Pediatric Orbital Fractures

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Abstract

Keywords

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It is wise to recall the dictum “children are not small adults” when managing pediatric orbital fractures. In a child, the craniofacial skeleton undergoes significant changes in size, shape, and proportion as it grows into maturity. Accordingly, the craniomaxillofacial surgeon must select an appropriate treatment strategy that considers both the nature of the injury and the child’s stage of growth. The following review will discuss the management of pediatric orbital fractures, with an emphasis on clinically oriented anatomy and development.

Pediatric orbital fractures occur in discreet patterns, based on the characteristic developmental anatomy of the craniofacial skeleton at the time of injury. To fully understand pediatric orbital trauma, the craniomaxillofacial surgeon must first be aware of the anatomical and developmental changes that occur in the pediatric skull.

Anatomy and Development

Seven bones make up the orbit: frontal, maxilla, zygoma, ethmoid, lacrimal, greater and lesser wings of the sphenoid, and palatine. The outer rim of the orbit is comprised of the first three robust bony elements, protecting the more delicate internal bones of the orbital cavity. The orbital cavity is itself bound by the orbital roof, lateral and medial walls, and orbital floor. Some of these boundaries display changes in structural integrity—closely related to sinus pneumatization—during different stages of development. On viewing the cross-sectional anatomy of pediatric and adult skulls (►Fig. 1), the striking bony differences that occur with sinus development become obvious, revealing their relative strengths and weaknesses.

In utero, the sinuses and nasal cavity are a single structure. The ethmoid, frontal, and maxillary sinuses then subdivide from the nasal cavity in the second trimester. Subsequent sinus formation occurs in a predictable sequence. The maxillary sinuses are the first to develop. The growth of these paired sinuses is biphasic, occurring between 0 to 3 and 7 to

12 years of age. During mixed dentition, the cuspid teeth are immediately beneath the orbit: hence the term *eye tooth* in dental parlance. It is not until age 12 that the maxillary sinus expands, in concert with eruption of the permanent dentition. At 16 years of age, the maxillary sinus reaches adult size (►Fig. 2).¹ These changes are a quintessential example of the how ongoing sinus development impacts fracture patterns and susceptibility. Specifically, the presence of the unerupted maxillary dentition serves to resist orbital floor fracture in young children.²

The ethmoid sinuses are fluid-filled structures in a newborn child. During fetal development the anterior cells form first, followed by the posterior cells. They are not usually seen on radiographs until age 1. The cells grow gradually to adult size by age 12. The medial orbital wall becomes progressively thin during ethmoid sinus development. As a consequence, the medial wall of the orbit becomes increasingly susceptible to fracture in adulthood. The lamina papyracea is a portion of the ethmoid bone that abuts the developing ethmoid sinus. The Latin derivation of this term reveals its quality as a “paper-thin layer.”

Although the frontal bone is membranous at birth, there is seldom more than a recess present until the bone begins to ossify around age 2. Thus, radiographs rarely show this structure before that time. Frontal sinus pneumatization begins at ~7 years of age and completes development during adulthood. In children, frontal bone and orbital roof fractures are commonplace because of the high cranium-to-face ratio

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Figure 1 Coronal sections of the pediatric and adult orbit. (A) The thick pediatric orbital floor is contrasted with its diminutive orbital roof. (B) The delicate nature of the adult orbital floor and medial wall is apparent.

(►Fig. 3) and incomplete (or absent) pneumatization of the frontal sinus. In addition, the supraorbital rim and frontal bone are the most prominent structures on the pediatric craniofacial skeleton; consequently, they sustain the brunt of

initial impact. In the adult skeleton, however, the frontal sinus, supraorbital rim, and frontal bone protect the brain and orbital cavity from injury.³ Koltai et al reviewed a series of orbital fractures in children aged 1 to 16 years. Based on their data, they determined that at age 7, orbital floor fractures become more common than orbital roof fractures.⁴ The orbital floor becomes more susceptible to fracture in later childhood. Similar results were described by Fortunato and Manstein.⁵ This age coincides with development of the maxillary sinus, as stated previously. Incidentally, the orbit reaches an adult size at approximately the same age.

The lateral orbital wall is the only nonsinus boundary of the orbital cavity. Fractures of the lateral orbital wall are rare in children—owing to the strong zygomatic and frontal bones, which meet at the zygomaticofrontal (ZF) suture. When fractures of the zygomaticomaxillary complex do occur, the lateral orbital wall is disrupted at the articulation of the zygoma and greater wing of the sphenoid.

The contents of the orbital cavity include the globe, extraocular muscles, lacrimal gland, periorbital fat, and neurovascular bundles. The ligamentous support of the globe includes the medial and lateral check ligaments, the orbital septum, and Lockwood's suspensory ligament. The elasticity and resilience of these structures in the pediatric orbit provides additional stability. When the developing orbit is fractured, these ligaments may "splint" the orbital contents, resisting ocular displacement (►Fig. 4). Accordingly, enophthalmos is less commonly observed in children. The medial canthal tendon may be disrupted in lacrimal bone fractures, naso-orbito-ethmoid (NOE) fractures, or in centrally located lacerations, resulting in telecanthus.

In general, the immaturity of the pediatric facial skeleton serves to resist fracture; there are higher proportions of cancellous bone in children, and the growing sutures retain a cartilaginous structure. This allows pediatric facial bones to absorb more energy during impact without resulting in fracture. "Greenstick" and minimally displaced facial

Figure 2 Maxillary sinus development. The progressive increase in size of the maxillary sinus (green) is seen (A) at birth, (B) at 5 years of age, and (C) in the adult.

Figure 3 Cranium-to-face ratio. At birth and in early childhood, the cranium represents a relatively large volume of the craniofacial complex (8:1 and 4:1, respectively). Cranial and orbital fractures are therefore more common during this time. The cranium-to-face ratio in the adult is ~2:1.

fractures predominate in children, and pediatric bones can temporarily deform to elude fracture; correction occurs during growth. *Young's modulus*—the elastic modulus—describes the ability of a substance to deform in response to force prior to its breaking point (i.e., ultimate strength); in common terms, Young's modulus describes a substance's stiffness. Young's modulus has been shown to be lower in cancellous bone,⁶ making the pediatric craniofacial skeleton more elastic. Bone mineralization increases with age, conceptually changing the bones from elastic to rigid. For the elastic pediatric craniofacial bones to fracture, a significant force of impact must be endured. This premise is intimately related to the associated incidence of neurocranial injuries in pediatric facial fractures.

