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Aspects of Pediatric Oculoplastics Trauma: Floor Fracture and Canalicular Lacerations

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The management of orbital trauma utilizes different approaches to achieve the same final goal: the restoration of normal orbital and craniofacial anatomy and the return of function. Orbital floor fractures and canalicular lacerations in the pediatric age group require special considerations in diagnosis, management, and treatment. This issue of *Ophthalmology Rounds* addresses the unique clinical features and surgical management options for orbital floor fractures and canalicular lacerations in the pediatric age group.

Orbital floor fractures

History

In 1844, in Paris, MacKenzie first described orbital floor fractures.[1] In 1957, Smith and Regan used the term "blow-out fracture" to describe inferior rectus entrapment with decreased ocular motility in the setting of an orbital floor fracture.[2]

Mechanism of injury

One pathophysiological mechanism of injury that has been proposed is increased intraorbital hydraulic pressure.[2] There is a high-velocity impact to the globe and upper eyelid that transmits kinetic energy to the periocular structures and causes an increase in hydraulic pressure with a downward vector. This force usually targets the infraorbital groove, with most fractures occurring in the posterior medial region, which is the thinnest bony orbital area (0.5-1 mm). Another proposed mechanism, the buckling theory, states that fractures occur when an object strikes the inferior orbital rim, dispersing kinetic energy and causing buckling of the orbital floor.[3]

Clinical presentation and complications

Patients with facial injuries initially must be stabilized; airway security, hemodynamic stability, and cervical-spine integrity are the first priorities. Significant injury to the globe may occur in association with orbital fractures; therefore, a complete ophthalmic examination is necessary. In fact, a 30% incidence of a ruptured globe in association with orbital fractures has been reported.[4] Decreased visual acuity may indicate intraocular injury (traumatic optic neuropathy, retinal detachment, commotio retina, and/or macular hole). Pupillary function may be abnormal in traumatic optic neuropathy, third nerve/ciliary ganglion injury and traumatic mydriasis. Slit lamp examination is important to search for corneal abrasion, globe rupture, and traumatic hyphema, while a dilated fundus examination is essential in ruling out posterior pole trauma.

In patients with orbital fractures, significant periocular ecchymosis and edema are commonly present; however, in children the white-eyed blowout fracture with inferior rectus incarceration must be recognized.[5] Pediatric patients with floor fractures often present with a "white eye" and the absence of edema and ecchymosis, yet they have diplopia and significant limitation of upgaze with intractable nausea secondary to the oculocardiac reflex.[6] Since children have orbital bones that are very flexible and less likely to break, they commonly develop trap door fractures.[7] The inferior rectus muscle and/or orbital tissue can become entrapped within the fracture site and can lead to a compartment syndrome with muscle ischemia and necrosis. This prohibits the upward movement of the globe, causing diplopia, necessitating immediate surgery to restore normal muscle func-

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Figure 1: An 8-year-old patient with a left orbital floor fracture and inferior rectus incarceration.

Figure 2: Three months after immediate repair, there is no enophthalmos or diplopia and full motility.

tion (Figures 1 and 2). Radiologically, these fractures may not seem impressive, with minimal bone displacement, but the scans must be carefully examined in light of the clinical presentation. This is in contrast to adult orbital floor fractures where the lack of bony flexibility results in comminuted fractures.

The position of the globe should also be assessed. Although enophthalmos is rarely evident in the first days after injury due to edema of the orbital tissues, in fact, proptosis may be present initially. Hypoglobus and/or vertical diplopia may also be present as a result of muscle entrapment that will tether the globe downwards. In some cases, patients have a hypertropia in primary position that is associated with a posterior blowout fracture. With these fractures, computed tomography (CT) demonstrates a characteristic depressed fracture of the posterior orbital floor extending to the posterior wall of the maxillary sinus in all patients. In many of these patients, the inferior rectus loops inferiorly and then rises to contact the globe at a steep angle.[8] Extraocular muscle edema, hemorrhage, and nerve palsy may also cause diplopia. Traumatic rupture of an extraocular muscle has been reported and should be evident on the CT scan; it can be the cause of a hypertropia in primary position. Muscle entrapment is reported to be more frequent with small fractures that have less enophthalmos. In large fractures, enophthalmos is more likely because of increased orbital volume, while entrapment is less likely. Infraorbital nerve hypoesthesia is reported in as many as 60% of orbital floor blow-out fractures. Disruption of the mucosal integrity of the maxillary or ethmoidal sinus may result in subcutaneous or intraorbital emphysema, which can be worsened by nose blowing.

