approaches, the highest rate of complications and revisions in subciliary approaches, and the lowest revision rate with subtarsal incisions.[60] It should be noted that the transcaruncular incision pattern may be associated with increased ophthalmic complications, including nasolacrimal obstruction caused by scar tissue formation.[62] Although the rates of reported complications are low and this incision pattern is gaining in popularity, it is not currently in widespread use.

Floor Reconstruction

Once the orbital contents have been reduced, the orbital floor can be reconstructed using a variety of implant materials (Table 1). Biological materials offer the potential advantages of better biocompatibility, but come at the cost of donor-site morbidity. Conversely, synthetic grafts have historically been associated with higher rates of implant-related complications, including infection and extrusion, with the advantages of being readily available and without morbidity. Although the actual rates of these complications are quite low, and may be more attributable to the state of the orbital soft tissues at the time of repair, fear of implant-related complications continues to factor into clinical practice.

Biological Materials

Various biological materials are available for reconstruction of orbital floor defects, including autologous bone and cartilage grafts; bone and dural allografts; and porcine collagen or dermal xenografts. Autologous bone remains the criterion standard for orbital floor reconstruction because of its availability, rigidity, biocompatibility, and minimal immune activity.[63–65] However, autologous bone grafts are associated with donor-site morbidity that limits their utility.[65–68]

Alloplastic Materials

Alloplasts are available as resorbable or nonresorbable plates, each with their own distinct advantages and disadvantages.

Resorbable

Resorbable alloplasts, composed of poly-L-lactic acid, polyglycolic acid, polydioxanone, or composite (poly-L-lactic acid/polyglycolic acid) polymers, are readily available and able to offer long-term support to allow bony healing[69–72]; however, they may be associated with delayed enophthalmos and/or intense inflammation as the implant degrades.[72–74] As discussed below, these materials are particularly attractive for pediatric orbital floor reconstruction because of concerns over growth restriction with permanent alloplasts.

Permanent

Permanent alloplasts offer long-term rigid support for orbital floor reconstruction, but have a higher risk of implant-associated infections. Porous polyethylene is easy to mold and adapt and allows rigid fixation and vascular ingrowth; however, it may form adhesions to exposed extraocular muscles.[75–78] Titanium mesh implants, in contrast, are biocompatible and easy to contour, but are not easy to place, especially with deep orbital fractures, as the plate edges often catch on periorbital tissues. Also, titanium can be associated with intense fibrosis, making secondary surgery a challenge.[78–81] Newer materials, consisting of titanium mesh coated with porous polyethylene, are available and aim to capture the strengths of both materials. A recent survey of practicing plastic surgeons found that porous polyethylene/titanium and titanium mesh were the two most commonly used materials for orbital floor reconstruction.[53]

Other

Silicone sheeting has historically been used for repair of orbital floor defects and continues to be used routinely in many parts of the world. In the United States, silicone has largely been abandoned because of reports of implant extrusion and relatively high rates of implant removal compared with other materials.[82,83] It should be noted, however, that the true incidence of such

Table 1. Materials Used for Orbital Floor Reconstruction

Autogenous material
Bone
Cartilage
Temporalis fascia
Allogenic material
Lyophilized dura mater
Lyophilized cartilage
Irradiated fascia lata
Alloplastic material
Resorbable
PLLA plate
P(L/DL)LA 70/30 plate
PLLA/PGA sheet
PDS sheet
Polygalactin-910
Polygalactin-910/PDS
Xenografts
Porcine collagen
Porcine dermis
Permanent
Titanium mesh
PPE sheet
Titanium/PPE
Hydroxyapatite sheet

PLLA, poly-L-lactic acid; PGA, polyglycolic acid; P(L/DL)LA 70/30 plate, poly-L/DL-lactide; PDS, polydioxanone; PPE, porous polyethylene.

complications is likely unknown, as evidence is limited, antibiotic regimens have changed significantly since silicone was routinely used for orbital floor repair, and implant-related complications such as extrusion may present up to 20 years or more postoperatively.[82,83]

Teflon implants are also available, and have been shown to have a low incidence of infectious complications; however, there have been reports of delayed hemorrhagic complications with these implants.[84,85] Lastly, nylon and bioactive glass implants have been described with promising results but are not currently in widespread use.[86–88]

A recent systematic review evaluating materials for orbital floor reconstruction found that there is no conclusive evidence to suggest one material as "better" than another; rather, the surgeon must rely on his or her own experiences and the unique characteristics of each material to individualize treatment plans.[63] Regardless of the material chosen, forced duction testing should be performed after orbital floor reconstruction to rule out iatrogenic entrapment before leaving the operating room.

SPECIAL CONSIDERATIONS IN PEDIATRIC ORBITAL FLOOR FRACTURES

Although facial fractures in general are less common in children,[24,89] orbital fractures represent up to 45 percent of all pediatric facial fractures and may differ in their presentation and management.[89–92] The bone biology of children impacts the clinical examination findings and has a strong influence on both the decision to operate and the preferred methods of reconstruction.