Figure 4 Ligamentous support. The resilient connective tissues of the pediatric orbit allow for expectant management in certain cases of orbital floor fracture. The ligamentous structures prevent expansion of orbital volume.

Epidemiology

In developed countries, trauma is the leading cause of death among children. A review of the National Trauma Databank, 2001 to 2005, identified 12,739 facial fractures among 277,008 pediatric trauma patient admissions (4.6%).⁷ The relative paucity of facial fractures in children—when compared with adults—has been previously demonstrated.^{8,9} The preponderance of elastic, cancellous bone and the aforementioned high cranium-to-face ratio in children (► **Fig. 3**) are responsible for this finding. As a corollary, children are more likely to sustain skull fractures and brain injuries than facial fractures.^{10–12} Indeed, facial fractures are rare before the age of 5.¹³ Rowe, for example, reported that only 1% of all facial fractures occur in children 1 year old or younger.¹⁴ These results were echoed by other series,^{1,9,15} although the incidence may be underestimated, as these studies predate the use of routine computed tomography (CT) scans in craniofacial trauma.¹⁶

When considered anatomically, there is a downward shift—from cephalad to caudad—in facial fracture patterns with age. The frontal skull and orbital roof are prone to fractures in newborns to children aged 5 years, whereas midface and mandible fractures occur at higher frequencies in children ages 6 to 16 years. The nose and mandible then become the most commonly fractured facial bones in adults, due to their prominence.^{1,15}

The relative frequency of pediatric facial fractures according to anatomical location is seen in ► **Fig. 5**.⁷ In most series of pediatric facial trauma, orbital fractures comprise 5 to 25% of facial fractures.¹⁷ In the National Trauma Databank review, orbital fractures were identified in 10% of cases. Variability in facial fracture patterns has also been shown between urban and rural environments.¹⁸ As in adults, boys are twice as likely to sustain facial fractures than girls. A seasonal variation in pediatric facial fractures should also be noted. Logically, the peak month in the United States is July, corresponding to increased patterns of outdoor play.

The mechanisms of injury for orbital fractures are similar to the general causes of facial fractures in children. In one series of pediatric orbital fractures, 27% were the result of

Figure 5 The relative frequency of pediatric facial fractures. In most series of pediatric facial trauma, orbital fractures comprise 5 to 25% of facial fractures. (Adapted from Imahara⁷.)

activities of daily living.¹⁹ Motor vehicle accidents comprised the next causative segment, with 22% of cases. In this category, the prevalence all-terrain vehicle accidents should be noted, particularly in rural areas.¹¹ Sports injuries followed with 18% of orbital fractures. Children who sustained orbital fractures from activities of daily living were, on average, 6 years old, whereas children who sustained orbital fractures from violence were twice as old, with an average age of 13.6 years. Child abuse and assault are rare causes of facial trauma in children; nonetheless, a high index of suspicion should be maintained. The astute clinician will recognize a constellation of injuries that do not fit the given history. Skeletal surveys to assess for long bone fractures should be performed in suspected cases. Rib fractures are highly predictive of nonaccidental trauma,²⁰ and retinal hemorrhages are a classic finding in shaken baby syndrome.

Patterns of Injury

Facial trauma is a possible harbinger of life-threatening injury and indicates the potential of concomitant injury to the airway, neuraxis, viscera, and axial skeleton.²¹ The possibility of unstable hemodynamic phenomena from organic injury must also be recognized; the treatment of these concurrent, systemic injuries takes precedence over craniomaxillofacial reconstruction.

Nonetheless, patients with craniofacial injuries are often prematurely assigned to the care of subspecialty services. It is important that this team reevaluate the patient for the possibility of evolving injuries. Children with facial fractures exhibit increased injury severity scores, hospital and intensive care unit lengths of stay, number of ventilator days, and hospital charges when compared with those without facial fractures.⁷ In all trauma settings, adherence to Advanced Trauma Life Support protocol is essential.

The clinical diagnosis of pediatric orbital fractures may be complicated by the difficulty of achieving a complete examination in an uncooperative patient. When there is any suspicion of fracture or neurological injury, CT scanning should ensue. If head injury is a component of the pediatric trauma patient, the child must be accompanied to the radiology suite, and sedation should be avoided if possible.

Advances in the realm of CT have greatly enhanced diagnosis and preoperative planning. Nonetheless, in the pediatric patient, the immature craniofacial skeleton may obscure fracture lines, complicating the radiographic evaluation.²² Sections are generally taken 1.25 mm apart. Coronal reformatting is frequently performed to further delineate fracture lines in alternate planes. Coronal views are crucial in evaluating the orbital floor. Three-dimensional CT permits further analysis of fracture patterns.²³ With this modality, volume and proportionality may be directly assessed. Care must be taken in the interpretation of three-dimensional images of the craniofacial skeleton—particularly those involving the orbit. The thin bones of the orbital floor and medial wall may be erroneously absent owing to the derivative nature of the formatting process. In reviewing the two-dimensional images of the CT scan, particular clues to the radiographic diagnosis of orbital fractures include the presence of step-off deformities along the orbital rim, orbital emphysema, and sinus opacification. In cases of orbital floor fracture, orbital contents may be seen herniating into the maxillary sinus (►Fig. 6). Conversely, the orbital floor may appear uninjured in spite of these findings, as the bone may recoil back into native position.

Associated Injuries

Associated injuries are more common in children with facial fractures when compared with adults. In addition, pediatric

Figure 6 Orbital floor fracture. On this coronal section of a maxillofacial computed tomography scan, orbital contents can be visualized within the maxillary sinus.

patients with facial fractures have more severe associated injury to the head and chest and considerably higher overall mortality.⁷ In one series of 74 pediatric orbital fracture patients, 32 patients (43%) presented with neurological injury (concussion, depressed skull fracture, and/or intracranial hemorrhage); an additional 20% of patients had injuries beyond the head and neck (long bone fracture, pelvic fracture, and/or blunt chest/abdominal trauma).¹⁹ The importance of complete examination by the craniomaxillofacial surgeon—and timely involvement of other consulting services—must be emphasized. In the latter series, nonfacial lacerations were encountered with a frequency of up to 60%,¹⁹ highlighting the importance of a thorough secondary trauma survey.