The following are complications of floor fractures that may lead to loss of vision and should not be overlooked:

- traumatic retrobulbar hemorrhage and the development of an orbital compartment syndrome secondary to hemorrhage or emphysema
- traumatic optic neuropathy
- coexistent ocular injury such as globe rupture, traumatic hyphema, retinal detachment, macula hole, intraorbital/ ocular foreign body, and injury to the contralateral eye.

Imaging studies

CT scanning is the imaging study of choice in orbital fractures.[1] Axial, saggital, and coronal 3-mm sections should be performed to accurately assess the orbital floor for fractures and entrapped muscles (Figures 3 and 4). Although soft tissue windows are helpful for demonstrating the incarcerated tissue, bony windows can assist in visualizing subtle discontinuities in the bone. Sections of 1-mm thickness may be useful to assess optic-canal fractures and traumatic optic neuropathy. Three-dimensional reconstructed images of the orbit are valuable adjuncts for planning the surgical repair of complex fractures. Magnetic resonance imaging (MRI) has poor bony resolution, which limits its role in orbital trauma. In cases of orbital-apex trauma and traumatic optic neuropathy, however, MRI may serve to visualize the anatomy of the apex and any hemorrhaging in the sheath of the optic nerve. Plain radiographic films, paramount prior to the advent of CT scans, are still used to assess facial fractures and look for foreign bodies. Waters, Caldwell, submental, anteroposterior (AP), and lateral views depict the facial skeleton. Radiographic evidence of orbital fractures includes fragmentation and misalignment of bone contours, a fluid level in the maxillary sinus, air in the orbit, and prolapse of orbital soft tissue into the maxillary sinus.[9]

Figure 3: Sagittal CT scan of a left floor fracture with entrapment. Note the minimally displaced fracture.

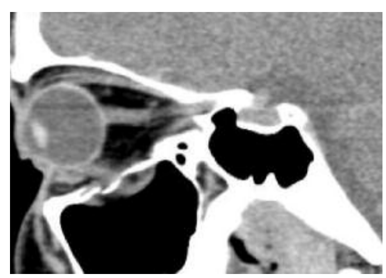
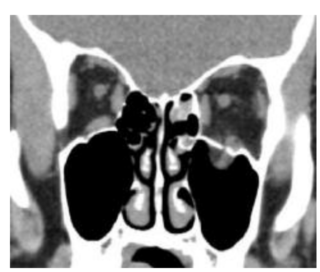


Figure 4: Coronal CT scan showing a left trap door fracture with inferior rectus incarceration.



Medical and surgical interventions

Indications for surgical repair include pediatric patients with isolated orbital floor fractures who have the "white-eyed" blowout fracture, severe limitations of extraocular motility from entrapment of the inferior rectus muscle, and nonresolving symptoms from the oculocardiac reflex (nausea and vomiting). These patients require immediate surgical repair to relieve the restriction in ductions and decrease systemic symptoms. For patients with severe limitation of ductions, early surgical repair within 7 days of injury results in more rapid improvement in ductions and diplopia than surgery performed later.[10] Surgery within 2 weeks is recommended in cases of persistent diplopia and evidence of orbital soft tissue entrapment on CT, or for large orbital floor fractures, that may cause latent enophthalmos or hypoglobus. Perioperatively, patients may require analgesia and antiemetics. The use of oral steroids (prednisone 1 mg/kg/d) has been advocated to decrease soft-tissue edema and oral antibiotics to decrease the likelihood of infection. Patients should avoid blowing their nose and performing the Valsalva maneuver to limit intraorbital emphysema.