Trapdoor Fractures

It has been well established that pediatric bones respond to external deformation with greater elasticity than do adult bones,[93,94] resulting in a higher incidence of greenstick fractures and plastic deformation without fracture in the setting of blunt trauma.[24] The softer, more pliable bones of the pediatric population make them more susceptible to greenstick fracture patterns. Similarly, trapdoor fractures are more common in pediatric populations.[49] As pediatric facial fractures are usually associated with lower velocity injuries than adults, including falls and sports-related accidents, minimal periorbital trauma may be observed. Pediatric trapdoor orbital floor fractures presenting without subconjunctival hemorrhage have been referred to in the literature as white-eyed blowout fractures.[49–52] The oculocardiac reflex is particularly strong in children and often includes nausea and emesis as primary features.[95] Clinicians must maintain a high degree of suspicion for these fractures in the pediatric population, as they often present with nausea and vomiting and minimal evidence of trauma.[96] Moreover, children may not perceive diplopia; nor will they be cooperative with all parts of examination, particularly, forced duction testing.[24]

All children with suspected orbital floor fractures should undergo computed tomographic scanning with thin coronal sections to determine the presence of periorbital tissue entrapment[97] (Fig. 1). When there are clinical signs of entrapment and computed tomographic evidence of periorbital tissue entrapment, early surgical intervention (<48 hours) has been associated with lower rates of persistent postoperative diplopia.[24,98] However, computed tomographic findings suggestive of entrapped periorbital tissue in the absence of clinical entrapment do not necessitate urgent surgical treatment. These patients can be managed expectantly without any increased risk of persistent diplopia.[24]

Conservative Management

Most studies defining computed tomography–based criteria for orbital floor reconstruction based on defect size have focused on adult populations[39–42]; however, there is evidence to

suggest that, regardless of the bony defect size, pediatric orbital blowout fractures can be managed conservatively in the absence of acute entrapment, enophthalmos, or vertical orbital dystopia (**Level of Evidence: Therapeutic, IV**).[99] In one series, the rate of mild postoperative enophthalmos in untreated fractures was 30 percent, all of which fell below the 2-mm threshold of clinical relevance.[99] Moreover, there is an increased risk for adverse outcomes with operative treatment of pediatric orbital floor fractures, furthering the recommendations to manage these patients conservatively.[100]

Growth Considerations

When operative repair is indicated in pediatric orbital fractures, the potential for growth disturbance in this population must be considered when choosing materials used for reconstruction. The use of rigid alloplasts that do not grow with the child may restrict growth and/or become entrapped within the remodeling bone of the facial skeleton.[101–103] For these reasons, resorbable or biocompatible materials are preferred for orbital floor reconstruction and fixation in children.[24,101,104–110]

Several resorbable alloplastic materials are available for orbital floor reconstruction and offer the advantages of availability, no donor-site morbidity, and nonpersistence of the implant, theoretically minimizing the potential for growth restriction.[71] A recent systematic review of the literature showed that resorbable alloplasts are successful in reconstructing orbital floor defects of various sizes without a significant increase in complications (**Level of Evidence: Therapeutic, IV**).[111] Still, many authors prefer split calvarial bone grafts in patients who have a developed diploic space because of its biocompatibility, integration, minimal donor-site morbidity, and ease of dissection should subsequent surgery be necessary.[24,104,108–110]

COMPLICATIONS

The most common complications following surgical repair of the orbital floor are persistent postoperative diplopia, infraorbital nerve dysfunction, and enophthalmos. The incidence of postoperative diplopia ranges from 20 to 52 percent in several published series.[112–115] A recent review of a single-center experience revealed a 55 percent incidence of postoperative infraorbital nerve dysfunction, defined as hypesthesia or dysesthesia, and a 27.5 percent incidence of persistent enophthalmos; however, these complications were reduced when immediate (<2 days) repair was performed.[116] Although commonly cited as a complication of orbital floor reconstruction, lower lid retraction, including ectropion and entropion, is relatively uncommon. A review of more than 300 orbital floor repairs using standardized postoperative anthropometry revealed an overall ectropion rate of 2.6 percent in operated eyes, significantly higher than the rate in unoperated eyes, and no significant increase in the incidence of postoperative entropion.[117] Moreover, this same study used the eye fissure index, a more sensitive measure of lower lid retraction, to determine an increased incidence of scleral show in operated versus unoperated eyes (13.9 percent versus 4.4 percent).[117]

Many of the complications associated with orbital repair are secondary to enlargement of the bony orbit because of slight imperfections in reconstructing the orbital anatomy. Enlargement of the bony orbit occurs in approximately 8.5 percent of all traumatic orbital reconstructions using traditional methods.[118] This is attributable, at least in part, to inability to accurately recognize anatomical landmarks, particularly with high-velocity injuries, where bony destruction can be severe.[119,120] For this reason, preoperative computed tomographic scanning has been used for virtual surgical planning, where stereolithographic models can be created for prefabrication of reconstruction plates.[119] Further developments in this technology have allowed for intraoperative point-by-point guidance for placement of prefabricated plates using navigational markers.[119] Interested readers are directed to the references for further discussion.

Implant-associated infections include implant migration, infection, exposure, palpability, or local inflammatory reaction, as outlined above.[72–85] Serious complications include postoperative optic neuropathy, blindness, and retrobulbar hematoma. Although rare, these should be discussed in detail with each patient and treatment plans should be individualized to minimize risks and maximize outcomes.

Arun K. Gosain, M.D.
Division of Plastic and Reconstructive Surgery
Ann & Robert H. Lurie Children's Hospital of Chicago
225 East Chicago Avenue
Chicago, Ill. 60611
argosain@luriechildrens.org

APPENDIX: EVIDENCE RATING SCALE USED FOR THE LITERATURE REVIEW IN CREATING THIS MAINTENANCE OF CERTIFICATION ARTICLE

Level of Evidence	Qualifying Studies
I	High-quality, multicenter or single-center, randomized controlled trial with adequate power; or systematic review of these studies
II	Lesser-quality randomized controlled trial; prospective cohort study; or systematic review of these studies
III	Retrospective cohort or comparative study; case-control study; or systematic review of these studies
IV	Case series with pre/post test or only post test
V	Expert opinion developed via consensus process; case report or clinical example; or evidence based on physiology, bench research, or "first principles"

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