Orbital Floor and Medial Orbital Wall Fractures

Orbital fractures may involve only the orbital floor and/or medial wall, sparing the adjacent facial bones (—**Fig. 7**). The initial definition of a “blowout” fracture is attributed to Smith and Regan.²⁴ The term *blowout* has become somewhat of a colloquial term, referring to an explosive type of orbital fracture with characteristic bone fragment divergence away from the orbit. Some authors have also used the term to describe fractures of the orbital roof. In a similar manner,

“trapdoor” fractures describe fractures of the orbital floor that result in muscle entrapment, whereas “open door” fractures refer to orbital floor fractures without entrapment. Moreover, the terms *pure* and *impure* have also been used to describe isolated orbital fractures (*pure*) versus orbital fractures that occur in conjunction with other fractures (*impure*). To avoid the inherent confusion over this arcane nomenclature, orbital fractures should be clinically described based on: the mechanism of injury, the precise anatomic structures involved, and the presence or absence of entrapment.

There has been considerable controversy surrounding the causative mechanism of orbital blowout fractures, and two dominating theories have emerged: the *hydraulic theory* and the *bone conduction theory*.^{25–27} The hydraulic theory attributes orbital floor fractures to indirect pressure from the globe, whereas the bone conduction theory maintains that the thin internal orbital bones buckle under transmitted forces from the stronger orbital rim. Teleologically, it follows that the globe is able to fracture the orbital floor; if the converse were true, the globe would be ruptured with greater frequency, and posttraumatic visual deficits would become more common.

The treating surgeon should perform a thorough eye examination, regardless of eventual consultation by an ophthalmologist. This examination should include assessment of globe integrity, extraocular movements, visual fields, visual

Figure 7 Midface and maxillary fracture patterns.

acuity, and pupillary response. In the unconscious patient, a forced duction test should also be performed. Although diplopia is defined as subjective double vision, entrapment indicates the limitation of extraocular movements, which can be objectively measured on physical examination (►Fig. 8).

Figure 8 Entrapment. The inferior oblique and/or inferior rectus muscles can become entrapped within an orbital floor fracture. The limitation of extraocular movements can be seen on vertical gaze during physical examination. The patient will experience diplopia during this maneuver. Operative intervention is mandated.

Diplopia may be the result of swelling within the orbit, caused by extraocular muscle edema, chemosis, and/or hematoma. In these cases, double vision is commonly present in all fields of view. The distinguishing characteristic of entrapment is the presence of diplopia on forced gaze, often in the upward vector. Muscle entrapment is confirmed with the inability to perform forced duction on the anesthetized child. The most commonly affected muscles are the inferior rectus and inferior oblique.

Although periorbital edema, laceration, contusion, and hematoma are common signs of an orbital fracture, they may be absent altogether from the physical examination in the pediatric patient. Such an absence of physical findings has been referred to as a “white-eyed” blowout fracture.²⁸ Because of the inherent difficulty in the examination of the pediatric trauma patient, more subtle surrogates of entrapment may be observed. In one study, nausea and vomiting were highly predictive of entrapment, being observed in five of six patients with a trapdoor fracture.²⁹

Children with orbital floor/medial orbital wall fractures are prone to entrapment.³⁰ The elastic quality of pediatric facial bones enables the orbital floor to sustain a greenstick fracture, whereby the orbital adnexa become ensnared in a temporary defect in the orbital floor (i.e., the trapdoor phenomenon). Adults, in contradistinction, are more likely to sustain comminuted fractures of the orbital floor; extraocular muscles can still become entrapped in these cases via spiculated fracture margins. One case series of 70 patients with orbital floor fractures found that entrapment was more commonly encountered in children when compared with adults: 81% versus 44%, respectively (odds ratio = 5.4; $p = 0.01$).³¹ The authors attribute this observation to the “spring-like restoring force of the [pediatric] inferior orbital wall.” Another study corroborated these findings, with entrapment observed in 93% of all pediatric orbital floor fractures,³² though such high incidence was not found in other series.³³

When entrapment is diagnosed, ischemia of the involved extraocular muscle can cause permanent damage, hence the treatment of these fractures is considered a surgical emergency. Volkmann’s ischemic contracture of the extraocular musculature is difficult to correct surgically,³⁴ and may require the use of prism glasses to avoid persistent diplopia.

Enophthalmos may also be observed following orbital floor and medial wall fractures. This finding, however, may be difficult to appreciate in the acute setting. Rather, it may be seen posttraumatically after resolution of edema. Late enophthalmos is due to a discrepancy between the orbital contents and bony orbital volume.^{35,36} Escape of orbital fat, fat necrosis, entrapment, cicatricial contraction of the retrobulbar tissues, and enlargement of the orbital cavity have all been cited as causative mechanisms.³⁷ Vertical ocular dystopia (discrepant positioning of the globes in the vertical plane) is an indication that both the ligamentous *and* bony support of the globe have been disrupted, bolstering the indication for operative intervention.³⁸ The term *vertical ocular dystopia* is preferred to *vertical orbital dystopia* in this context, as the globe—and not the orbital rim—has been displaced.

The infraorbital nerve—cranial nerve (CN) V₂—is the most commonly injured nerve associated with orbital floor fracture. The orbital floor demonstrates an area of weakness along the course of the nerve. Hypesthesia is frequently related to neuropraxia, resolving over the convalescent period. Unfortunately, permanent sensory disturbance in the cheek, upper lip, and nasal sidewall may also occur.