Surgical repair is performed under general anesthesia. Prior to any surgical incision, forced duction testing is performed to confirm extraocular muscle restriction and for intraoperative comparison. The orbital floor is most efficiently accessed through a transconjunctival approach, although transcutaneous exposure or a transmaxillary approach may be used. Access to this region allows for exploration and release of displaced or entrapped soft tissue, thereby correcting any extraocular motility disturbances. In addition, repair of the bony defect allows for restoration of the partition between the orbit and maxillary antrum, thereby preserving orbital volume and eliminating any impingement on soft tissue structures. Because pediatric floor fractures are noncomminuted in nature, successful repair does not typically require the use of an orbital floor implant. If necessary, a number of implants are available for reconstruction of the orbital floor, including:

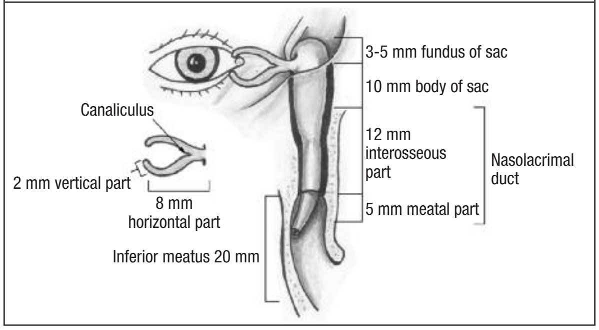
- autogenous materials (split cranial bone graft, cartilage, fascia)
- nonabsorbable alloplastic materials (titanium, silicone, porous polyethylene, Teflon®)
- alloplastic absorbable materials (polydioxanone, polylactide, polyglactin, and allogenic dura).[9]

If the fracture is repaired in a timely manner, motility correspondingly improves within days to weeks. In patients who present several weeks after the trauma, it may take several months before maximal recovery is achieved.

Canalicular lacerations

Canalicular lacerations following penetrating or blunt trauma occur in 16% of eyelid lacerations.[11] They can be managed by a number of surgical techniques involving different types of stents for canalicular intubation. Improper management can lead to significant long-term morbidity including epiphora, ocular irritations, and recurrent discharge and infections.

Figure 5: Anatomy of the lacrimal system[13]



Anatomy

The anatomy of the canalicular system includes the 2-mm vertical portion, which drains the puncta, and the 8-mm horizontal limb, which lies approximately 2 mm from the lid margin. The horizontal portion of the canaliculi converges to form the common canaliculus. Canaliculi pierce the lacrimal fascia before entering the lacrimal sac (Figure 5). The canaliculus runs along with the medial canthal tendon. This structure is often disrupted from the trauma and must be repaired to reestablish anatomic position and lid function.

The eyelid can be divided into the larger 'non-lacrimal' portion (consisting of skin, orbicularis muscle, lid retractors, tarsal plate, and conjunctiva) and the smaller "lacrimal" medial portion (consisting of skin, orbicularis, canaliculi, and conjunctiva). The canalicular portion of the eyelid has no tarsal support and because of its superficial location in the medial lid, it is vulnerable to trauma. When the eyelid is stretched against its fixed tendonous attachments past its internal elastic limit, it ruptures at its weakest point, the canaliculus.[11]

Mechanism of injury

Although many classifications of canalicular injury have been described, three of the most common types include direct (penetrating) injury, indirect (avulsive), or diffuse (avulsive).

Direct/penetrating injuries: A sharp object directly lacerates the canaliculus (eg, a knife or a tree branch).

Indirect injuries: A blunt object strikes a point remote from the canalicular portion of the eyelid resulting in avulsion of the canaliculi (eg, a fist).