The indications for operative intervention in children with orbital floor and/or medial wall fractures are different than those in adults. In children with significant injury to the orbital floor (e.g., defect of 2 to 3 cm² or 50%),³⁹ operative intervention may be deferred in the absence of entrapment, enophthalmos, and vertical ocular dystopia, although some surgeons still prefer to explore large orbital floor defects.⁴⁰ The resilient and elastic connective tissues of the pediatric orbit may prevent expansion of the orbital adnexa and subsequent late enophthalmos (►Fig. 4).¹⁹ This premise is supported by earlier descriptions of “a fine network of ligamentous attachments throughout the orbital fat” connecting to the surrounding periosteum⁴¹ and the ability of the pediatric periosteum to resist tearing.^{42,43} Close follow-up with clinical observation should be employed in these cases.

The authors’ preferred method to access the orbital floor is via the transconjunctival approach. Corneal shields are essential when any orbital exposure is attempted. The lower eyelid is retracted with a Desmarres retractor and an incision is made above the inferior fornix. A retroseptal dissection is carried through the periorbitum, directly onto the orbital floor. When combined with a lateral canthotomy and cantholysis, exposure of the lateral orbital wall is also possible.

A subciliary or midlid incision also allows for exposure of the orbital floor, but requires an external cutaneous incision. Although providing superior exposure, these incisions carry a higher risk of visible scars and subsequent ectropion. The transcaruncular incision is primarily used to visualize isolated fractures of the medial orbit. When used appropriately, this approach may obviate the need for a coronal incision. A Caldwell-Luc antrostomy has also been described to access these fractures via the maxillary sinus.⁴⁴

After reaching the orbital floor, the complete bony perimeter of the fracture is exposed. All herniated muscle and orbital fat is then removed from the fracture and repatriated to the orbit. Corticosteroids are routinely indicated if extensive manipulation is required intraoperatively.⁴⁵ One must recall that in children, the optic nerve may be closer than the standard 4-cm distance from the inferior orbital rim. Autologous bone is used to reconstruct the orbital floor defect; a thin, split calvarial bone graft is harvested such that the graft retains its periosteum.⁴⁶ The presence of periosteum helps to resist fracture during contouring of the graft to the orbital floor, and the grafts thinness makes it pliable and less brittle. Mild overcorrection with slight proptosis is the rule when bone grafting the orbital floor, to combat settling, resorption, and remodeling. The graft often does not require fixation; if needed, however, resorbable microplates may be used to secure the graft to the infraorbital rim.

Proper eye position in the vertical and the anteroposterior dimension (i.e., correction of hypoglobus and enophthalmos, respectively) is the surgical endpoint for correcting orbital floor and/or medial wall fractures. Improper graft placement should be considered if these conditions are not met. At the conclusion of the procedure, forced duction should again be performed to confirm ocular mobility.

Orbital Roof/Skull Base Fractures

As the frontal sinus pneumatizes, the transmission of force from the superior orbital rim to the anterior cranial base is diminished. Concordantly, orbital roof fractures are rare in adulthood, replaced by a predominance of frontal sinus fractures. In childhood, however, orbital roof injuries are commonplace, and must be considered as fractures of the skull base. As such, neurological injuries are frequently coincident.

In children, the most common fracture pattern extends along the frontal bone through the supraorbital foramen, and then progresses to involve the orbital roof/anterior cranial base.⁴⁷ Generally these craniofacial fractures do not require open reduction and internal fixation (ORIF) unless a significant displacement is observed. In such cases, a so-called “growing skull fracture” may occur. The growing skull fracture is a unique entity among pediatric orbital fractures.⁴⁸ John Howship, an English surgeon, first reported this condition 1816 as “partial absorption of the (right) parietal bone, arising from a blow on the head.”⁴⁹ When a dural tear occurs beneath a displaced orbital roof fracture, a leptomeningeal cyst may form during the regenerative process. The cyst interferes with osseous healing, and frontal bone nonunion results (►Fig. 9). Pulsatile exophthalmos ensues, due to compression of the orbital cavity. Vertical ocular dystopia results, and vision is threatened from orbital compartment syndrome and optic nerve compression.⁵⁰ The treatment of

Figure 9 The growing skull fracture. When a dural tear occurs beneath a displaced orbital roof fracture, a leptomeningeal cyst may form during the regenerative process. The cyst interferes with osseous healing, and frontal bone nonunion results. Pulsatile exophthalmos ensues, due to compression (arrows) of the orbital cavity.

growing skull fractures involves a transcranial approach. Excision of the leptomeningeal cyst is followed by reconstruction of the dural and bony defects.^{51,52} Split calvarial bone grafting is required for the latter defect, and resorbable plates and screws are used to secure the graft.

An orbital “blow-in” fracture is another possible injury within the spectrum of orbital roof fractures in children. These injuries are the result of supraorbital impact directed inferiorly, effectively collapsing the orbital roof. When multiple fracture segments compress the orbital contents or cause dystopia or diplopia, operative relief is indicated.

For these and other displaced orbital roof and/or frontal bone fractures, a coronal approach is utilized. This permits direct visualization of the fracture for optimal reduction. A “lazy-S” or “sine wave” incision is fashioned along the scalp to minimize apparent cicatricial alopecia. Care should be taken to avoid placing an incision directly above the anterior fontanelle to avoid inadvertent injury to the sagittal sinus.

NOE Fractures

Nasal bone fractures account for nearly one-third of all pediatric facial fractures (►Fig. 5). When a significant traumatic force is imparted to the central nasal region, NOE fractures may ensue. The pathophysiology of an NOE fracture consists of an implosion of the nasal bones with concomitant fractures that collapse the paired nasal, lacrimal, and ethmoid bones. Fractures of the medial orbital wall and infraorbital rim may also be present, either unilaterally or bilaterally (►Fig. 7). This central facial region may be conceptualized as a pyramid, whose square base consists of the aforementioned bony support, and whose pyramidal apex is the nose. NOE fractures collapse this pyramid into the skull base. Accordingly, severe NOE fractures may involve the cribriform plate, resulting in anosmia and cerebrospinal fluid rhinorrhea. Neurosurgical consultation should be obtained when the latter finding is suspected.

Characteristic physical examination findings include a flattened nasal pyramid, retruded nasal bridge, and increased nasolabial angle. NOE fractures are often misdiagnosed as isolated nasal fractures; the astute clinician will notice increased width of the upper midface and telecanthus, avoiding this common error. When the diagnosis of an NOE fracture is missed in a child, detrimental midfacial growth disturbances will follow. These secondary deformities are difficult to correct. The assessment of medial canthal ligament integrity is crucial when these findings are observed. This critical structural element becomes separated from its attachments to the anterior and posterior lacrimal crests or the canthal bearing segments themselves can become detached. Telecanthus ensues, with increased distance between the medial canthi. The “bowstring” test should be performed if canthal injury is suspected.⁵³ By pulling laterally on the lower eyelid, the taut attachment of the medial canthal tendon to the anterior lacrimal crest should be appreciated. If the canthal bearing segment is impacted, however, the lower lid may appear taut despite the loss of structural integrity.

To correct an NOE fracture, the flattened “pyramid” must be disimpacted from the skull base. This maneuver may reveal a previously undisclosed CSF leak; the neurosurgical service should be on standby during the reduction of a severe NOE fracture. In addition to intranasal manipulation for bone reduction, transnasal wiring may be required to secure the canthal-bearing segment.⁵⁴ A coronal approach is usually required for exposure, but a transcaruncular incision may be used in select cases. Although resorbable plates have previously been advocated in pediatric craniomaxillofacial surgery, titanium microplates or wires are better suited for reduction of the small bone fragments commonly encountered in this fracture pattern.

Epiphora is a common early sequelae of NOE fractures. Frequently, it results from tissue edema alone, but it may also be the result of damage to the lacrimal system. In cases of localized edema, epiphora may resolve spontaneously; in cases of damage associated with bone displacement attendant to an NOE fracture, the treatment of choice is fracture reduction, which will often lead to a return of lacrimal drainage. If excessive tearing persists, injury to the nasolacrimal apparatus may be present. In these cases—and in cases of penetrating trauma to this region—the integrity of the lacrimal system must be assessed intraoperatively. The canaliculi should be probed and irrigated; Jones I and II tests may also be performed to establish level and severity.⁵⁵ Canicular injury requires repair over Silastic (Crawford; FCI Ophthalmics; Marshfield Hills, MA) tubing at the time of injury. With severe nasolacrimal duct obstruction, delayed recannulation via dacryocystorhinostomy is required; lacrimal obstruction can result in dacryocystitis. If not treated judiciously, orbital cellulitis may ensue.

Le Fort II and III Fractures

Midface fractures are uncommon in children, accounting for less than 5% of pediatric facial fractures under age 12; these fracture patterns are even less common in children under 6.²² When significant force is involved—as in motor vehicle accidents—Le Fort I, II, and III fractures can occur (►Fig. 7). The orbit is involved in Le Fort II and III fractures. Le Fort II fractures result in injury to the orbital floor and/or medial orbital wall. Le Fort III fractures, which are particularly rare in children, involve the lateral and medial orbital walls and the orbital floor. The treatment of Le Fort II and III fractures in children requires exposure via gingivobuccal sulcus and coronal incisions; additional orbital approaches are often required as well. The medial orbital wall and orbital floor should be inspected after Le Fort fracture reduction to determine the need for bone grafting—or other means of repair—if orbital volume must be restored. ORIF at the nasomaxillary and zygomaticomaxillary buttress (and lateral orbital rim in Le Fort III fractures) is required. Because these fractures violate the support system of the facial skeleton, the cranio-maxillofacial surgeon must weigh the strength of titanium microplates against their tendency to compound growth restriction (growth disturbances are nearly universal following pediatric Le Fort injuries). Resorbable plates, on the other

hand, are less likely to restrict growth, but may not possess the requisite strength to support the buttress system. Last, specialized maxillomandibular fixation techniques are required to reestablish the pediatric occlusion.

The oculocardiac reflex may be activated in cases of severe orbital trauma, as in cases of Le Fort fracture. The reflex is a triad of bradycardia, nausea, and syncope. Transient bradycardia may be witnessed perioperatively during ocular manipulation for fracture reduction. Although this finding rarely has any clinical hemodynamic significance, fatal cardiac arrhythmia has been reported in the literature.⁵⁶ This reflex is often associated with entrapment, and if present, urgent operative intervention is indicated.⁵⁷

Significant orbital trauma may also cause CN paresis. Superior orbital fissure syndrome consists of paralysis of the CNs that travel through its aperture (CN III, IV, V₁, and VI). When accompanied by visual loss (CN II involvement), the diagnosis of orbital apex syndrome is then made. An afferent pupillary defect or Marcus-Gunn pupil—paradoxical dilation of the pupil on swinging flashlight testing—therefore differentiates orbital apex syndrome from superior orbital fissure syndrome. All of these findings are ominous, and all are harbingers of significant injury within the orbit. Surgical relief of retrobulbar pressure via lateral canthotomy and cantholysis may be required. Priority should be given to protecting the eye and visual axis prior to consideration of facial fracture repair.

Zygoma Fractures

Zygoma fractures are rare in young children. The incidence has been reported at 16.3% for zygoma fractures with an orbital floor/medial orbital wall component and 4.7% for zygoma fractures alone.¹⁵ Notably, children are more likely than adults to sustain isolated fractures of the orbital rim. When zygoma fractures do occur in children, a fracture-dislocation pattern is frequently observed, owing to incomplete union at the pediatric ZF suture. In this injury pattern, the lateral orbital wall is disrupted as the zygoma articulates with the greater wing of the sphenoid (→Fig. 7). A downward displacement of the fracture and its attached lateral canthus often imparts an antimongoloid slant to the palpebral fissure. In these cases, vertical orbital dystopia is present as a result of the displaced orbital bone.

Operative treatment and approaches for the repair of zygomatic fractures in children are similar to those utilized in adults. An upper blepharoplasty incision may be used to access fractures of the lateral orbital wall and ZF suture. One should avoid placing incisions within the brow, which yields a conspicuous result. Gingivobuccal sulcus incisions may also be used to access the infraorbital rim and the zygomaticomaxillary buttress, as well as the inner surface of the zygomatic arch (which may aid in arch reduction). Transconjunctival or subciliary incisions may also be utilized.