Diffuse: Trauma is directed across the entire midface with multiple forces acting in several directions for injury, creating an avulsion injury as well as other soft tissue and/or bony injuries, but there is no evidence of a direct penetrating injury (eg, a motor vehicle crash).[14]

In a study by Jordan et al,[14] of 236 patients reviewed, direct canalicular injuries were detected in 128 (54.2%), indirect injuries were detected in 60 (25.4%), and diffuse injuries were detected in 48 (20.3%). A smaller study by Wulc and Arterberry[12] found that direct injuries were seen in 4 of 25 patients (16%), whereas avulsions resulting from indirect or diffuse trauma were much more common and occurred in 18 patients (84%).

Assessment and repair of canalicular lacerations

Many comprehensive ophthalmologists view canalicular lacerations with some apprehension, but there are essentially only 3 steps in successful repair.

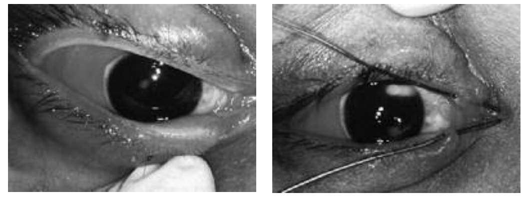
Step 1: Making the diagnosis

Ancillary considerations in the patient with canalicular trauma include damage to the levator muscle, the integrity of the medial and lateral canthal tendons, as well as the intraocular, intraorbital, and/or intracranial structures. If canalicular damage is suspected, an attempt should be made to gently dilate, probe, and irrigate (if possible) the canaliculus at the slit lamp. In many cases, this can be performed with topical anesthetic alone. Many patients are referred for post-traumatic ocular examinations who have undiagnosed canalicular lacerations (Figure 6); while many patients referred with monocanalicular lacerations have coexisting bicanalicular lacerations (Figure 7). For this reason the upper and lower canalicular systems of all of these patients must be examined to rule out unsuspected injuries.

Step 2: Finding the medial cut end

Surgical repair of pediatric monocanalicular lacerations is almost always performed under general anesthesia. Magnification and good lighting are essential for these cases. The singlemost important step in successful surgical repair is locating the medial cut end of the lacerated canaliculus. However, because this is difficult, particularly in cases with extensive facial trauma, a number of techniques have been developed to assist this location process. Although the diagnosis may be quite obvious in some cases (Figure 8), direct visualization and gentle tissue manipulation with cotton pledgets can help localize the medial cut end of the canaliculus. If this is unsuccessful, different solutions (eg, saline, antibiotics, methylene blue, sodium hyaluronate, and fluorescein) can be injected into the normal canaliculus, while maintaining pressure on the lacrimal sac to induce fluid egress through the medial cut end.[15] Lastly, the passage of a pigtail probe into the normal canaliculus will cause it to emerge from the medial cut end. However, there was the potential for damage to the uninvolved canaliculus and/or common canaliculus with the earlier types of pigtail probes. The early Worst pigtail probe, for example, was known to cause lacrimal damage because it had a sharp barb on the end that invariably caused tissue damage (to the common canaliculus and uninvolved canaliculus) when it was externalized back through the lacerated medial end. The newer pigtail probes are safer because they have a smooth-tip and an eyelet at the end, with no sharp barb.

Figure 7: Post traumatic right lower lid canalicular laceration with concomitant right upper lid canalicular laceration.



Step 3: Stenting the lacrimal system

After identifying the medial cut end, a stent is placed in the system to allow for correct anatomical healing. The choice of stent depends on whether the laceration is monocanalicular or bicanalicular in nature.

Bicanalicular lacerations: The simplest bicanalicular stent is a loop of nonabsorbable suture (nylon, polypropylene) that is threaded through both the involved and uninvolved canaliculi with a knot that is tied and rotated into the canaliculus. This can be performed with a smooth-tipped pigtail probe. Potential drawbacks of this technique include cheese wiring of the suture though the system and contracture of the healing canalicular lumen to the circumference of the suture. This is a suboptimal result, since the lumen should be as large as possible. As a result, most surgeons elect

Figure 6: Ocular trauma with repaired right upper and lower lid cutaneous lacerations and undiagnosed bicanalicular lacerations.

Figure 8: Avulsed left lower lid and canalicular laceration secondary to display hook.

Figure 9: Note junction of polypropylene suture and silicone tubing of Ritleng tubes.

Figure 10: Ritleng probe with suture passing through hollow lumen.

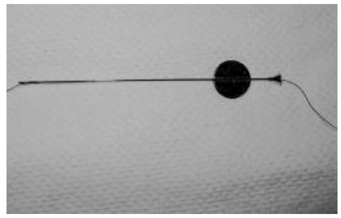
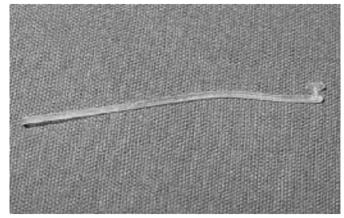


Figure 11: Mini-Monoka tube: note beveled end fits snuggly into the punctum



to additionally thread a small piece of silicone tubing over the suture and then internalize the knot to maximize canalicular volume. This technique is referred to as an "annular intubation."

Silicone tubes are commonly used to stent the lacerated canalicular system during the healing stages. Crawford bicanalicular silicone tubes (FCI Ophthalmics, Marshfield Hills, MA) are typically placed in patients under general anesthesia and routinely used for bicanalicular lacerations. After dilation of the punctum and localization of the medial and lateral cut ends of the lacerated canaliculus with a Bowman probe, the olive tip of the metallic guide of the silicone tube is passed through the canaliculus, down the nasolacrimal duct and into the nose. This is performed for each canaliculus and a Crawford hook is used to engage the olive tip and externalize the silicone tubes. The tubes can be secured with either a suture or several knots tied in the tubing, followed by retraction under the inferior turbinate.

Because the Crawford hook can induce epistaxis from trauma to the nasal mucosa, a less-traumatic method of bicanlicular intubation is also available to the surgeon. Ritleng silicone tubes (FCI Ophthalmics, Marshfield Hills, MA) differ from Crawford tubes, since there is a polypropylene suture swedged to the end of the silicone tubing (Figure 9). After the hollow Ritleng probe is introduced into the nasolacrimal system and down the duct, the polypropylene end of the silicone tube is fed through the lumen of the probe and emerges in the inferior meatus (Figure 10). The suture can be easily engaged and externalized with a blunt muscle hook, and the silicone tubing is also externalized. The same sequence is then performed for the other canaliculus and the tubing is secured intranasally.

Finally, bicanalicular lacerations can be repaired with two individual monocanalicular stents, as described in the following section.

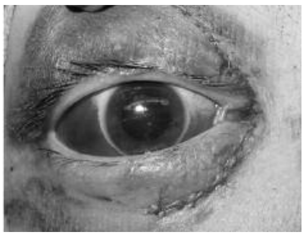
Monocanalicular lacerations: Anatomically, the region that requires stenting in the patient with a monocanalicular laceration is not the uninvolved canaliculus or the nasolacrimal duct (as in congenital nasolacrimal duct obstruction), but only the involved canaliculus. Although some physicians may prefer to place bicanalicular silicone tubes in all patients with canalicular lacerations, monocanalicular silicone stents (mini-Monoka – FCI Ophthalmics, Marshfield Hills, MA) have been developed exclusively for the case where bicanalicular intubation is unnecessary (Figure 11).[15] Because intranasal manipulation is not required with this system, it affords the surgeon the potential to carry out the procedure with local anesthesia alone. The punctum is first dilated and the proximal and distal cut ends of the canaliculus should be re-identified using a Bowman probe. The mini-Monoka is usually much longer than the length required to stent the region of the laceration, so a portion is usually trimmed and discarded. It is passed through the punctum and medial cut end until the beveled end snaps into place at the punctum (Figure 11). The distal portion of the tubing is then passed through the medial cut end. If there is significant resistance, it is possible that excess tubing remains making it impossible to achieve the 90° turn down the nasolacrimal duct. The additional portion of the tubing should be removed and, at this point, it should rest in the system without tension (Figure 12).