In general, bioabsorbable fixation should be used for fixation of the pediatric craniomaxillofacial skeleton.⁵⁸ The diminutive infraorbital and lateral orbital rims of the young child may not accommodate the larger resorbable plating systems. Titanium microplates may be needed in such cases.

Postoperative Care

An overnight hospital stay is advocated for pediatric patients with significant orbital fractures that require surgical repair. This permits frequent ophthalmological examinations and assessment of neurologic status. Mild postoperative diplopia is common, with early resolution. Visual acuity should be assessed in the postanesthesia care unit, routinely during the hospitalization, and by the family following discharge. Increasing eye pain or changes in visual acuity require immediate bedside assessment. Blindness may result from undiagnosed orbital compartment syndrome or an untreated retrobulbar hemorrhage postoperatively (see Complications). The patient should also be cautioned against nose blowing in the convalescent period, particularly following repair of orbital floor and/or medial orbital wall fractures; orbital emphysema can cause orbital compartment syndrome and blindness from optic nerve compression. An orbital-antral fistula may also develop, which can cause recurrent infectious sequelae within the orbit, and is difficult to correct.⁵⁹

A fastidious surgeon will follow results with a critical eye, to assess the quality of the reconstruction. In this regard, serial photography is a helpful modality through which the clinician (and the family) can assess outcomes. Worm's-eye and frontal views are most helpful in demonstrating eye position at follow-up clinic visits. Hertel exophthalmometry may also be used in this regard, albeit with some difficulty in the obstinate child.

Complications

Alloplastic Implant Complications

The placement of titanium and MEDPOR implants (Stryker; Kalamazoo, MI) into the growing orbit is highly discouraged. These substances can extrude or become displaced (into the globe or maxillary sinus) during growth. When placed on the skull, metal hardware can transcranially migrate from the cranial surface to the endocranium⁶⁰ and may even restrict craniofacial growth if placed across an active suture.⁶¹

Bone grafts are less likely to experience these untoward effects of alloplastic implants in the child, but require expertise and carry attendant risks of harvest. More recently, some surgeons have advocated the use of resorbable mesh plates in the orbital floor⁶²; outcome studies regarding the long-term efficacy of such an approach, however, are still wanting.

Persistent Diplopia

Binocular diplopia results from strabismus, which may persist following appropriate treatment of orbital fractures. Transient muscle ischemia and scarring within the extraocular musculature may result in imbalance, which often requires surgical correction. Direct or indirect injury to the nerves of ocular motility (CN III, IV, VI) can also impact eye movement. In cases of increased intracranial pressure after trauma, the abducens nerve (CN VI) may be compressed along its intracranial course, leading to a related palsy. Migratory implants or bone grafts may also result in this phenomenon, and repeat imaging should be considered. The

treatment of diplopia includes the use of prism glasses, extraocular muscle botulinum toxin injection, and/or strabismus surgery.⁶³

Persistent Enophthalmos

Persistent enophthalmos results from inadequate restoration of posttraumatic orbital volume. As previously mentioned, this complication is rare in children, owing to the strong ligamentous support of the pediatric orbit. Operative restoration of orbital volume is indicated when enophthalmos is persistent or severe. Hertel exophthalmometry, although a useful objective measurement of enophthalmos, is difficult to perform in the younger or acutely injured child. As indicated previously, serial worm's-eye photographs are the best way to follow progression or resolution of this finding.

Ectropion

Ectropion is a common, iatrogenic sequelae of orbital fracture exposure, particularly when the anterior lamella of the lower lid is divided to access the orbit. Disinsertion of the lower lid retractors is another possible culprit. Increased scleral show will be seen on examination, and lagophthalmos is possible in severe cases. Resuspension with anatomic reapproximation of periorbital tissues (reinsertion of the lower lid retractors) must be performed following osseous reduction. Tarsorrhaphy and/or intermarginal (Frost) sutures may be placed to temporarily suspend the lower lid postoperatively. In minor cases, conservative treatment with scar massage may improve symptoms. Lateral canthal tightening via canthotomy, cantholysis, and canthopexy to the periorbitum overlying Whitnall's tubercle (the tarsal strip procedure) restores lower lid position in the more severe cases. Division of the cicatrix, with full-thickness skin grafting to the lower lid may also be required. Conversely, entropion may occur with violation of the posterior lamella, as in the subconjunctival approach. Everting (Quickert) sutures can be used as a means to correct this less common phenomenon.⁶⁴

Ocular Injuries

Globe injury following orbital trauma ranges from 7.2 to 30%.^{7,65} These injuries may range from corneal abrasion to globe rupture, which is the most common cause of blindness following orbital trauma. Additional acute traumatic ophthalmological conditions include retinal detachment, vitreous hemorrhage, and optic nerve compression. Visual loss can also occur from central retinal artery occlusion or thrombosis of the orbital veins. Pediatric orbital fractures carry a higher incidence of blinding injuries.⁶⁶ Blindness at the time of presentation is usually the result of direct optic nerve injury (CN II). Postoperative blindness resulting from reduction of facial fractures is exceedingly rare.⁶⁷ When observed, it is related to increased pressure within the optic canal. Sudden proptosis and unilateral loss of vision herald a retrobulbar hemorrhage, which must be treated emergently. Surgical decompression via lateral canthotomy and cantholysis must occur emergently to salvage vision.

Routine ophthalmological consultation should be obtained in *all* pediatric patients with orbital trauma. Injuries

to the eye and visual axis take precedence in the triage of orbital fracture repair. Coordination with the ophthalmologists as to the timing of fracture repair and the ability to manipulate the globe at the time of repair is of the utmost importance.

Conclusions

Pediatric orbital fractures occur in discreet patterns based on the characteristic developmental anatomy of the craniofacial skeleton at the time of injury. Although uncommon in children, orbital fractures can be devastating to both vision and appearance. Meticulous physical examination techniques, coupled with the previously outlined treatment principles, will allow the craniomaxillofacial surgeon to achieve successful outcomes in the management of these injuries. The treating surgeon must focus his or her intervention on delivering the best possible result, placing a premium on the future development of the pediatric craniofacial skeleton.

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References

- 1 Enlow D. Handbook of Facial Growth. Philadelphia, PA: WB Saunders; 1982
- 2 Dixon A, Hoyte DA, Rönning O. Fundamentals of Craniofacial Growth. Salem, MA: CRC Press; 1997
- 3 Messinger A, Radkowski MA, Greenwald MJ, Pensler JM. Orbital roof fractures in the pediatric population. *Plast Reconstr Surg* 1989;84:213–216; discussion 217–218
- 4 Koltai PJ, Amjad I, Meyer D, Feustel PJ. Orbital fractures in children. *Arch Otolaryngol Head Neck Surg* 1995;121:1375–1379
- 5 Fortunato M, Manstein G. Facial bone fractures in children. *Plast Reconstr Surg* 1982;70:650
- 6 Rho JY, Ashman RB, Turner CH. Young's modulus of trabecular and cortical bone material: ultrasonic and microtensile measurements. *J Biomech* 1993;26:111–119
- 7 Imahara SD, Hopper RA, Wang J, Rivara FP, Klein MB. Patterns and outcomes of pediatric facial fractures in the United States: a survey of the National Trauma Data Bank. *J Am Coll Surg* 2008;207:710–716
- 8 Posnick JC, Wells M, Pron GE. Pediatric facial fractures: evolving patterns of treatment. *J Oral Maxillofac Surg* 1993;51:836–844; discussion 844–845
- 9 Zerfowski M, Bremerich A. Facial trauma in children and adolescents. *Clin Oral Investig* 1998;2:120–124
- 10 Singh DJ, Bartlett SP. Pediatric craniofacial fractures: long-term consequences. *Clin Plast Surg* 2004;31:499–518, vii
- 11 Demas PN, Braun TW. Pediatric facial injuries associated with all-terrain vehicles. *J Oral Maxillofac Surg* 1992;50:1280–1283
- 12 Reedy B, Bartlett SP, eds. Pediatric Facial Fractures. *Pediatric Plastic Surgery*, ed. M. Bentz. Stamford, CT: Appleton & Lange; 1998: 463–486
- 13 Dufresne C, Manson PN. Pediatric facial injuries. In: Mathes SJ, Hentz VR, eds. *Mathes Plastic Surgery*. Philadelphia, PA: Saunders Elsevier; 2005:424–434