Soft tissue closure: Once the system is adequately stented, soft tissue closure is the last step in repair. These steps are the same whether one is managing a monocanalicular laceration or bicanalicular laceration (Figures 13 and 14). The cut ends of the canaliculus may be directly anastomosed with a 7-0 polyglactin suture and the deeper obicularis muscle tissue may be sutured with a 5-0 polyglactin suture. It is important to note that the medial lid has no tarsus; therefore, the deep soft

Figure 12: Mini-Monoka tube in correct position prior to soft tissue repair.

Figure 13: Well-healed lower lid canalicular laceration with monocanalicular stent in place (same patient as in Figure 8).

Figure 14: Bicanalicular silicone tubes following bicanalicular laceration (same patient as in Figure 6).



tissue anchoring sutures must be secure. If damaged, the medial canthal tendon can be repaired with a double-armed 5-0 polyglactin suture. Lastly, a 6-0 plain gut suture is used to close the cutaneous wound.

Postop management

Patients are placed on topical and systemic antibiotics postoperatively. The silicone stents are left in place for 3 months, at which point they are removed. The monocanalicular stents can be easily removed using the slit lamp (age-dependent), but bicanalicular tubes often require a short inhalational anesthetic to facilitate removal.

Conclusion

In the pediatric age group, orbital floor fractures and canalicular lacerations present unique challenges that must be addressed in order to achieve successful long-term outcomes.

References

1. Ng P, Chu C, Young N, Soo M. Imaging of orbital floor fractures. *Australas Radiol.* 1996;40(3):264-268.
2. Smith B, Regan WF Jr. Blow-out fracture of the orbit; mechanism and correction of internal orbital fracture. *Am J Ophthalmol.* 1957;44(6):733-739.
3. Fujino. T. Experimental "blowout" fracture of the orbit. *Plast Reconstr Surg.* 1974;54(1):81-82.
4. Wilkins RB, Havins WE. Current treatment of blow-out fractures. *Ophthalmology.* 1982;89(5):464-466.
5. Jordan DR, Allen LH, White J, Harvey J, Pashby R, Esmaeli B. Intervention within days for some orbital floor fractures: the white-eyed blowout. *Ophthal Plast Reconstr Surg.* 1998;14(6):379-390.
6. Sires BS, Stanley RB Jr, Levine LM. Oculocardiac reflex caused by orbital floor trapdoor fracture: an indication for urgent repair. *Arch Ophthalmol.* 1998; 116(7):955-956.
7. 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(8):723-727.
8. Seiff SR, Good WV. Hypertropia and the posterior blowout fracture: Mechanism and management. *Ophthalmology.* 1996;103(7):995-996.
9. Cohen AJ. *Facial Trauma, Orbital Floor Fractures (Blowout).* www.emedicine.com/plastic/topic485.htm. Accessed: November 3, 2008.
10. Egbert JE, May K, Kersten RC, Kulwin DR. Pediatric orbital floor fracture: direct extraocular muscle involvement. *Ophthalmology.* 2000;107(10):1875-1879.
11. Herzum H, Holle P, Hintschich C. Eyelid injuries: epidemiological aspects. *Ophthalmologe.* 2001;98(11):1079-1082.
12. Wulc AE, Arterberry JF. The pathogenesis of canalicular laceration. *Ophthalmology.* 1991;98(8):1243-1249.
13. Parwar B. *Nasolacrimal System Anatomy.* www.emedicine.com/ent/topic5.htm. Accessed: November 4, 2008.
14. Jordan DR, Ziai S, Gilberg SM, Mawn LA. Pathogenesis of canalicular lacerations. *Ophthal Plast Reconstr Surg.* 2008;24(5):394-398.
15. Buerger DE. Repair of canalicular laceration can be simplified. *Ophthalmology Times.* July 1, 1998.

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