- 14 Rowe NL. Fractures of the facial skeleton in children. *J Oral Surg* 1968;26:505–515
- 15 McCoy FJ, Chandler RA, Crow ML. Facial fractures in children. *Plast Reconstr Surg* 1966;37:209–215
- 16 Shultz R, Meilman J. Complications of facial fractures. In: Goldwyn R, Cohen M, eds. *The Unfavorable Results in Plastic Surgery*. Boston, MA: Little Brown; 1984
- 17 Bales CR, Randall P, Lehr HB. Fractures of the facial bones in children. *J Trauma* 1972;12:56–66
- 18 Sherick DG, Buchman SR, Patel PP. Pediatric facial fractures: analysis of differences in subspecialty care. *Plast Reconstr Surg* 1998;102:28–31
- 19 Losee JE, Afifi A, Jiang S, et al. Pediatric orbital fractures: classification, management, and early follow-up. *Plast Reconstr Surg* 2008;122:886–897
- 20 Barsness KA, Cha ES, Bensard DD, et al. The positive predictive value of rib fractures as an indicator of nonaccidental trauma in children. *J Trauma* 2003;54:1107–1110
- 21 Plaisier BR, Punjabi AP, Super DM, Haug RH. The relationship between facial fractures and death from neurologic injury. *J Oral Maxillofac Surg* 2000;58:708–712; discussion 712–713
- 22 Rowe N, Williams JC. Children's fractures. In: Roew N, Williams JC, eds. *Maxillofacial Injuries*. New York, NY: Churchill Livingstone; 1985:538
- 23 Millman AL, Lubkin V, Gersten M. Three-dimensional reconstruction of the orbit from CT scans and volumetric analysis of orbital fractures: its role in the evaluation of enophthalmos. [editorial] *Adv Ophthalmic Plast Reconstr Surg* 1987;6:265–268
- 24 Smith B, Regan WF Jr. Blow-out fracture of the orbit; mechanism and correction of internal orbital fracture. *Am J Ophthalmol* 1957;44:733–739
- 25 King EF, Samuel E. Fractures of the orbit. *Trans Ophthal Soc U K* 1944–1945;64:134–153
- 26 Le Fort R. Etude experimentale sur les fractures de la machoire superieure. *Rev Chir* 1901;23:208, 360, 479
- 27 La Grange F. *Les Fractures de l'Orbite Parprojectiles de Guerre*. Paris, France: Masson et Cie; 1917
- 28 Jordan DR, Allen LH, White J, Harvey J, Pashby R, Esmaeli B. Intervention within days for some orbital floor fractures: the white-eyed blowout. *Ophthalm Plast Reconstr Surg* 1998;14:379–390
- 29 Cohen SM, Garrett CG. Pediatric orbital floor fractures: nausea/vomiting as signs of entrapment. *Otolaryngol Head Neck Surg* 2003;129:43–47
- 30 Grant JH III, Patrinely JR, Weiss AH, Kierney PC, Gruss JS. Trapdoor fracture of the orbit in a pediatric population. *Plast Reconstr Surg* 2002;109:482–489; discussion 490–495
- 31 Kwon JH, Moon JH, Kwon MS, Cho JH. The differences of blowout fracture of the inferior orbital wall between children and adults. *Arch Otolaryngol Head Neck Surg* 2005;131:723–727
- 32 de Man K, Wijngaarde R, Hes J, de Jong PT. Influence of age on the management of blow-out fractures of the orbital floor. *Int J Oral Maxillofac Surg* 1991;20:330–336
- 33 Tong L, Bauer RJ, Buchman SR. A current 10-year retrospective survey of 199 surgically treated orbital floor fractures in a nonurban tertiary care center. *Plast Reconstr Surg* 2001;108:612–621
- 34 Smith B, Lisman RD, Simonton J, Della Rocca R, Volkmann's contracture of the extraocular muscles following blowout fracture. *Plast Reconstr Surg* 1984;74:200–216
- 35 Manson PN, Clifford CM, Su CT, Iliff NT, Morgan R. 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
- 36 Manson PN, Grivas A, Rosenbaum A, Vannier M, Zinreich J, Iliff N. Studies on enophthalmos: II. The measurement of orbital injuries and their treatment by quantitative computed tomography. *Plast Reconstr Surg* 1986;77:203–214
- 37 Kawamoto HK Jr. Late posttraumatic enophthalmos: a correctable deformity? *Plast Reconstr Surg* 1982;69:423–432
- 38 Losse J, Jiang S. Pediatric facial trauma. In: Guyuron B, Eriksson E, Persing JA, eds. *Plastic Surgery: Indications and Practice*. Philadelphia, PA: China Saunders Elsevier; 2009:651
- 39 Manson PN, Iliff N. Management of blow-out fractures of the orbital floor. II. Early repair for selected injuries. *Surv Ophthalmol* 1991;35:280–292
- 40 O-Lee T, Koltai P. Pediatric facial fractures. In: Pereira K, Mitchell RB, eds. *Pediatric Otolaryngology for the Clinician*. New York, NY: Humana Press; 2009:91–95
- 41 Koornneef L. New insights in the human orbital connective tissue. Result of a new anatomical approach. *Arch Ophthalmol* 1977;95:1269–1273
- 42 McGuirt WF, Salisbury PL III. Mandibular fractures. Their effect on growth and dentition. *Arch Otolaryngol Head Neck Surg* 1987;113:257–261
- 43 McGraw BL, Cole RR. Pediatric maxillofacial trauma. Age-related variations in injury. *Arch Otolaryngol Head Neck Surg* 1990;116:41–45
- 44 Chen CT, Chen YR. Endoscopically assisted repair of orbital floor fractures. *Plast Reconstr Surg* 2001;108:2011–2018; discussion 2019
- 45 Rubin PAD, Bilyk JR, Shore JW. Management of orbital trauma: fractures, hemorrhage, and traumatic optic neuropathy. *Clin Mod Ophthalmol* 2007;1004:7
- 46 Tessier P, Kawamoto H, Posnick J, Raulo Y, Tulasne JF, Wolfe SA. Taking calvarial grafts, either split in situ or splitting of the parietal bone flap ex vivo—tools and techniques: V. A 9650-case experience in craniofacial and maxillofacial surgery. *Plast Reconstr Surg* 2005;116(5, Suppl):54S–71S; discussion 92S–94S
- 47 Moore MH, David DJ, Cooter RD. Oblique craniofacial fractures in children. *J Craniofac Surg* 1990;1:4–7
- 48 Lende RA, Erickson TC. Growing skull fractures of childhood. *J Neurosurg* 1961;18:479–489
- 49 Howship J. *Practical Observations in Surgery and Morbid Anatomy*. London, UK: Longman, Hurst, Rees, Orme and Brown; 1816:494
- 50 Menkü A, Koç RK, Tücer B, Kurtsoy A, Akdemir H. Growing skull fracture of the orbital roof: report of two cases and review of the literature. *Neurosurg Rev* 2004;27:133–136
- 51 Manson P. Growing skull fractures and their craniofacial equivalents. (Commentary) *J Craniofac Surg* 1995;6:111
- 52 Havlik RJ, Sutton LN, Bartlett SP. Growing skull fractures and their craniofacial equivalents. *J Craniofac Surg* 1995;6:103–110; discussion 111–112
- 53 Furnas DW, Bircoll MJ. Eyelash traction test to determine if the medial canthal ligament is detached. *Plast Reconstr Surg* 1973;52:315–317
- 54 Markowitz BL, Manson PN, Sargent L, et al. Management of the medial canthal tendon in nasoethmoid orbital fractures: the importance of the central fragment in classification and treatment. *Plast Reconstr Surg* 1991;87:843–853
- 55 Jones L. Conjunctivodacryocystorhinostomy. *Indian J Ophthalmol* 1967;15:86–93
- 56 Mendelblatt FI, Kirsch RE, Lemberg L. A study comparing methods of preventing the oculocardiac reflex. *Am J Ophthalmol* 1962;53:506–512
- 57 Sires BS, Stanley RB Jr, Levine LM. Oculocardiac reflex caused by orbital floor trapdoor fracture: an indication for urgent repair. *Arch Ophthalmol* 1998;116:955–956
- 58 Kumar AV, Staffenberg DA, Petronio JA, Wood RJ. Bioabsorbable plates and screws in pediatric craniofacial surgery: a review of 22 cases. *J Craniofac Surg* 1997;8:97–99
- 59 Joughin K, Antonyshyn O, Wilson KL, Riding M. Persistent posttraumatic orbital-antral fistula. *Ann Plast Surg* 1993;30:77–79

- 60 Manson P. Commentary on the long-term effects of rigid fixation on the growing craniomaxillofacial skeleton. *J Craniofac Surg* 1991;2:69
- 61 Eppley BL, Platis JM, Sadove AM. Experimental effects of bone plating in infancy on craniomaxillofacial skeletal growth. *Cleft Palate Craniofac J* 1993;30:164–169
- 62 Hollier LH, Rogers N, Berzin E, Stal S. Resorbable mesh in the treatment of orbital floor fractures. *J Craniofac Surg* 2001;12:242–246
- 63 Scott AB. Botulinum toxin injection into extraocular muscles as an alternative to strabismus surgery. *J Pediatr Ophthalmol Strabismus* 1980;17:21–25
- 64 Ho SF, Pherwani A, Elsherbiny SM, Reuser T. Lateral tarsal strip and quickert sutures for lower eyelid entropion. *Ophthal Plast Reconstr Surg* 2005;21:345–348
- 65 Giroto JA, MacKenzie E, Fowler C, Redett R, Robertson B, Manson PN. Long-term physical impairment and functional outcomes after complex facial fractures. *Plast Reconstr Surg* 2001;108:312–327
- 66 Holt GR, Holt JE. Incidence of eye injuries in facial fractures: an analysis of 727 cases. *Otolaryngol Head Neck Surg* 1983;91:276–279
- 67 Giroto JA, Gamble WB, Robertson B, et al. Blindness after reduction of facial fractures. *Plast Reconstr Surg* 1998;102:1821–1834